APPLICATION OF FLUID-STRUCTURE INTERACTION TO NUMERICAL SIMULATIONS IN THE LEFT VENTRICLE

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ABSTRACT
Numerical simulations of blood flow and myocardium motion for an average canine left ventricle (LV) with fluid-structure interaction were performed. The temporal variations of the LV cavity pressure and wall stress during the cardiac cycle were consistent with previous literature. LV cavity volume was conserved from one period to the next, despite sub-physiological ejection volumes and brief periods of backflow during early filling. This study improves on previous ones by presenting details of the models and results for both the fluid and solid components of the LV.

Keywords: biomechanics; cardiovascular system; left ventricle; fluid-structure interaction.

APPLICATION DE L’INTERACTION FLUIDE-STRUCTURE À LA SIMULATION NUMÉRIQUE DU VENTRICLE GAUCHE

RÉSUMÉ
Nous présentons une modélisation numériques par interactions fluide-structure de l’écoulement sanguin et de la déformation du myocarde pour un ventricule gauche (VG) d’un coeur moyen de chien. La variation temporelle de la pression dans la cavité du VG et des contraintes dans les parois ventriculaires sont conformes avec les résultats publiés antérieurement. Le volume de la cavité du VG est conservé d’une période à l’autre, en dépit d’une fraction éjecté inférieur aux données physiologiques et de courtes périodes avec écoulement inversé au début de la phase de remplissage. Notre étude améliore les travaux antérieurs en présentant des modèles et des résultats détaillés obtenus par couplage du fluide et de la paroi du VG.

Mots-clés : biomécanique; système cardio-vasculaire; ventricule gauche; interaction fluide-structure.
## NOMENCLATURE

<table>
<thead>
<tr>
<th>Symbol</th>
<th>Description</th>
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<tbody>
<tr>
<td>$a$</td>
<td>outer semi-major axis length (mm)</td>
</tr>
<tr>
<td>$a_i$</td>
<td>sigmoid function slope parameter (–)</td>
</tr>
<tr>
<td>$b$</td>
<td>outer semi-minor axis length (mm)</td>
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<tr>
<td>$b_i$</td>
<td>sigmoid function centre location parameter (–)</td>
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<tr>
<td>$C$</td>
<td>damping matrix (N/mm·ms)</td>
</tr>
<tr>
<td>$C_1$</td>
<td>first passive material parameter value (kPa)</td>
</tr>
<tr>
<td>$C_2, \ldots, C_6$</td>
<td>second through sixth passive material parameter values (–)</td>
</tr>
<tr>
<td>$c$</td>
<td>distance from the apex (mm)</td>
</tr>
<tr>
<td>$D$</td>
<td>diameter (mm)</td>
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<tr>
<td>$D_i$</td>
<td>active material parameter values (kPa)</td>
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<tr>
<td>$E$</td>
<td>Green’s strain tensor (–)</td>
</tr>
<tr>
<td>$EF$</td>
<td>ejection fraction (–)</td>
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<td>$F$</td>
<td>forcing function (–)</td>
</tr>
<tr>
<td>$F^B$</td>
<td>force vector equivalent to the element stresses</td>
</tr>
<tr>
<td>$f$</td>
<td>body force per unit volume (N/mm³)</td>
</tr>
<tr>
<td>$h$</td>
<td>total height (mm)</td>
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<tr>
<td>$J_i$</td>
<td>reduced invariants of $E$ (–)</td>
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<tr>
<td>$K$</td>
<td>stiffness matrix (N/mm)</td>
</tr>
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<td>$L$</td>
<td>length (mm)</td>
</tr>
<tr>
<td>$M$</td>
<td>mass matrix (N/mm·ms²)</td>
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<tr>
<td>$p$</td>
<td>pressure (kPa)</td>
</tr>
<tr>
<td>$Q$</td>
<td>exponent in strain energy density function</td>
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<tr>
<td>$R$</td>
<td>radius (mm)</td>
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<tr>
<td>$R$</td>
<td>external load vector (N)</td>
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<tr>
<td>$Re$</td>
<td>Reynolds number (–)</td>
</tr>
<tr>
<td>$t$</td>
<td>time (ms); also distance from the exterior wall (mm)</td>
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<tr>
<td>$T_e$</td>
<td>effective Cauchy stress (kPa)</td>
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<td>$T_i$</td>
<td>principal Cauchy stresses (kPa)</td>
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<td>$T_{ij}$</td>
<td>components of Cauchy stress tensor (kPa)</td>
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<td>nodal displacement vector (mm)</td>
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<td>$\dot{U}$</td>
<td>nodal velocity vector (mm/ms)</td>
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<td>$\ddot{U}$</td>
<td>nodal acceleration vector (mm/ms²)</td>
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<td>$v$</td>
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<td>strain energy density function (kJ/m³)</td>
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<td>width (mm)</td>
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<tr>
<td>$w$</td>
<td>mesh velocity vector (mm/ms)</td>
</tr>
<tr>
<td>$x, y, z$</td>
<td>global Cartesian coordinates (mm)</td>
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### Greek symbols

<table>
<thead>
<tr>
<th>Symbol</th>
<th>Description</th>
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<tbody>
<tr>
<td>$\alpha$</td>
<td>angle between the left ventricle outflow tract and the basal plane (°)</td>
</tr>
<tr>
<td>$\beta$</td>
<td>angle between the left ventricle inflow tract and the basal plane (°)</td>
</tr>
<tr>
<td>$\theta$</td>
<td>angle between the aortic and mitral valve planes (°)</td>
</tr>
<tr>
<td>$\kappa$</td>
<td>bulk modulus (kPa)</td>
</tr>
<tr>
<td>$\lambda_i$</td>
<td>principal stretches (–)</td>
</tr>
<tr>
<td>$\mu$</td>
<td>viscosity (kg/m·s)</td>
</tr>
<tr>
<td>$\rho$</td>
<td>density (kg/m³)</td>
</tr>
<tr>
<td>$\rho_m$</td>
<td>density of the compressible fluid(kg/m³)</td>
</tr>
<tr>
<td>$\tau$</td>
<td>dimensionless time (–)</td>
</tr>
<tr>
<td>$\chi$</td>
<td>active stress multiplier (–)</td>
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1. INTRODUCTION

