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Modelling of a human mitral valve within left ventricle with fluid-structure interaction

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Computational modelling of the mechanical interaction of the mitral valve with the blood flow, particularly within the context of the left ventricular (LV) environment, can improve our understanding of valvular mechanics, and potentially lead to more efficient approaches to MV repair and replacement. To date, very few studies have reported valvular-heart modelling with fluid-structure interaction (FSI). In this study, we present a stand-alone MV model that includes physiologically detailed descriptions of the leaflets and the chordae tendineae with three different mathematical representations [1]. We then present an integrated MV-LV model based on magnetic resonance images acquired from a healthy volunteer [2]. The new MV-LV model incorporates the important valvular features, LV contraction, nonlinear soft tissue mechanics, as well as FSI. The MV and the MV-LV FSI model are developed using an open-source immersed boundary-finite element simulation framework.

The MV model is first verified under static boundary conditions against the commercial FE software ABAQUS, and simulated dynamically under physiological pressure conditions. The performance of the MV model is then studied by an energy budget analysis. Results show that a physiologically-correct chordal structure is necessary for achieving effective MV closure. The chordal structure also affects energy distributions within the FSI system. Our results also show that a psuedo-fibre chordal structure could be a good candidate for modelling MV dynamics because it is easy to construct and less intensive computationally as compared to more complex chordal models.

The MV-LV model is reconstructed from in vivo imaging data at early-diastole. First, we use this model to simulate cardiac function from diastole to systole. In diastole, a normal LV end-diastolic pressure (EDP) of 8 mmHg is applied to the left atrium side. To model active myocardial relaxation in diastole, an additional pressure (P_{endo}=16 mmHg) is applied directly onto the endocardial surface. In systole, myocardial active contraction is triggered simultaneously across the whole ventricle. Our results agree with in vivo measurements reasonably well. For example, in diastole, blood enters the ventricle with an increased volumetric flow rate across the MV, which gradually decreases towards end-of-diastole. In systole, the increased LV pressure due to the myocardial contraction closes the MV and pumps blood through the aorta valve (AV). The maximum flow rate across

the AV is 468 mL/s, the total ejection duration is around 243 ms, and the ejection fraction is 51%. These are comparable to clinical measurements of a healthy volunteer. We further investigate how P_{endo}=16 affects the MV-LV dynamics by varying its value from 8 mmHg to 16 mmHg, as well as the effects without changing P_{endo}=16 but increase EDP from 8 mmHg to 20 mmHg. We find that with an increased P_{endo}=16, the peak flow rate across the MV during the filling phase increases, followed by a prolonged ejection duration, a shorter isovolumetric contraction duration, and a higher ejection fraction.

In summary, the coupled MV-LV model with FSI can quantify the cardiac function in a more comprehensive way, with more pathological details for risk stratification in MV diseases.

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