

Study of the mitral motion in healthy and IMR patients

T. Company Messa¹, M. Iglesias Blanco¹, J. Martinez Gonzalez¹, and M. Martinez Mas¹

¹BCN MedTech, Universitat Pompeu Fabra, Roc Boronat, 138 - 08018 Barcelona Barcelona

ABSTRACT

Aims

By modelling the left part of the heart, the aim of the present paper is to compare the haemodynamics of a healthy patient against a patient who suffers from Ischemic Mitral Regurgitation. Attention is focused on the mitral valve and on calculating the regurgitant volume, a strong assessment parameter of this disease severity.

Materials and Methods

4D Mitral models provided by the University of Pennsylvania were joined to the remaining left heart components, the left atrium, left ventricle, and the aorta artery. Three 3D different models were developed using the softwares Meshmixer and Meshlab, two for systole and one for diastole. Blood was assumed to be Newtonian, incompressible and isothermal, therefore its behavior was described by Navier-Stokes equations. The boundary conditions for the ventricular systole models were defined as velocity inlet for the pulmonary veins, and pressure outlet in the aorta, whereas for the ventricular diastole only a velocity inlet was applied, and the aorta was considered to have wall-behavior. Due to the difficulty that carries the geometry coupling, models were considered motionless and isolated with respect to the others. All the simulations were run with ANSYS (Fluent) and Paraview was the chosen software for the post-processing.

Results

Three coupled models are successfully achieved with quite realistic physiological behavior (especially in systole) of the blood thought the left heart chambers. Due to the simplifications imposed in the model, the developed mathematical approach for the regurgitant volume calculations were not enough to determine the severity of the disease with this parameter.

Key words: Mitral Regurgitation – 3D models – Regurgitant Volume – Mitral valve – Blood hemodynamics – Fluid Analysis

1. Introduction

The mitral valve (MV), also known as bicuspid, is a valve with two flaps in the heart, that lies between the left atrium (LA) and the left ventricle (LV). In normal conditions, blood flows through an opened mitral valve during diastole and leaves the ventricle thanks to a contraction during ventricular systole, when the mitral valve remains closed. These changes can occur as a consequence of pressure differences.

Mitral insufficiency, which is the second most common valve disease in Europe (affecting approximately 31 % population¹), causes a regurgitation or backflow of blood due to the incomplete closure of the valve. The consequence is a volume overload in the ventricle, submitting the heart to a higher atrial contraction for expel the excess of blood leading to heart dilation. The most common cause of this disease is myocardial infarction, hence, this project will be focused on mitral regurgitation caused by ischemias.

The main challenges and limitations in the MV modelling are the model uncertainties, the boundary conditions chosen and doing a patient specific model, since it is difficult to estimate all the proper parameters for in vivo data. Although the necessary information available for these projects is limited due to the fact that medical imaging cannot provide information about mechanical properties. The numerical simulations allow to test different scenarios and can give a better access to the hemodynamics, which in vivo data cannot. In previous works, it has been studied aortic valve stenosis²³⁴, computational models of fluid - tissue interactions⁵, coupled MV - LV models with FSI⁶ or mitral valve stenosis studies.⁷

Modelling this disease as it is proposed in this paper, has not been done yet, for this reason, we aim to present a model for Ischemic Mitral Regurgitation. The resulting dynamics (systolic and diastolic) are studied in physiological and pathological conditions by means of a static model (movement not considered), to approach the specific mechanical alterations suffered by the heart more in detail. The hemodynamics of the left heart side of a healthy and IMR patient are compared and a study of the severity degree is carried out in order to recommend surgical intervention depending on the regurgitant volume (RV).

The paper is structured as follows. Details for the different simulations and their possible outcomes are posed on materials and methods. Then, the results for all the situations considered are presented, validated and commented. Finally we allocate a section for limitations and conclusions of our work.

2. Materials and methods

In order to achieve the simulation objectives, two domains have been considered. The structural, focused on MV¹ (three different conformations considered), LV² and LA;³, and the fluidic, which consists of a blood flow that goes from the pulmonary veins towards the aorta. Attention is especially paid in systole since it is the cardiac cycle moment in which the volume overload and the consequences of the disease are mostly emphasized.

¹Provided by *University of Pennsylvania*.

²Provided by *Barcelona Super Computing Center*

³Provided by *Aalst Hospital* (Belgium) and improved by Antonia Alomar - Bachelor Student of Biomedical Engineering, *Universitat Pompeu Fabra*.

2.1. Left Heart Geometry

In order to study the hemodynamics of both, healthy and IMR cases, the left heart parts, consisting of MV, LV (Fig (1), and LA (Fig (2)), should be coupled.

Before starting the models, the right ventricle was removed with *Meshmixer*⁴ software because it does not play a major role for the wondered fluid simulations.

Once the LV was isolated, two options were considered for the global coupling. Joining the LA first and further the MV or the other way round. Finally the coupling was done joining the LA with MV and then the LV. This procedures were done three times with a different MV geometry (Fig (3)) (systolic for healthy and IMR, and diastolic). The coupled model for the healthy MV was especially challenging, since the MV provided had meshing problems by itself. Consequently we were forced to mimic its geometry by modifying the diseased one.

As soon as the structures were properly joined, the addition of the aorta was carried out by adding a cylinder with a flat surface. This procedure was already done with the pulmonary veins (PV) in the LA. Smoothing filters, such as *Taubin Smooth* and remeshing tools from *Meshmixer* and *MeshLab*⁵ softwares have been applied to improve the meshes. To reduce errors in the meshes, the intersecting faces were removed and the holes were closed with *MeshLab*. Finally, in order to perform the simulations of the 3D mesh of the whole coupling (Fig (A.1)) (*Gmsh*⁶ software) was the decisive step.

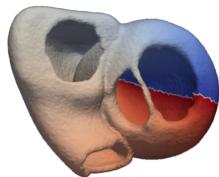


Fig. 1: **Whole Ventriles.** Concerning to the ventriles, it is possible to appreciate the holes that belong to Mitral, Aortic and Triscuspid valves. This segmentation was used to create the geometry by deleting its right part (right ventricle).