The heart is a prominent example of a system whose operation is governed by the mutual interaction of fluid (blood) and deformable solid (myocardium) components. A complete numerical model of the mechanics of the heart must include models for both the blood and the myocardium, each with appropriate geometry, material model, and boundary and initial conditions. Moreover, the fluid and solid numerical models must be coupled and solved together, thus accounting for fluid-structure interaction (FSI) effects. The numerical simulation of the mechanics of the entire heart, including its four deformable chambers and four moving valves, remains a challenging problem, especially if the goal of the study is to calculate detailed physiologically-relevant results for both the fluid and solid components. To reduce the model complexity and required computational resources, researchers have generally focused their simulations on a single heart chamber, typically the left ventricle (LV), which is the main pumping chamber of the heart and is responsible for pumping blood through the body.

Previous numerical studies of the mechanics of the heart may be classified into three groups: solid-only simulations, in which the motion of the muscle is simulated using a specified pressure variation as a boundary condition; computational fluid dynamics (CFD) simulations, in which blood flow is simulated using the motion of the heart wall as a boundary condition; and FSI simulations, which solve the motions of the fluid and solid parts and couple them together on the fluid-solid boundaries. Solid-only simulations of LV myocardium mechanics [1–3] help us to understand the mechanical behaviour of the myocardium. The physiological relevance of these simulations is compromised by their common assumption that the blood pressure acting on the inner surfaces of the LV cavity is uniform and their inability to account for the spatial and temporal changes in this pressure in response to the deformation of the LV wall. CFD simulations of the LV examine the flow of blood into and out this chamber. These simulations rely on the specification of myocardium motion, either through simplifying assumptions [4, 5] or through the definitions of multiple fluid meshes throughout the cardiac cycle based on imaging techniques [6–8]. In such studies, myocardium motion is prescribed and its complex non-uniform deformation over the cardiac cycle under the influence of blood pressure and wall shear stress cannot be resolved. FSI simulations of the heart can be further divided into two general groups. Studies in the first group use the immersed boundary method (IBM) [9, 10], often for the entire heart. IBM models are capable of including multiple heart chambers and moving valves, but cannot calculate stresses and strains for the myocardium. Those in the second group use finite element or finite volume techniques [11–13] and generally simulate a single heart chamber. These FSI studies present detailed blood flow results, but have in general not included detailed spatial and temporal variations of myocardium stresses and strains.

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Fig. 1. Diagram showing the solid (a) and fluid (b) geometries along with key dimensions and geometric landmarks.

This article focuses on the mechanics of the LV during the cardiac cycle and builds upon our previous work, which described the development of the myocardium material model [14] and the calculation of an end-diastolic state to be used as an initial state for the present cardiac cycle simulations [15]. Our first objective was to utilize the FSI capabilities of the commercial finite element software ADINA v.8.5.2 (ADINA R & D, Inc., Watertown, MA, USA) to perform FSI simulations of the mechanics of a canine LV over the cardiac cycle. Our second objective was to calculate and present detailed spatial results for the blood and heart wall at the same time points, which has not been done in previous studies. The present simulations are for an idealized, "average" canine LV geometry. An effort was made to validate, as much as possible, all steps of the simulation process vs. available experimental and computational data.

2. METHODS

2.1. Cardiac Cycle Definitions

The period for our cardiac cycle simulations has been chosen to be 600 ms, which corresponds to a heart rate of 100 beats/min. Simulations were started at end diastole and proceeded through the four phases of the cardiac cycle, namely, isovolumetric contraction (IVC) and ejection, which are together referred to as systole, and isovolumetric relaxation (IVR) and filling, which are together referred to as diastole. The durations of these phases were $\tau = 0.055$ for IVC, $\tau = 0.257$ for ejection, $\tau = 0.073$ for IVR, and $\tau = 0.615$ for filling, where $\tau$ is dimensionless time, defined as the ratio of the corresponding time interval and the cycle period.

