The contraction of the heart muscle combines with the opening and closing of the cardiac valves to generate a complex flow in the heart. Predicting this flow presents a significant challenge for computational models, a challenge that Meschini et al. (J. Fluid Mech., vol. 834, 2018, pp. 271–307) tackle head-on by simulating not only the flow in a modelled left ventricle of the heart, but also the coupled dynamics of the mitral valve. The model is validated against a well-designed companion experiment and the authors then proceed to examine the effects of cardiac and valvular dysfunction, as well as prosthetic valves, on ventricular haemodynamics. The insights provided by this study extend from the functional morphology of the left ventricle to the implications of the choice of valve implant on ventricular function.

Key words: biomedical flows, blood flow

1. Introduction

The mammalian heart has been sculpted by millions of years of evolution into a flow pump par excellence. During the typical lifetime of a human, the heart will beat over three billion times and pump enough blood to fill over 60 Olympic-sized swimming pools. Furthermore, each beat will raise the hydrostatic head of the blood by over 1.5 m, sufficient to overcome the flow resistance of the billions of blood vessels in the body, and provide adequate perfusion to all the tissues of the body.

While it is the heart muscle that powers this astonishing mechanical performance, the movement of blood through the four chambers of the heart is controlled by the cardiac valves. The two-way interaction between the blood flow and the valve leaflets is therefore crucial to the functioning of the heart, and has been the subject of numerous studies (see Yoganathan, He & Jones 2004; Sotiropoulos, Le & Gilmanov 2016). The first known scientific study of cardiac haemodynamics was carried out by none other than Leonardo da Vinci, who used grass seeds to visualize the flow in glass casts of animal aortas (Robicsek 1991). In the modern era, a variety of in vivo and in vitro approaches have been used to study intra-cardiac and valvular haemodynamics (Yoganathan et al. 2004), but the limitations inherent to such experiments have left many questions unanswered.

The continuously increasing processing power of computers has opened the door for computational modelling of cardiac haemodynamics, but such modelling is also not...
without its challenges (Mittal et al. 2016). Chief among these are the requirement to (i) accurately simulate a highly unsteady, turbulent flow in a geometrically complex, significantly deforming three-dimensional domain, (ii) model fluid–structure interaction between the blood flow and the valve leaflets, and (iii) extract useful insights from the extremely large datasets that emerge from such models. Meschini et al. (2018, hereafter MTQV) tackle all of these challenges and provide a powerful demonstration of the potential of such models to provide insights that range from fundamental aspects of cardiac function, to practical aspects of surgical interventions.

2. Overview

MTQV focus on the modelling of blood flow through the mitral valve into the left ventricle (LV), which is the primary driver of oxygenated blood to the body. The mitral valve controls the flow between the left atrium and ventricle and is quite prone to dysfunction and replacement. The typical Reynolds number of the flow through the mitral valve is approximately 4000, and this combines with the pumping action to generate a pulsatile turbulent flow in the left ventricle with a wide range of spatial and temporal scales.

Many of the previous computational modelling studies of intra-ventricular flows have focused on singular aspects of the problem such as evaluation of the functional morphology of a specific feature of the ventricle (Pedrizzetti & Domenichini 2005; Vedula et al. 2016) and/or valves (Seo et al. 2014), or the dynamics of natural and prosthetic valves (Peskin & McQueen 1980; Choi, Vedula & Mittal 2014) or validation (Vedula et al. 2014). The focus of MTQV is on the effect of the mitral valve on the haemodynamics of the LV, but a unique aspect of MTQV is that they tackle this problem from multiple fronts.

The natural mitral valve has an extremely complex anatomy, including a non-circular, time-dependent annulus, inhomogeneous tissue properties and chord-like structures (the chordae tendineae) that connect the two leaflets to the walls of the LV (Standring 2016). Modelling the fluid–structure interaction of this valve is therefore a significant challenge. MTQV employ a relatively simple model of the natural mitral valve, one that incorporates some key features of its anatomy, but is still realizable in computational models and experiments. The flow is solved using an immersed boundary method (de Tullio & Pascazio 2016) and the dynamics of the valve leaflets employ an interaction potential approach (Spandan et al. 2017) that models non-dissipative deformation of membranes with extensional and bending stiffness.