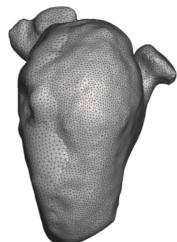


Fig. 2: **Left Atrium.** The atrium is unprocessed in this image, for the coupling, an improvement with remeshing and smoothing tools was done and cylinders were added.

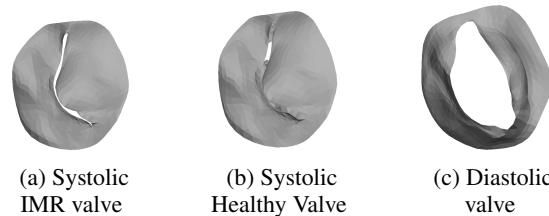


Fig. 3: **Valve Frames.** The IMR valve is a very thin layer segmented from UltraSound(US) images and provided by UPenn. For the healthy case, it has to be taken into account that ideal cases do not exist (i.e., always espaces fluid) so, this behavior was mimicked by closing the IMR valve and turning it into a healthy case.

2.2. Numerical Method

2.2.1. Structural Domain

As it has been said, cylinders were added in arteries and veins, this structures are desirable to be at the main entrances of any model with strong gradients and wall shear stresses due to viscous flows as blood, therefore, they are extremely important in the cardiovascular biomechanics to stabilize the flux.

Another crucial point that has to be considered are the physiological sizes. As mentioned above, the meshes were provided from different institutions, hence, they belong to different patients. In order to frame the simulations in a realistic context, the left heart geometry obtained is assumed to belong to a woman in a normal range of 100 mm of heart height. All the specifications were given by *Hospital del Mar* and *Organs and Systems Modelling* lessons.

Structure	Diameter Range (mm)
Pulmonary Veins	10-25
Mitral Valve	28-42
Aortic Valve	18 - 23

Table 1: Vessels diameter.

Structure	Size (mm)
LA	45x33
LV	50x40

Table 2: Left Heart Sizes.

2.2.2. Fluidic Domain

Blood was assumed to be newtonian, incompressible and isothermal, therefore velocity \mathbf{u} and pressure p are governed by Navier-Stokes equations, which in non - dimensional form are:

$$\frac{\partial(\mathbf{u})}{\partial(t)} + \mathbf{u} \cdot \nabla(\mathbf{u}) = -\nabla(p) + \frac{1}{Re} \nabla^2 \cdot \mathbf{u} + \mathbf{f} \quad \nabla \cdot \mathbf{u} = 0 \quad (1)$$

The assumption that blood behaves as a Newtonian fluid is consistent since non-Newtonian features become relevant only in vessels with small diameters. The diameter of the RBC (Red Blood Cells), the responsible of changes in the viscosity, can be neglected and we can approximate the viscosity of the blood to the viscosity of the plasma. Based on these assumptions, the blood viscosity is between $3.5 * 10^{-3}$ and $4 * 10^{-3}$

⁴Meshmixer 3.5 - <http://www.meshmixer.com> .

⁵MeshLab 2016 - <http://www.meshlab.net/> .

⁶Gmsh 4.1.5 - <http://gmsh.info> .

$\text{kg}/(\text{m} * \text{s})$ for a consideration an ejection fraction $\text{EF} = 60\%$. The blood density is assumed between 1050 and 1060 kg/m^3 .

The Boundary Conditions for systole are the same for healthy and IMR cases, this assumption is also consistent since the case of IMR considered, is assumed to be a chronic case (i.e., the heart has had time to adapt and overcome volume and pressure changes), thus, the ejection fraction is maintained. The no-slip boundary condition of the velocity at the fluid/structure interfaces is imposed by means of the body force in Eq. (1). The inlet velocity profile of the pulmonary veins follows the Poiseuille's law, the value of the velocity in the wall is around 0 , and its maximum will be in the center. These specifications are implemented as a curve as shown in **Fig (A.2)**. The profile of approximate pressures at the exit of the aorta was implemented using *CircAdapt* software.⁷

The BC's for diastole were defined following the same assumptions than in systole, but implemented based on characteristic diastolic functions (**Fig (A.2)**), velocity inlets in the pulmonary veins and a wall behavior in the aortic valve.

The regurgitant volume is the most representative parameter related with the severity of the IMR. This regurgitant volume is obtained with US-Doppler, but it needs an approximation of the section which its backflow passes through and thus the results obtained are not very precise, therefore being able to calculate the regurgitant volume from the model could lead to meaningful and beneficial changes in the clinical context.

The RV can be obtained by performing a series of calculations with Paraview⁸, Microsoft Excel⁹ and Matlab¹⁰ softwares. The main formulas for calculating the regurgitant volume are:

$$\text{ASV} = \int_0^{0.38} f_{\text{systole}}(x)dx \quad (2)$$

$$\text{MDV} = \int_0^{0.45} f_{\text{diastole}}(x)dx \quad (3)$$

$$\text{MDV} - \text{ASV} = \text{RV} \quad (4)$$

Where MDV stands for Mitral Diastolic Volume, ASV Aortic Systolic Volume (from the Left Ventricle Outflow Tract) and RV Regurgitant Volume. The $f_{\text{systole}}(x)$ and $f_{\text{diastole}}(x)$ functions are the flow (mm^3/s) approximations over time in the aorta and MV respectively.

Paraview integrates the values of velocity in a determined slice (in this case, MV and TSVI slices) over all simulation time steps. This data is exported to Microsoft Excel in order to obtain an accurate function of flux over time. Using the approximation function of a polynomial equation, the integral (**Eq.(2)**, **Eq.(3)**) is computed over time with Matlab Software. By doing this integration of the flow over time, the total volume that passes through that slice is calculated.