2.2. Geometry and Mesh

Our reference geometry was defined at diastasis, which is the middle phase of diastole, during which the muscle fibres are fully relaxed and both the pressure in the LV and its difference from the pressure in the left atrium are small [16]. It is from this reference geometry that the LV was inflated to an end-diastolic state prior to the start of the cardiac cycle simulations. The reference solid geometry was defined as a truncated prolate ellipsoid with an outer semi-major axis of $a = 46.9$ mm, an outer semi-minor axis of $b = 27.8$ mm, a thickness at the apex of $t_a = 6.8$ mm, an equatorial thickness of $t_b = 12.4$ mm, and a total height of $h = 60.1$ mm, as shown in Fig. 1a. This computational geometry consisted of six wall layers, each with their own unique muscle fibre orientation, ranging from $-60^\circ$ in the outer layer to $+60^\circ$ in the inner layer, with the angles defined with respect to the local circumferential direction. This range of fibre angles was chosen to represent average values measured for canine LVs from two independent studies [17, 18].
The parts of the computational geometry that contain fluid are shown in Fig. 1b. This figure also illustrates three geometrical landmarks: the basal plane, which is the top plane that truncates the ellipsoid; the equatorial plane, which is the horizontal plane passing through the centre of the full ellipsoid; and the apex, which is the lowest point of the LV. The fluid geometry was divided into two sections by the basal plane; the upper section, which was taken to be rigid, and the lower section, which was taken to be deformable. The boundary of the lower section coincided with the inner boundary of the corresponding solid geometry, thus allowing for matching FSI boundary conditions to be applied to the fluid and solid models. The upper section included two cylindrical tubes of equal lengths $L_1$, which represented the inflow and outflow tracts of the LV and contained idealized zero-thickness mitral and aortic valves, respectively, at a distance $L_2$ from the distal end of the cylinders. The reason for not defining the valve planes at the proximal ends of the LV inflow and outflow tracts was that, in that case, the two valves would have a common point, at which flow would erroneously pass through a valve when it was closed. To complete the fluid geometry, the part of the upper section outside the inflow and outflow tracts was fitted with a section of a sphere, whose radius $R$ and centre were chosen such that the sphere passed through all three points of the triangle that makes up the lower edges of the inflow and outflow tracts, as shown in Fig. 1b. The dimensions for the fluid geometry were $D_{AV} = 9.8$ mm, $D_{MV} = 18.9$ mm, $L_1 = 9.8$ mm, $L_2 = 7.8$ mm, $w_1 = 26.7$ mm, $\alpha = 60^\circ$, $\beta = 75^\circ$, $\theta = 135^\circ$, and $R = 18.9$ mm.

The solid geometry was meshed using 53,920 ten-node tetrahedral elements (78,252 nodes) on an unstructured grid. This mesh was found to be sufficiently fine for resolving accurately the LV cavity volume changes and stretches. Nevertheless, as demonstrated by Doyle et al. [15] for solid-only simulations, a finer mesh would have been desirable for resolving fully the stresses. Attempts to use a refined solid mesh caused our FSI simulations to diverge. Through a series of simulations performed with different time steps (results not shown), it has been confirmed that divergence was not caused by insufficient time step and so it is tentatively attributed to some undetermined subtle code requirement concerning the ratio of fluid and solid meshes. This limitation of the solid mesh is not expected to affect significantly our results and conclusions.

The fluid geometry was meshed using 4-node tetrahedral mini elements on an unstructured grid. Two meshes were considered: mesh 1, having 132,667 elements (24,023 nodes), and mesh 2, having 233,811 elements (41,733 nodes). To determine the dependence of the solution on the fluid mesh density, simulations were performed over the interval $0 \leq \tau \leq 0.312$ for these two fluid meshes. The LV cavity pressure $p_{LV}$ at the centre of the basal plane and LV cavity volume $V_f$ were calculated for the two fluid meshes and differences were found to be less than 1%. Therefore, mesh 1, which is coarser than mesh 2, was deemed to be sufficient for use for the remainder of the simulations. The use of an even more refined mesh would be required for a more complete assessment of the mesh dependence of the solution, but this would also require a substantial increase of the computational cost.

2.3. Numerical Methods

Simulations were performed using the ADINA code, for which separate solid and fluid models are defined and linked through coincident FSI boundary conditions. Simulations were run in parallel on the clusters of the High Performance Computing Virtual Laboratory (HPCVL), using at least 32 processors, based on the results of our parallelization study [19].

2.3.1. Governing equations for the myocardium

Simulations were performed dynamically, using the total Lagrangian formulation for the solid model to account for large deformations and material nonlinearities. The appropriate form of the solid model governing equation is

$$
M\ddot{U}(t + \Delta t) + C\dot{U}(t + \Delta t) + K(t)[U(t + \Delta t) - U(t)] = R(t + \Delta t) - F(t),
$$

(1)
where $U$, $\dot{U}$, and $\ddot{U}$ are the nodal displacement, velocity and acceleration vectors, respectively, $M$ is the mass matrix, $C$ is the damping matrix, $K$ is the stiffness matrix, $R$ is the external load vector, $F$ is the force vector equivalent to the element stresses, and $t$ is time [20]. Following the definition of a suitable stiffness matrix, the solid equations are solved using the implicit Newmark method, described in detail by Bathe [21].

The myocardium, which is the thick middle layer of the LV wall comprised of muscle fibres surrounded by an extracellular matrix, was modelled as a slightly compressible transversely isotropic hyperelastic material, with properties that differ in the fibre and cross-fibre directions. The other two wall layers, the endocardium and the epicardium are thin and, following common practice, were neglected in defining the present material model.

The myocardium material model was defined by a strain energy density function $W$, which consisted of passive ($p$) and active ($a$) parts. In this context, passive stresses are the stresses caused by the deformation of the myocardium and active stresses are stresses caused by the contraction of the muscle fibres. The total stresses were then calculated as the sum of the passive and active stresses. By varying the application of the active stresses in time, the contraction and relaxation of the muscle fibres was modeled. The strain energy density function used in this study has been described in detail by Doyle et al. [14] and is defined as

$$ W = W_p + W_a, $$

where

$$ W_p = C_1 (e^Q - 1) + 0.5 \kappa_s (J_3 - 1)^2, $$

$$ Q = C_2 (J_1 - 3)^2 + C_3 (J_1 - 3)(J_4 - 1) + C_4 (J_4 - 1)^2 + C_5 (J_1 - 3) + C_6 (J_4 - 1), $$

$$ W_a = D_0 + D_1 (J_1 - 3)(J_4 - 1) + D_2 (J_1 - 3)^2 + D_3 (J_4 - 1)^2 + D_4 (J_1 - 3) + D_5 (J_4 - 1). $$

In Eqs. (3–5), $C_i$ and $D_i$ are passive and active material parameter values, respectively, $\kappa_s$ is the bulk modulus, which governs the material compressibility, and $J_i$ are reduced invariants of Green’s strain tensor $E$. The passive material parameters have been previously [15] calculated as $C_1 = 2.117$ kPa, $C_2 = 0.498$, $C_3 = 0.237$, $C_4 = 0.0332$, $C_5 = 0.01$, $C_6 = 0.0005$, and $\kappa_s = 1 \times 10^7$ kPa.