MTQV begin with a validation study where they compare their computed results against a companion experiment that consists of a flexible ventricle with a mitral valve (see figure 1), both made of silicone rubber. The expansion–relaxation of the ventricle in this experiment is driven by a time-varying flow into and out of a sealed Plexiglas tank that surrounds the ventricle. Thus, both the deformation of the ventricle and opening and closing of the mitral valve are a result of fluid–structure interaction, and MTQV show that their model provides a reasonably accurate prediction not only of the movement of the ventricle and the valve leaflets, but also of the flow profiles inside the ventricle.

Following the validation, MTQV establish the characteristics of the flow and valve kinematics for a healthy ventricle (ejection fraction (EF) = 60%) with a ‘natural’ healthy mitral valve. The mitral jet is found to generate a circular flow pattern from the lateral to the septal wall of the LV that increases ‘washout’ in the apical region of the LV. MTQV then shift their focus to cases with cardiac pathologies and examine the effect of leaflet stiffness and narrowing (stenosis) of the valve outlet area. They find that the limited mobility of the leaflets leads to a significant increase in jet velocity as well as the pressure drop across the valve. The latter is particularly

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Matters of the heart

![Diagram of heart valves](image)

**Figure 1.** (a) Computational model with natural mitral valve leaflets employed in MTQV. (b) Companion experiment used for validation of the computations. From Meschini et al. (2018).

![Visualization of flow in ventricle](image)

**Figure 2.** Visualization of the flow in the ventricle for the three valves at peak flow rate: natural, bioprosthetic and mechanical with EF = 60%. One isosurface of velocity magnitude is shown, coloured with the vertical velocity: range $-2$ (red) to $+2$ (blue). It is clear that each valve type generates a very distinctive flow pattern and jet penetration. From Meschini et al. (2018).

Important from the clinical viewpoint since increased pressure drop can translate into reduced cardiac output and a rise in blood pressure. MTQV also simulate a case with healthy valves but a reduced EF of 40%, a value that generally signifies borderline mild heart failure (https://my.clevelandclinic.org/health/diseases/17069-heart-failure-understanding-heart-failure/ejection-fraction). The simulations of MTQV indicate that, even with this reduced EF, the washout in the apical region is preserved.

The subsequent sections of MTQV address the haemodynamics of the LV with two types of valve implants: a bileaflet mechanical valve and a trileaflet bioprosthetic valve. The former is the most commonly used valve prosthesis in the mitral position, and MTQV conduct a number of simulations with this valve and find that it accelerates the breakdown of the mitral jet and consequently diminishes the penetration of the jet into the LV (see figure 2). Simulations with a bioprosthetic valve carried out for two different EFs indicate that the performance of this valve in terms of the LV haemodynamics is noticeably superior to the mechanical valve, although it still cannot beat the natural mitral valve in terms of overall ventricular washout. A more detailed analysis of the haemodynamic performance of the three valves in terms of LV thrombosis risk, effective orifice area (an oft-used metric for characterizing the flow blockage associated with cardiac valves (Yoganathan et al. 2004)), pressure drop across the valves and the ‘global circulation’ generated in the LV consistently shows that the bioprosthetic valve outperforms the mechanical valve but the natural valve exceeds both the others in every computed metric.
3. Future

The incidence of heart disease is reaching epidemic proportions and, while therapies for heart diseases have generally become more effective over time, the associated cost of treatment has also increased rapidly (Mittal et al. 2016). Computational modelling such as the type on display in MTQV offers the opportunity to harness the ever-increasing power of computers to deliver cost-efficient improvements in the management of heart disease. Recent inroads made by computational fluid dynamics into clinical cardiology (see Taylor, Fonte & Min 2013) only scratch the surface of what is possible. The type of computational modelling represented by the work of MTQV could find use in many aspects of clinical cardiology such as in the diagnosis of heart conditions, design and analysis of cardiac prosthesis, and planning of cardiac surgeries. However, significant advances are still needed before these computational models are ready for deployment in the clinic, and MTQV identify two of these as directions that they are pursuing: adding more realistic anatomical features, and including the physics of activation and contraction of the cardiac muscle.

References


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