⁷CircAdapt Simulator v1.1.0 - <http://www.circadapt.org/home>

⁸Paraview 5.6.0 - <https://www.paraview.org>

⁹Microsoft Excel 360 - <https://products.office.com/es-es>

¹⁰Matlab 2018b - <https://es.mathworks.com>

2.3. Fluid - Structure Coupling

2.3.1. Simulation

The meshing was coupled using the mesh generators *Meshmixer*[®], *MeshLab*[®] and *Gmsh*[®] as explained in 2.1.1 sections, it must be as soft and regular as possible in order to obtain the right interpolations. Once achieved, fluid simulation with *Ansys (Fluent)*¹¹ were carried out, this software takes into account the Navier-Stokes law explained above together with the specifications of the rest of the assumptions. Finally, for the post-processing and visualization ParaView software was used.

2.3.2. Validation

Since the literature of this field is rather scarce, the validation is carried out with data obtained with US-Doppler that Dr. Lluís Molina Ferragut, cardiologist of the Hospital del Mar, provided us, together with useful physiology information and modelling advices.

Finally, the flow behavior is compared in our patient with IMR to the healthy one and see which are the main hemodynamic differences. The calculation of the regurgitation volume (RV) is done in order to determine a threshold value of RV for which the disease is in its limit of compensation and chronicity, thus surgery would be required.

3. Results

Despite the difficulty of having to build models based on meshes from different patients, we have obtained several consistent results, without dismissing, however, that all the following results are based on static models.

The principal affected part of the cardiac cycle in our framework (IMR disease) is the systole, because it is characterized by the maximum pressure load, thus, causing the greatest consequences in the MV valve closure. So, here lies the reason why we have developed two different models for systole and only one for dyastole.

The following sections give detailed explanations on what is happening in every simulation and every part of the geometry. Pictures of selected time steps are added, besides, more images in different time steps can be found in the appendix section.

3.1. Left Atrium Hemodynamics

3.1.1. Systole

The blood inflow in the left atrium from the pulmonary veins is analyzed in the present section. **Fig (4)**, shows the different velocities that characterize the blood circulation in the atrium for a mid time step (see **Fig (B.1)** for visualizing a wider vision of the systolic cycle). During early systole the flow starts accelerating from 0.15 m/s to highest values around 0.35 m/s . This part is aimed to explain how the flow circulates through the atrium, the abnormalities that IMR disease may cause, such as the characteristic regurgitation, are further developed in section 3.3 (MV Hemodynamics).

¹¹Ansys 2019 R1 - <https://www.ansys.com/products/fluids/ansys-fluent>

There are three distinguishable zones of higher velocities that correspond to the pulmonary veins, the fourth is located in behind the clip view in **Fig (4)**. In the late systole, the higher velocity values are located in the mitral valve. In addition, it must be mentioned that the velocities inside the atrium which does not correspond to the pulmonary veins or near the MV, are very close to 0 in whichever time step.

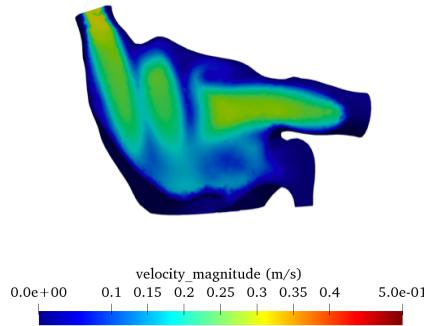


Fig. 4: Atrium systolic velocities. Image from the 26th time step of the simulation, the highest velocity values are appreciable in these moment as opposed to other time steps.

3.1.2. Diastole

Diastole hemodynamics are mostly determined by the applied boundary conditions in the pulmonary veins. Therefore, at the beginning of the diastole ($T=0$) the model presents very low velocities in all the atrium. As times goes by, they increase reflecting this changes in the atrium cavity, and gradually filling it up, until they reach, at several time steps, its maximum velocity 0.5m/s.

Since it is not possible to show the entire simulations, in **Fig (B.7)** it is possible to see diastole simulations that were found interesting to point out in the discussion section.

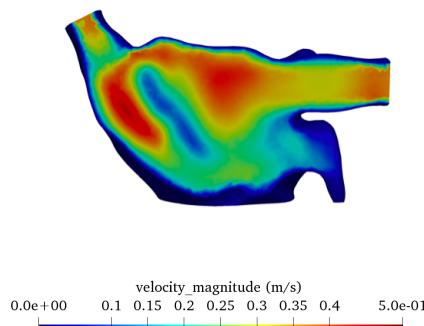


Fig. 5: Atrium diastolic velocities. Image from the 26th time step of the simulation, values approximately around 0.45 m/s. The velocity increases as time progresses (see **Fig (B.2)**).

3.2. Left Ventricle Hemodynamics

3.2.1. Systole

Regarding the aim of the paper, analyzing how the mitral valve affects the systolic ventricular hemodynamics becomes the fundamental part.

Simulation begins with low velocity values and as time progresses, it starts increasing in the whole ventricle. At the mid steps velocity in the aorta reaches its maximum with a peak of 1.2 m/s approximately. Finally, it is shown how the velocity magnitude decreases from the step 26 right up to the end, since the colormap starts showing cold-colored values.

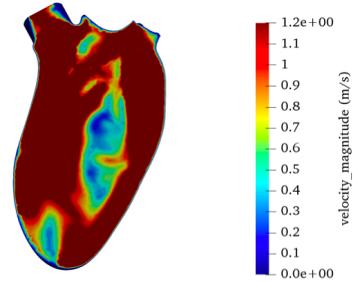


Fig. 6: Ventricle systolic velocities. Image from the 26th time step of the simulation, values around 1.2 m/s in almost all the ventricle. This time step shows the approximately peak velocity of the simulation, since from now on it starts to decrease (see **Fig (B.3)**).

3.2.2. Diastole

Following the idea of the previous section and considering that in diastole the mitral valve is opened, the applied boundary conditions at the PVs has its corresponding effect in both atrium and ventricle. Hence, early simulation steps show velocities quite low except for a small acceleration in the mitral valve area.