For the active material model, to account for the contraction and relaxation of the muscle fibres, the material parameters $D_i$ are defined as

$$ D_i = FD_{i,\text{max}}, $$

where $F$ is a forcing function, which varies between 0 when the muscle fibres are fully relaxed and 1 when the muscle fibres are fully contracted, and $D_{i,\text{max}}$ are the values of the active material parameters when the muscle fibres are fully contracted [11]. By varying the value of $F$ over the interval $0 \leq F \leq 1$, the contraction and relaxation of the muscle fibres were modelled. This forcing function was defined by two sigmoid functions (one increasing and one decreasing) as

$$ F = \begin{cases} 
1/(1 + e^{-a_1(\tau-b_1)}), & 0 \leq \tau < 0.170 \\
1/(1 + e^{-a_2(\tau-b_2)}), & 0.170 \leq \tau \leq 1,
\end{cases} $$

where $a_i$ control the slopes of the sigmoid functions and $b_i$ control the locations of their centres. Through multiple simulations, the parameters for the sigmoid functions were adjusted to their final values of $a_1 = 100.00$, $b_1 = 3.06$, $a_2 = 60.61$, and $b_2 = 10.61$, such that the resulting LV pressure waveform resembled previous measurements [16] over the majority of the cardiac cycle.

The values of $D_{i,\text{max}}$ were defined in terms of a multiplier $\chi$, which multiplies the active stresses from measurements of rabbit active stresses from Lin and Yin [22] to approximate the active stresses in a canine LV, as described by Doyle et al. [14]. The following expressions were used for $D_{i,\text{max}}$: $D_{1,\text{max}} = -0.352\chi$ kPa, $D_{2,\text{max}} = 2.476\chi$ kPa, $D_{3,\text{max}} = 2.660\chi$ kPa, $D_{4,\text{max}} = 0.0251\chi$ kPa, and $D_{5,\text{max}} = 0.632\chi$ kPa. Note
that because only the derivative of \( W \) is required to calculate stresses, the value of \( D_0 \) in Eq. 5 is not needed and has been set to be zero.

Simulations were performed for several values of \( \chi \) to determine the influence of \( \chi \) on the results. From these simulations, we determined that \( \chi \) must be between 4 and 6 to have both model numerical convergence and blood exiting the LV at the start of ejection. We have chosen \( \chi = 4.5 \) for the remainder of our simulations.

2.3.2. Governing equations for blood

Blood is assumed to be a Newtonian fluid with a density \( \rho_f = 1050 \, \text{kg/m}^3 \) (defined at zero pressure) and a viscosity \( \mu = 0.00316 \, \text{kg/m} \cdot \text{s} \) [23]. To ensure solution convergence during IVR and IVC, during which both valves are closed and the LV cavity is deforming, blood was set to be a slightly compressible fluid with a bulk modulus \( \kappa_f = 1 \times 10^7 \, \text{kPa} \), which matches the value for the myocardium. The bulk moduli of the fluid and the solid were set to be equal in order to prevent compression of the material that would have the smaller bulk modulus.

To account for fluid mesh deformation during FSI simulations, an arbitrary-Lagrangian-Eulerian (ALE) approach was used, in which moving boundaries were tracked using the Lagrangian approach, stationary boundaries made use of the Eulerian approach, and the ALE method was used at all points in between [21]. The ALE form of the continuity and momentum equations for slightly compressible Newtonian fluids are defined as

\[
\frac{\rho_f}{\kappa_f} \left( \frac{\partial p}{\partial t} + (v - w) \cdot \nabla p \right) + \rho_m \nabla \cdot v = 0, \tag{8a}
\]

\[
\frac{\partial v}{\partial t} + \rho_f (v - w) \cdot \nabla v = -\nabla p + \mu \nabla^2 v + f^B, \tag{8b}
\]

where \( p \) is pressure, \( v \) is the velocity vector, \( w \) is the mesh velocity vector, \( f^B \) is the body force per unit volume, and \( \rho_m \) is the density of the compressible fluid, defined as [24]

\[
\rho_m = \rho_f \left( 1 + \frac{p}{\kappa_f} \right). \tag{9}
\]

Because the governing equations for the fluid are nonlinear, the solution of the fluid system of equations requires two steps: outer iterations, which are used to generate a system of linear equations, and the solving of this linearized system. For the outer iterations, the Newton-Raphson method was used, and for the linearized system of equations, a direct sparse solver was used [24].

2.3.3. Fluid-structure interaction

Simulations were performed using a direct FSI coupling method, in which the equations for the fluid, solid, and fluid-solid interface are solved simultaneously. Further details on this method, along with the FSI boundary conditions enforced in ADINA, can be found in [24].