Moving forward in time, the velocity through the MV increases, and therefore in a coherent way, a velocity gain can also be seen in the ventricle. 0.33 m/s is the value for the maximum velocity reached, although the aorta maintains zero velocity value during all the simulation.

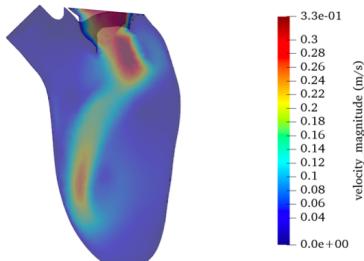


Fig. 7: Ventricle diastolic velocities. Image from the 26th time step of the simulation, only punctual values of 0.33 m/s appear on the image. This time step shows the approximately peak velocity of the simulation, since from now on it starts to decrease (see **Fig (B.4)**).

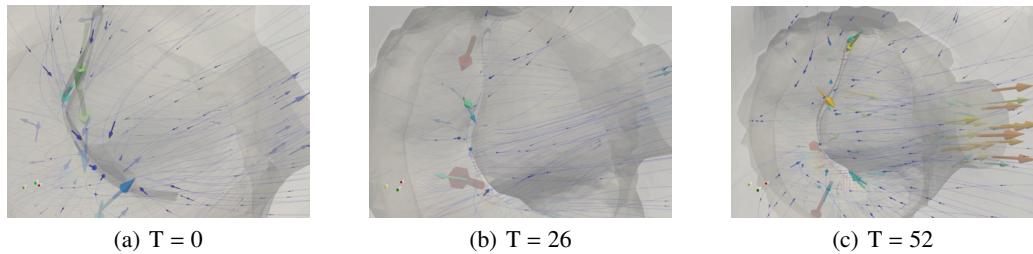


Fig. 8: Ischemic Mitral Valve close up. Backflow arrows appear in few time steps with different velocities.

3.3. Mitral Valve Hemodynamics

It is important to take into consideration that MV hemodynamics are going to be accounted only for systole. Since the same diastolic simulation is carried out without making any valve distinction, diastole is only aimed for the ventricular volume inflow calculations.

3.3.1. IMR

Significant flow changes can be observed in the mid time steps (**Fig (8)**) where velocities are appreciated above mitral valve. The outlet velocities in the aorta are around 1.2 m/s in mid systole. In contrast, they present lower values in early or late time steps (around 0.6 m/s).

Fig (8) also shows that when looking at the velocity vector field, upward arrows exhibiting quite large velocity values thought the mitral valve can be differentiable.

3.3.2. Healthy

It is possible to appreciate that for the healthy case the mitral valve is closed and all the flux goes through the aorta following a parabolic-shaped path; low velocities at the first simulation time steps and maximum peak velocity at mid simulation followed by a velocity decrease at the end. An absence of regurgitant volume it is also appreciable.

The following images display the different behaviors when looking at the whole geometry for the different valve situations. Worth mentioning that the atrium have the same dynamics as in **Fig (4)** but due to scaling constraints they are not represented in these images.

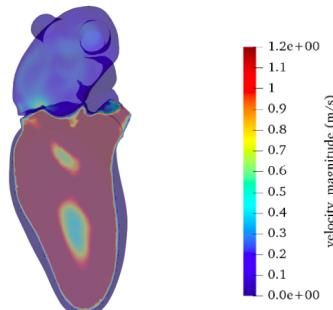


Fig. 9: Whole Healthy Heart Hemodynamics. The atrium flow patterns more visible than in the following case. Ventricular hemodynamics are the same as described in **Fig (6)**.

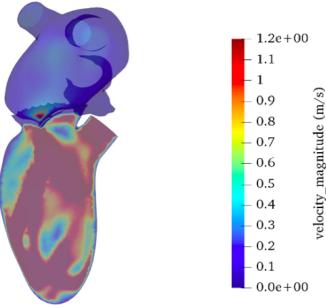


Fig. 10: Whole IMR Heart Hemodynamics. Visible blood acceleration in the MV zone although its direction is not visible.

3.4. Regurgitant Volume

Following the calculations explained in Materials and Methods section, flux over time plots were obtained automatically integrating the velocity components in a mitral valve slice in diastole and in an aortic slice in systole (**Fig (B.8)**). Afterwards, the area under this curves was computed by integrating them over time and the results are the following :

$$\begin{aligned} MDV &= 8.97mL \\ ASV &= 30mL \\ RV &= -21.03mL \end{aligned}$$

These results are further developed in the discussion section.

4. Discussion

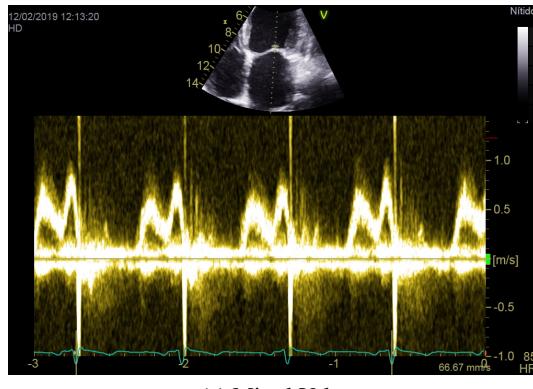
4.1. Results interpretation and Validation

The validations of these models, systole and diastole , will be done with PW Doppler images, provided by Hospital del Mar, paying special attention to velocities in the aorta and the mitral valve.

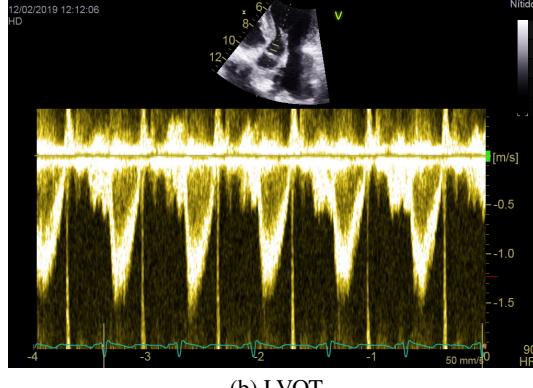
4.1.1. Healthy Systole

It can be appreciated that the velocity magnitude obtained are the expected, since they accurately mimic the physiological behavior shown in the PW Doppler images.

Hemodynamics close to the aorta are correlated with the BC imposed, considering that at the beginning exhibits low values, which are gradually increased reaching a peak velocity and decreasing at the end. The maximum peak velocity in the Doppler corresponding to the LVOT (Left Ventricle Outflow Tract) (**Fig (11)**), is around -1.5 m/s since it is going contrary to the direction of the transducer, and the value obtained in the



(a) Mitral Valve



(b) LVOT

Fig. 11: **Doppler Validation.** PW Doppler of a Healthy Patient.

model its value is 1.2 m/s, a quite good approximation.

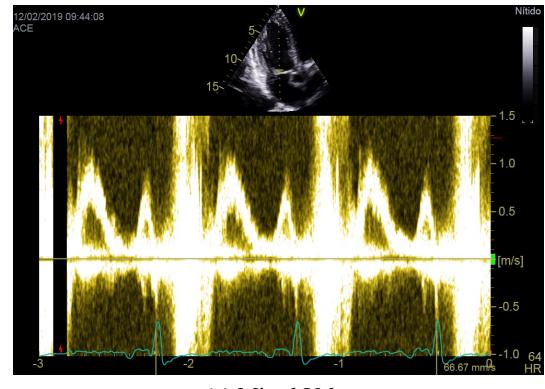
It is commonly accepted that during the systolic period, *a priori*, there is no flow through the MV. Nevertheless, a quick bidirectional velocity peak can be appreciated in the supplied Doppler (**Fig (11)**). This small regurgitant peak appears because even real healthy cases have non-ideal behaviors. However, this level of detail is not reached in our models.

Finally another remarkable aspect are the high velocities below the mitral valve. They are consequence of the imperfect leaflets closure. It must be taken into account that any available mitral valve was provided, thus, several modifications in the diseased one were performed to obtain the desired healthy geometry.

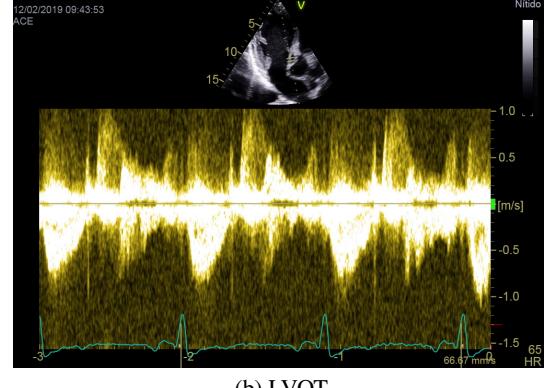
4.1.2. IMR Systole

Owing to the physiological reduced mobility, the posterior leaflet is not strongly affected by the insufficiency. In contrast, the anterior, is the one that more determines severity of IMR. In this case, there is a prolapse that causes the not desired closure those yielding an abnormal reflux from the ventricle through the mitral channel.

For achieving a deeper comprehension of this situation, the behaviour of the MV and AA areas in our model were compared and contrasted with a given Doppler from a person suffering from IMR (**Fig (12)**).



(a) Mitral Valve



(b) LVOT

Fig. 12: **Doppler Validation.** PW Doppler of an IMR Patient.

Concerning to the aortic velocities, a maximum peak of 1 m/s can be observable in the provided Doppler (**Fig (12)**). In this aspect, the designed models present a realistic physiological behaviour since they maximum velocities of 1.2 m/s. It is true, however, that if we compare this results with the obtained in the previous case this peak velocity values are less sustained in time.

4.1.3. Diastole

In a physiologic diastole two parts can be distinguished: the early filling, corresponding to the passive filling and to the E wave of the PW Doppler figures, and the systolic filling, as a result of the contraction of the LA and which corresponds to the A wave in a PW Doppler. Without going into details we can consider that this two waves oscillate between 0.75 and 1 m/s. If we compare this data, with our models we can affirm that, even though the restrictions of a motionless model, velocities of 0.5 m/s have been successfully reached.

Since in this model, heart dynamics are not being considered, diastole can occur exclusively due to the blood flow of the velocity inlet imposed in the pulmonary veins.

Although the presence of limitations, coherent results have been registered. For the early simulation steps, blood velocities are almost zero, but they start increasing, reaching maximum velocities of 0.5 m/s and gradually filling up all chambers. Concerning to the aorta, its behaviour is also physiologically consistent since the velocity in its area is null for all time steps.

Despite these significant results it must be highlighted a point in the left atrium where high velocities appear without corresponding to any plausible physiological behaviour ((textbf{Fig (B.7)})). Consequently, we attribute this to a mesh abnormality that was not possible to detect by the mesh post-processing softwares.

4.2. Severity Determination of Ischemic Mitral Regurgitation

The physiological values seen in **Table(3)** were provided by Dr Molina from Hospital del Mar. By comparing both tables we can conclude that we could achieve the desired values for systole which were obtained from the flux through the aorta of the IMR patient model so, this leads us to believe that the proposed calculations are correct.

Regarding to the diastole values it is also possible to see that the obtained results are considerably far from the expected, consequently, we could not mathematically calculate the regurgitant volume from our data. However, it must be said that this results are coherent with our simulation results. It must be considered, that this volume values come from computing the integral over time of the flux across the mitral valve during diastole. Therefore, since the flux values strongly depend on the velocity values, and we only could reach maximum velocities of 0.5 m/s in several time steps so it was foreseeable to obtain such low values.

This results were rather predictable since we are mixing two independent simulations (diastole and systole). But, even being aware of this limitation, it was decided to simulate diastole and use its results because it was the best possible approach. The option of integrating the flux in the mitral valve during systole was rejected being that, no way for isolate and only integrate the velocities corresponding to the backflow, was found.