For time integration, we used the implicit second-order ADINA composite method [24]. To determine the dependence of the solution on the time step, simulations were performed over the interval \( 0 \leq \tau \leq 0.312 \) for two time step sizes. In the first case, 600 time steps of 1 ms were used, whereas, in the second case, 303 time steps were used, which consisted of 297 time steps of 2 ms and 6 time steps of 1 ms. The 1 ms time steps in the second case were used during the times when the valves opened or closed so that these times matched for both cases. The times at which the valves opened or closed were chosen based on the pressure boundary conditions from Sabbah and Stein [16] and three of these four times were most closely approximated by odd integers, which is the reason for using these additional time steps. The differences in the resulting LV pressure and cavity volumes for the two cases were less than 1%, which are deemed to be insignificant. Therefore, all subsequent simulations were performed using 303 time steps.
2.3.4. Initial and boundary conditions

Before beginning cardiac cycle simulations, initial conditions at end diastole were generated by inflating the reference geometry at diastasis to an end-diastolic pressure of $p_{LV} = 2$ kPa using a procedure described by Doyle et al. [15]. In the previous study, only the solid geometry was considered and the simulations were performed statically. In the present study, both the fluid and the solid were considered, and the simulations were performed quasi-statically, that is with a sufficiently large time step ($\Delta t = 80$ ms) as to minimize the velocity of the incoming blood flow, such that its effect on the deformation of the myocardium was negligible. The resulting value of $V_f$ at end diastole was 56.3 mL, which was essentially the same as $V_f = 56.2$ mL for the solid-only simulations. In view of the discussion presented by Doyle et al. [15], the end-diastolic state calculated from the FSI simulations was deemed suitable for use as an initial state for the present cardiac cycle simulations.

Coincident FSI boundary conditions were applied to the inner surfaces of the solid geometry and the outer surfaces of the ellipsoidal part of the fluid geometry. The basal plane of the solid geometry was fixed in the vertical direction, whereas its inner edge was fixed in all three directions. For the upper part of the fluid geometry, no-slip wall boundary conditions were applied to all outer walls. Considering that the heart is immersed in pericardial fluid in a nearly neutrally buoyant manner, gravity has been neglected.

To control the flow direction, idealized mitral and aortic valves were modelled as instantly opening and closing planar boundaries at a distance $L_2$ from the distal ends of the LV inflow and outflow tracts, as shown in Fig. 1b. The choice of instantly opening and closing valves was made to reduce computational time and complexity in the numerical model. The opening and closing of the valves were controlled by "gap" boundary conditions, which remove or add wall boundary conditions using time functions. The times at which the valves opened or closed were chosen to coincide with the start of each phase of the cardiac cycle; the AV opened at the start of ejection ($\tau = 0.055$) and closed at the end of ejection ($\tau = 0.312$), whereas the mitral valve (MV) opened at the start of filling ($\tau = 0.385$) and closed at the end of filling ($\tau = 1.0$).

Because the LV does not operate in isolation, but is coupled to upstream and downstream components of the cardiovascular system, appropriate inflow and outflow boundary conditions must be defined. Based on previous measurements [16], we imposed pressures at the distal ends of the LV inflow and outflow tracts. These pressures are plotted in Fig. 2, where $p_{Ao}$ is the outflow boundary condition applied when the AV is open and $p_{LA}$ is the inflow boundary condition applied when the MV is open.

3. RESULTS

3.1. Left Ventricle Cavity Pressure Changes

Figure 2 is a plot of $p_{LV}$ as a function of $\tau$ over the first two periods of the cardiac cycle simulations, for which $p_{LV}$ was calculated at a node in the fluid mesh located at the centre of the basal plane. The calculated temporal variations of $p_{LV}$ show fairly good agreement with previous measurements, especially during the first three phases of the cardiac cycle, IVC, ejection, and IVR, but differ from the measurements during the early part of filling. During IVC, $p_{LV}$ increased in both the present simulations and the previous measurements. Trends of $p_{LV}$ for the present simulations were comparable for most of the two periods; however, at the start of IVC for the second period, $p_{LV} < 0$ for a single time step, as a result of the sudden closure of the MV. At $\tau = 0.055$, which signifies the opening of the AV and the end of IVC, $p_{LV}$ was calculated to be 13.5 kPa for both periods, which is slightly higher than the measured value $p_{LV} = 12.7$ kPa. During the early part of ejection, $p_{LV}$ increased to a maximum in both studies, before decreasing throughout the rest of ejection. The maximum value of $p_{LV}$ in the present study exceeded the measured value by approximately 5 kPa, but at the end of ejection during period 1, pressures in both studies were nearly equal. During period 2, $p_{LV}$ spiked upward at the end of ejection due to the sudden closure of the AV. Another noticeable difference between the trends of $p_{LV}$ for the two periods was that, whereas for period 1
Fig. 2. Temporal variation of LV cavity pressure from present simulations along with measured values of $p_{LV}$, $p_{LA}$, and $p_{Ao}$ from Sabbah and Stein [16]. Circles denote the start of a phase of the cardiac cycle and the opening or closing of a valve.

$p_{LV}$ decreased after reaching its maximum during ejection, during period 2 $p_{LV}$ decreased, then increased, and decreased again, before finally increasing at the end of ejection. During IVR, $p_{LV}$ decreased in both studies. At $\tau = 0.385$, which signifies the opening of the MV and the end of IVR, $p_{LV}$ in the present study was 2.6 kPa for period 1 and 2.9 kPa for period 2, both of which are close to the measured value of $p_{LV} = 2.6$ kPa. Despite starting IVR from very different pressures, the trends and values of $p_{LV}$ at the end of IVR were comparable. Lastly, the calculated trends in $p_{LV}$ differed from the measurements during rapid filling. Rather than continuing to decrease towards a minimum pressure near $\tau = 0.5$, the calculated $p_{LV}$ increased shortly after the start of filling until it exceeded $p_{LA}$. This difference between the simulations and measurements led to a non-physiological backflow during filling, as will be discussed in Section 3.3. After exceeding $p_{LA}$, $p_{LV}$ decreased such that, during the latter part of filling, the calculated trend in $p_{LV}$ was comparable to the measurements, with the magnitude of $p_{LV}$ being approximately equal to the measured $p_{LA}$. For the second period, the maximum value of $p_{LV}$ during filling was lower than that during the first period and its location was shifted to a later time; however, backflow was still present.