Furthermore, the regurgitation did not appear in all time steps, only in few, for this reason, the patient, that was assumed to be in a normal range, has coherent regurgitation results (concerning to velocities), thus, the Boundary Conditions assumed gain even more meaning.

Phase	Physiological (mL)	Obtained (mL)
Systole	12-42	30
Diastole	46-106	8.97

Table 3: **Volumes.** Physiological vs. Obtained

4.3. Limitations

The greatest limitation of this project and its consequences have been commented along the paper: a dynamic mesh would have been the most accurate option. However, due to a lack of time this possibility was not contemplated. As a consequence, because of this simplification interesting parameters such as pressure were not possible to be assessed.

The second strong limitation is the isolation between simulations. Considering future improvements it will be more clinically applicable to achieve a full cardiac cycle simulation.

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Appendix A: Materials and Methods.

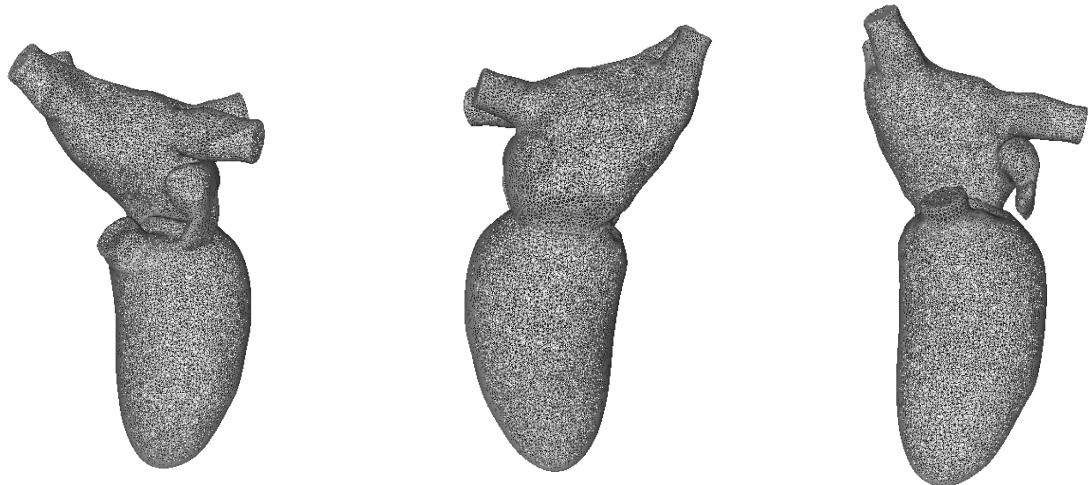
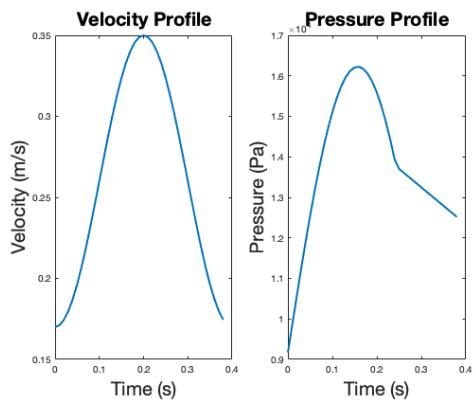
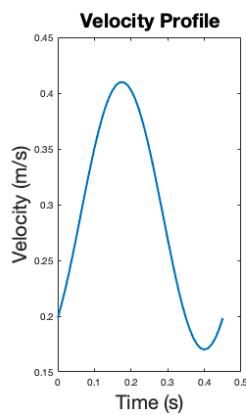


Fig. A.1: **Left heart geometry.** Coupled done by joining que LA with the MV, which is duplicated and added to the LV. Finally, both layers of the valve are connected in order to let flow go through them.



(a) Systolic pressure and velocity profiles



(b) Diastolic pressure profile

Fig. A.2: **Boundary Conditions.** Systolic and Diastolic velocities taken from a function provided by Jordi Mill and Andy Luis Olivares, lecturers of Organs and Systems Modelling. The systolic pressure profile was a function created taking pressure values in the aortic valve. The pressure values are in Pascals since is the unit with the simulation program Fluent works.

Appendix B: Results.

In this section are found more images concerning to the results analyzed. All of them are chosen from 3 time steps, in order to visualize what is happening in early, mid and late instants of the simulations.

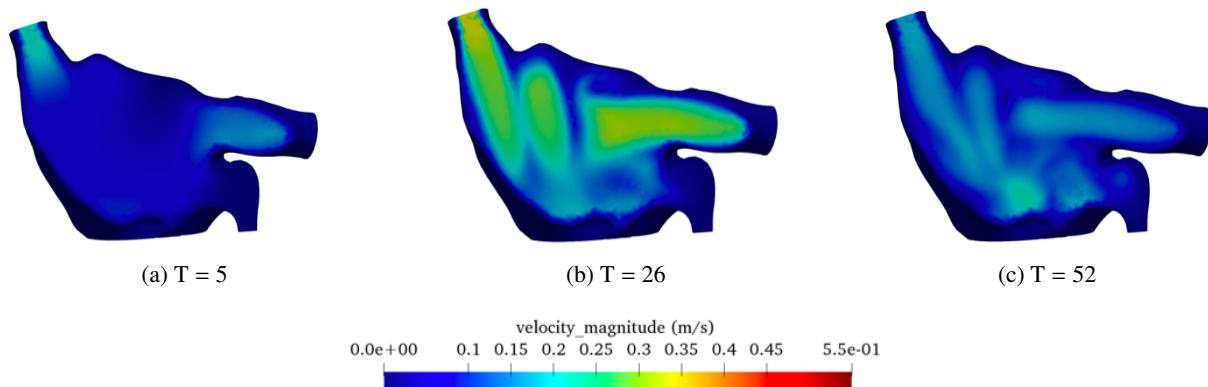


Fig. B.1: Atrium velocities. Systole.

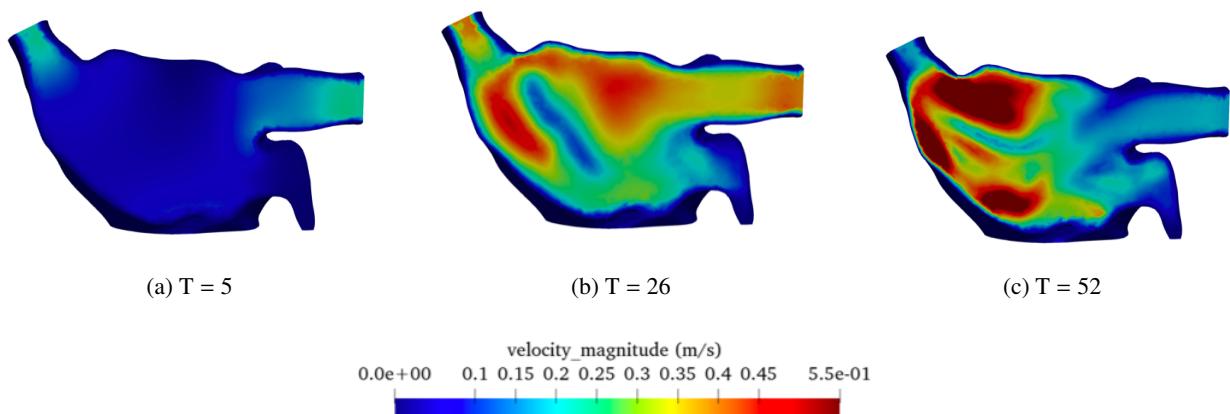


Fig. B.2: Atrium velocities. Diastole.

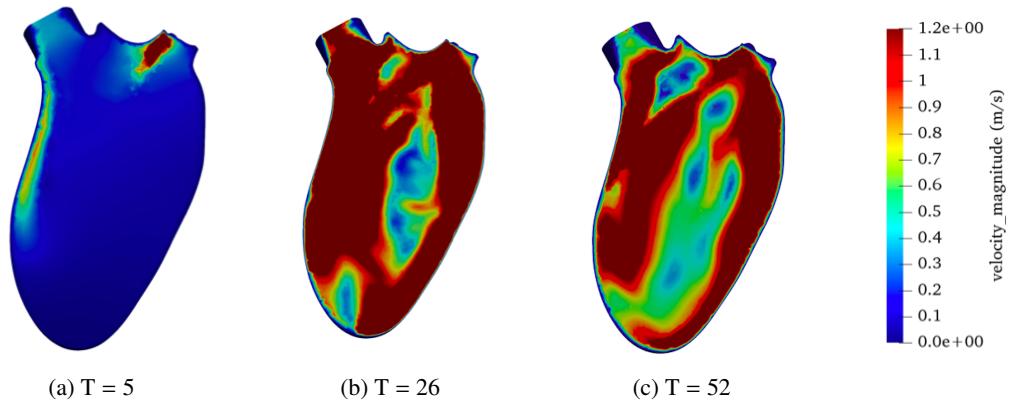


Fig. B.3: Ventricle velocities. Systole.

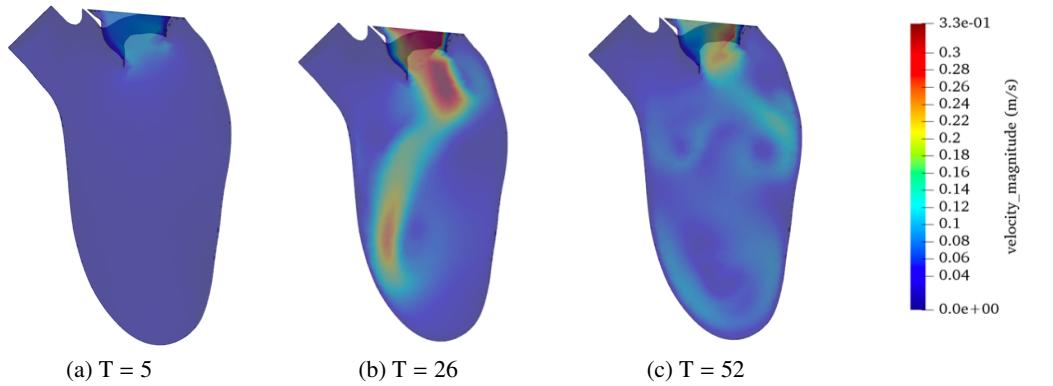


Fig. B.4: **Ventricle velocities.** Diastole.

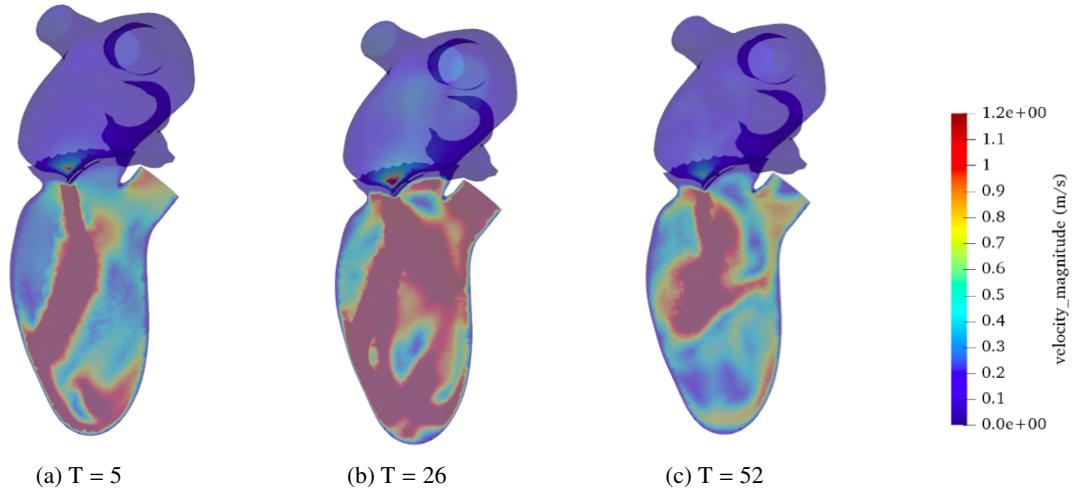


Fig. B.5: **Whole left heart.** Systolic heart with IMR valve.