3.2. Myocardium Stresses

Figure 3 is a plot of the temporal variations of the principal Cauchy stresses, denoted as $T_i, i = 1, 2, 3$, for two periods of the cardiac cycle; these stresses were calculated as the average of values obtained at eight nodes, located at a relative elevation of $z/h = 0.5$ in the reference geometry, and on either side of the centreline through the thickness of the myocardium at circumferential locations corresponding to the positive and negative $x$- and y-axes. It was found that the directions of the principal stresses changed with position and, following the application or removal of the active stresses, with time as well (data not shown). Even so, one can make some general observations concerning the relative orientations of these stresses. The orientation of $T_3$ was roughly perpendicular to the LV outer wall surface throughout the cardiac cycle, while $T_1$ and $T_2$ were perpendicular to each other in the plane tangent to the LV outer wall surface. The blood pressure force, which was applied normal to the LV inner wall surface, influenced the orientation of $T_1$ and $T_2$, particularly near this inner surface. During systole, the majority of the active stresses were applied in the fibre direction, and these active stresses influenced the orientation of $T_1$ and $T_2$ during that phase.
Fig. 3. Representative temporal variations of principal Cauchy stresses for two periods of the cardiac cycle.

The trends in $T_1$ and $T_2$ were consistent with the trend in $p_{LV}$ shown in Fig. 2a, that is, the stresses and pressure increased to maxima during rapid ejection, decreased gradually during the reminder of ejection, decreased more significantly during IVR and then fluctuated during filling. Differences between $T_1$ in the two periods are most apparent during ejection; during period 1, $T_1$ decreases monotonically from its maximum at early ejection, whereas, during period 2, $T_1$ has two peaks during ejection, before decreasing from the latter part of ejection through IVR.

In vivo measurements of stresses in the myocardium have not yet been obtained. However, such stresses have been estimated for canine LVs by DeAnda et al. [25] using Laplace’s Law. The trend of the temporal variation of the average wall stress calculated by DeAnda et al. [25] is comparable to the trend of $T_1$ found in the present study. This is to be expected because both the average wall stress and the maximum principal stress $T_1$ increase and decrease with corresponding increases and decreases in pressure.

3.3. Volume Change

This section describes the cyclic variation of the LV cavity volume $\Delta V_f$, normalized by the initial, end-diastolic LV cavity volume $V_{f,ED}$, which was calculated at $\tau = 0$. During IVC, $\Delta V_f/V_{f,ED}$ increased by 0.01 for period 1 and by 0.08 for period 2, before decreasing during ejection. Although no fluid entered or exited the LV cavity during IVC, these small volume changes are consistent with the slightly compressible form of the material model for blood. The change in $V_f$ during ejection can be characterized by the ejection fraction $EF$, defined as $EF = (V_{f,ED} - V_{f,ES})/V_{f,ED}$. During ejection, a small amount of blood exited the LV cavity, with $EF = 0.081$ for period 1 and 0.084 for period 2, both of which are lower than the physiological value for canine LVs, which is approximately 0.44 [2, 26]. During IVR, $\Delta V_f/V_{f,ED}$ increased by 0.01 for period 1 and decreased by 0.01 for period 2; both changes are sufficiently small not to cause much concern. At the start of filling, $\Delta V_f/V_{f,ED}$ increased rapidly for both periods to a maximum volume that was significantly larger than the original value of $\Delta V_f/V_{f,ED}$. The period of time over which this rapid increase in $\Delta V_f/V_{f,ED}$ occurred corresponded to the range of times over which $p_{LV}$ exceeded $p_{LA}$. Near $\tau = 0.5$, $\Delta V_f/V_{f,ED}$ reached a maximum, and then decreased, as the result of non-physiological backflow. During the remainder of filling, $\Delta V_f/V_{f,ED}$ increased and decreased two more times before reaching an end-diastolic value that was approximately 98% of the end-diastolic $\Delta V_f/V_{f,ED}$ at $\tau = 0$ for period 1 and 99% for period 2. Although the ejection fraction was sub-physiological and some backflow occurred during filling, the initial end-diastolic volume was essentially preserved at the end of each of the two periods that were simulated.
3.4. End Systole

The effective stress $T_e$ is a scalar stress calculated as

$$T_e = \sqrt{\frac{1}{2} \left[ (T_{xx} - T_{yy})^2 + (T_{xx} - T_{zz})^2 + (T_{yy} - T_{zz})^2 + 6 (T_{xy}^2 + T_{xz}^2 + T_{yz}^2) \right]},$$

where $T_{ij}$ are the components of the Cauchy stress tensor. Figure 4a shows the myocardium displacement and contours of the effective stress, while Fig. 4b shows pressure contours and velocity vector maps for the LV cavity; all properties are presented in the $y-z$ centre-plane at end systole ($\tau = 0.312$ and 1.312).

Both displacement magnitudes and effective stresses were found to decrease from period 1 to period 2, particularly near the apex. At the current contour resolution, pressure in the LV cavity appears to be fairly uniform for period 1, whereas it decreases from the equatorial plane to the apex for period 2. Due to the closing of the AV, $p_{LV}$ was large near the start of the LV outflow tract. The velocity vectors at end systole show noticeable differences between periods. For period 1, the velocities throughout the LV cavity were relatively small, whereas, for period 2, velocities of the order of 0.5 m/s were observed near the apex, suggesting that the LV cavity is deforming downward in this region. This difference between periods is consistent with the differences in pressure between periods.