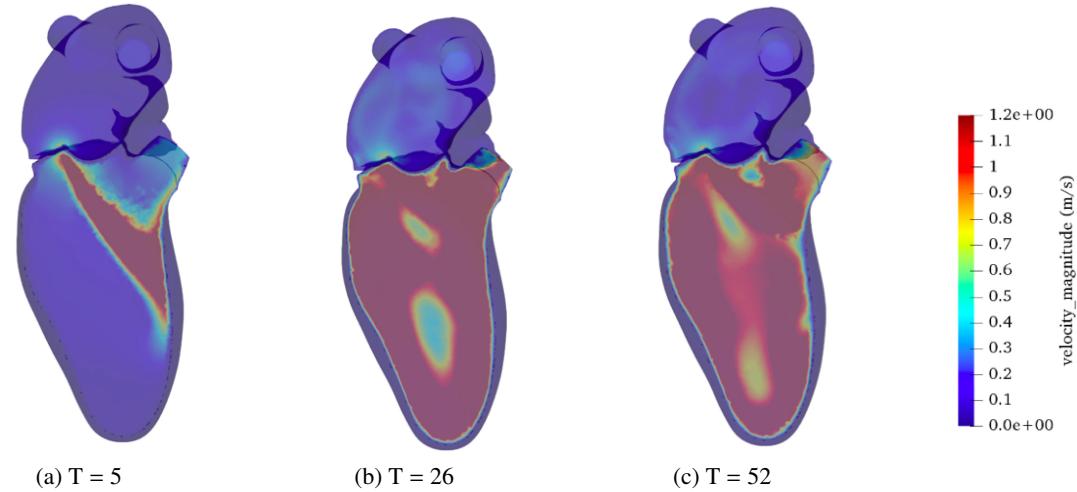


Fig. B.6: **Whole left heart.** Systolic heart with healthy valve.

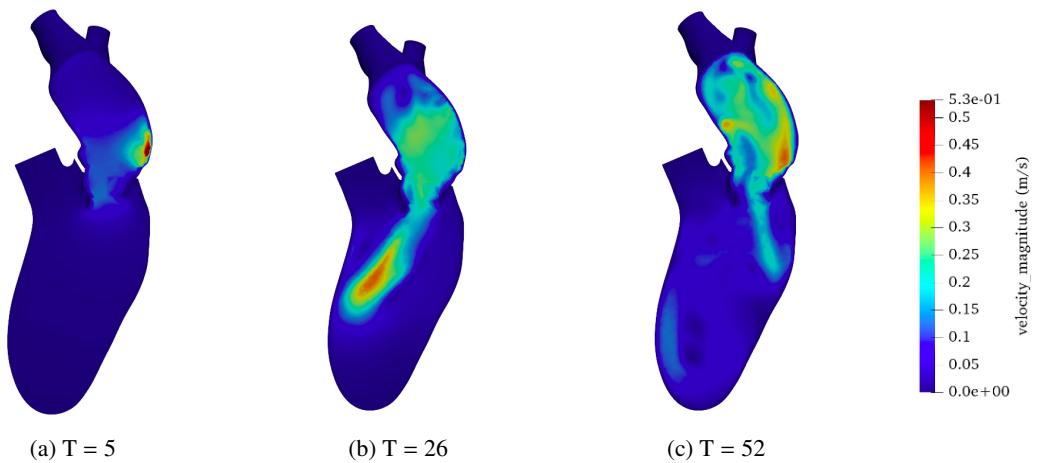


Fig. B.7: Whole left heart. Diastolic heart. Inserted picture in order to show a mesh abnormality in the left atrium, further explained in discussion section.

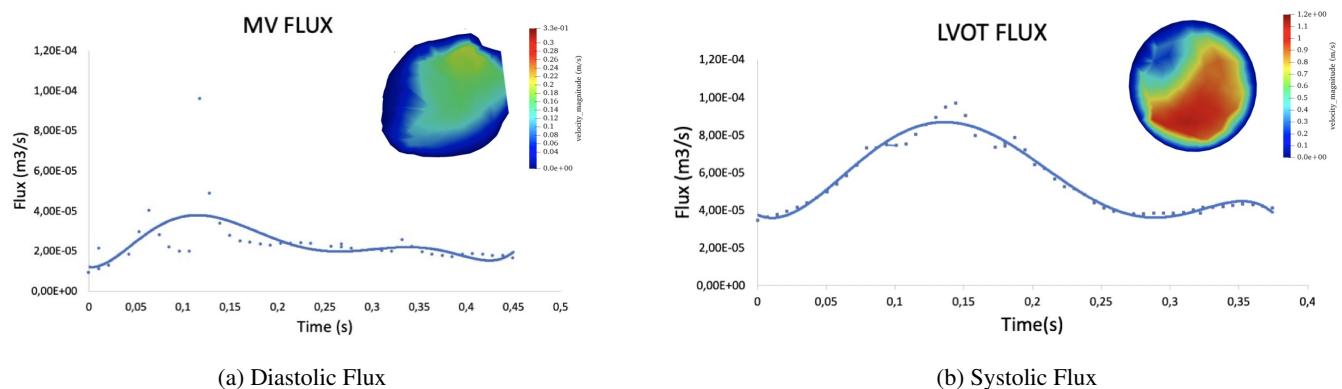


Fig. B.8: Flux vs. Time curves. In these plots it is possible to see a cross- section of the slices done for integrating the velocities. The discontinuous line represents the real data while the continuous one is the approximated function.