Figure 5 contains plots of the transmural (from the exterior to the interior) and longitudinal (from the apex to the base) variations of the myocardium principal stretches and stresses at end systole, where the principal stretches $\lambda_i, i = 1, 2, 3$ are defined as the ratios of deformed and undeformed lengths. In the present study, these stretches were calculated with respect to the reference geometry at diastasis. The transmural variations of the principal stretches and stresses were calculated in the equatorial plane ($z/h = 0.67$ in the
Fig. 5. Representative transmural (a, c) and longitudinal (b, d) variations of the principal stretches (a, b) and principal stresses (c, d) in the myocardium at end systole for two periods; $\tau = 0.312$ and 1.312.

reference geometry). At each relative depth $t/t_{\text{max}}$ from the exterior surface in the deformed geometry, the average principal stretches and stresses were calculated as the averages of corresponding values obtained along the positive and negative $x$- and $y$-axes. Longitudinal variations of the stretches and stresses were calculated at a location halfway through the myocardium. At each relative height $c/c_{\text{max}}$ from the apex in the deformed geometry, average principal stretches and stresses were calculated as the averages of values obtained at circumferential locations corresponding to the positive and negative $x$- and $y$-axes.

The deformations in the myocardium increased from the exterior to the interior surface in a nearly-linear fashion (Fig. 5a). The corresponding principal stresses (Fig. 5c) also increased in magnitude from the exterior to the interior surfaces, but had nonlinear variations, in conformity with the form of the myocardium material model. Visible shifts in the stresses near $t/t_{\text{max}} = 0.83$ are a consequence of insufficient mesh resolution in this region, which would likely be removed by mesh refinement.

Longitudinally, the principal stretches were nearly constant away from the apex or the base; changes in $\lambda_1$, $\lambda_2$, and $\lambda_3$ near the basal plane are a consequence of the imposed fixed boundary conditions along this plane and its inner edge. Trends of the longitudinal variations of $T_2$ and $T_3$, shown in Fig. 5d, are consistent with trends of the corresponding stretches. $T_1$, which contains a significant amount of the active stresses representing the contraction of the muscle fibres, increased dramatically from the apex until it reached a maximum near $c/c_{\text{max}} = 0.50$ for period 1 and near $c/c_{\text{max}} = 0.67$ for period 2. In both cases, $T_1$ decreased from its maximum with increasing distance from the apex, before increasing slightly at the basal plane.
Fig. 6. Displacement and effective stress in the myocardium (a) and pressure and velocity vectors in the LV cavity (b) in the y–z centre-plane at end diastole for two periods; \( \tau = 1.000 \) (top row) and 2.000 (bottom row).

For systole, average Reynolds numbers were calculated for each period, based on the definitions proposed by Krittian et al. [13]. The Reynolds number is defined as \( \text{Re} = \rho f v D / \mu \), where, for systole, \( D = D_A V \) and \( v = \Delta V_{f,\text{eject}} / \left[ \left( 0.6 \Delta t_{\text{eject}} \right) \pi (D_A V / 2)^2 \right] \). For period 1, \( \text{Re} = 1749 \) and for period 2, \( \text{Re} = 2941 \). The Reynolds number calculated for period 2 happens to be comparable to the value 3431 calculated by Krittian et al. [13] for a human LV. It should be noted, however, that comparisons of Re for LVs of different species are not necessarily meaningful, because of differences in the sizes and pulsation rates of the LVs.

3.5. End Diastole

Figure 6a shows contour maps of the myocardium displacement magnitudes and effective stresses in the y–z centre-plane at end diastole for periods 1 and 2, whereas Fig. 6b shows the corresponding pressure contours and velocity vectors in the LV cavity. Displacement magnitudes for period 2 are more symmetric than for period 1. Stresses in the myocardium are noticeably smaller at end diastole than at end systole, because, at end diastole, the muscle fibres are fully relaxed and have no active stresses. Noticeable pressure differences are visible in Fig. 6b within the LV cavity, however, it should be noted that the magnitudes of these differences are relatively small (pressure contours have a resolution of 0.17 kPa). Higher velocities are visible in Fig. 6b for period 1 than for period 2, particularly near the apex.

Figure 7 contains plots of the transmural and longitudinal variations of myocardium principal stretches and stresses at end diastole. Both deformations and the magnitudes of the principal stresses increased with increasing distance from the exterior surface of the myocardium. The trends of the principal stretches are comparable to the trends found at end systole as are the trends for \( T_2 \) and \( T_3 \); however, the trends for \( T_1 \) differed between end systole and end diastole due to the presence of the active stresses during systole,
which have a functional form that is different from that of the passive stresses. Longitudinally, the principal stretches were nearly constant, with small variations near the apex and near the base. The corresponding values of $T_2$ and $T_3$ were also nearly constant, with $T_2$ increasing from the apex towards the midpoint and then decreasing near the base. $T_1$ increased substantially from the apex until it reached a maximum near $c/c_{\text{max}} = 0.6$ and then decreased towards the base. At end diastole, the maximum value of $T_1$ occurred at roughly the same value of $c/c_{\text{max}}$ for periods 1 and 2, whereas during systole there was a noticeable shift in the corresponding locations of the maximum value of $T_1$ between periods.

Diastolic $Re$ were calculated as $Re = \rho_f v D/\mu$, where $D = D_{\text{MV}}$ and $v = \Delta V_{f,\text{fill}}[(0.6\Delta t_{\text{fill}})\pi(D_{\text{MV}}/2)^2]$. $Re$ was found to be 193 and 359 for periods 1 and 2, respectively. For diastole, Krittian et al. [13] calculated $Re = 2288$ for a human LV, which is much larger than the values for the canine LV calculated in the present study, however, as with systole, the significance of this difference is unclear.

4. DISCUSSION

In the present work, we have successfully applied a direct fluid-structure interaction procedure to analyze an important problem in bioengineering. We simulated the interacting myocardium deformation and blood flow in the left ventricle during the entire cardiac cycle and reported, for the first time, detailed results of wall stresses and strains as well as flow characteristics. This work is not conclusive and has produced some
non-physiological results, which could be attributed to the several approximations we had to introduce in view of the scarcity of information required for constructing and validating the computational model and the enormous computer power and computing time that would be necessary for achieving a quasi-periodic solution. In particular, a limitation of this work is that it only extended for two cycles, which are clearly insufficient for a steady-state solution. Nevertheless, it is encouraging that the coupling of solid and fluid motions was done successfully, culminating in solution convergence, which may not be taken for granted in such types of simulations. In the following discussion, we will outline some of the problems we faced during this work and suggest some possible solutions to assist future researchers in making further progress on cardiac cycle simulation.

Spikes in the pressures were evident, particularly during the second period, near the times when a valve was closed. These spikes were caused by the instantaneous opening and closing of the valves, which represents an important limitation of our study. Modelling appropriately valve dynamics remains an open challenge in these types of simulations. Carmody et al. [27] performed FSI simulations of aortic valve dynamics using a two-step approach in which a LV model was used to obtain flow boundary conditions for a separate aortic valve model. Only recently [28] has a solid-only model of the mechanics of the LV been developed that included mitral valve motion. Additional work would be needed to include the motion of the aortic valve in these simulations, and even more to model the valve operation with FSI. For finite element simulations using an ALE approach to model the fluid, there is an issue with the closing of the valves in three-dimensions, related to the contact of the solid elements with each other at the tips of the valve leaflets. Even if the contact problem could be solved, including such valve motion with a sufficiently accurate solid model would require substantially more computational time; this would likely increase the computational resources required for such simulations beyond currently available levels, especially when considering the need for computing multiple cycles. An alternative method to address the spikes in the pressure would have been to couple the LV model with electric circuit models of the circulatory system both upstream and downstream of the LV. Although this has been done by previous researchers [11], in the present study attempts were made to match the pressures without introducing such coupling.

Blood flow in the LV was assumed to be laminar. However, the flow, in fact, may be transitional or turbulent during parts of the cardiac cycle, particularly during phases of deceleration. By modelling the flow as laminar, we are solving the full Navier–Stokes equations and the results of these simulations could be considered to be a coarse direct numerical simulation (DNS). Fluid mesh refinement would be required to resolve small scale motions.

One of the strengths of the present model was the inclusion of a validated myocardium material model that was transversely isotropic for both the passive and active parts, and for which the passive material properties were calculated for the species of interest. Recent studies of heart mechanics [11–13] that have included FSI effects have utilized simplified myocardium material models and/or passive material parameters adapted from species different from the ones considered in their studies. Watanabe et al. [11] used a material model that was similar to the one in the present study to perform simulations of the mechanics of the human LV, but chose their passive material parameter values to be proportional to values for rabbit LV, suggested by Lin and Yin [22]. Tang et al. [12] used a modified version of the Mooney-Rivlin model to model the passive and active parts of the myocardium rather than one more suitable for modelling ventricular mechanics. Krittian et al. [13] used a transversely-isotropic material with parameter values calculated for porcine LVs to model their human LV. All of these previous studies presented very few or no results for the myocardium.

One of the challenges in modelling the cardiac cycle using an ALE finite element approach concerns the isovolumetric phases, during which both the aortic and mitral valves are closed and the fluid mesh is deforming. In general, it is difficult to achieve numerical convergence for an incompressible fluid in an enclosed geometry undergoing large deformations due to FSI effects. To avoid this difficulty, we used a
slightly compressible fluid model. Previous studies either neglected the isovolumetric phases during their FSI simulations of the cardiac cycle [12], or did not discuss how they addressed this issue [13]. Although the introduction of a slight compressibility to blood led to small changes in volume during IVC and IVR, it allowed for the inclusion of these phases during cardiac cycle simulations, resulting in a more realistic simulation of the cardiac cycle than if these phases had been neglected.

5. CONCLUSIONS

Numerical simulations of myocardium motion and blood flow in the canine LV with FSI effects were successfully performed for two periods of the cardiac cycle. Calculated LV cavity pressures for the two periods were in good agreement with previous measurements over most of the cycle, but differed from them during the early part of filling, causing non-physiological backflow. Calculated average wall stresses over the two periods were consistent with estimates made by DeAnda et al. [25]. Volume changes during the isovolumetric phases were small and could be plausibly attributed to the slightly compressible material model used to ensure convergence. Some of the present results deviated from physiological values: the calculated ejection fractions were sub-physiological and small backflow was observed during early filling. Even so, the end-diastolic volumes at the ends of the two periods were nearly equal to the initial value at the start of the cardiac cycle simulations, which is a positive indication that the present model captures LV mechanics with considerable realism.

Temporal variations of the displacements and stresses in the myocardium were presented in the form of contour plots at end systole and end diastole. Stresses were found to be much larger during systole than during diastole, in conformity with the expected effects of the contraction of the muscle fibres during ejection. Temporal variations of the LV cavity pressure and the blood velocities were presented for the same times during the cycles as the solid results. Pressure differences in the LV cavity were visible in the contour plots for ejection for period 2 only, whereas, during filling, pressure differences were visible in the contour plots for both periods. These pressure differences emphasize the importance of performing FSI simulations of the LV operation instead of solid-only simulations of myocardium mechanics for which uniform pressures are generally assumed as boundary conditions on the inner surface of the LV.

Detailed spatial variations of the myocardium principal stretches and stresses were presented at end systole and end diastole.

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