FLOW IN AN INTEGRATED MODEL OF HEART AND AORTA

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<u>Summary</u> CFD of blood flow was carried out in an integrated model of the left ventricle and the aorta. It was shown that the formation of recirculating flow beneath the aortic valve (AV) during diastole effectively redirected inflow to the outflow tract, accommodating systolic ejection. The results also demonstrated that blood flow through AV had markedly skewed velocity profiles with swirling flows, provoking helical flow in the aorta. These findings addressed the importance of inclusion of the ventricular flow in the analysis of the aortic flow.

INTRODUCTION

Many computational fluid dynamics (CFD) studies of aortic flow have been carried out to clarify the pathophysiological significance of hemodynamics on localization of vascular diseases [1 and references therein]. However, despite the fact that blood flowing into the aorta is pumped by the left ventricle (LV), in most studies, the aortic section is analyzed isolatedly and an inlet flow is ideally conditioned. Liu [2] stated that the swirl inflow condition could affect the flows downstream of the aorta significantly. Hence, for the analysis of the aortic flow, flow dynamics stemmed from LV has to be taken into account. In this study, we carried out CFD of blood flow in LV and the aorta in an integrated manner to investigate the effects of the intraventricular flow on the aortic flow. Simulated results of cross-sectional velocity profiles at AV were compared to the ones obtained with magnetic resonance (MR) phase-contrast mapping.

METHODS

A computational model was built by combining an aorta model [1] and a left ventricle (LV) model [3]. Briefly, the model of aorta was constructed on the basis of clinical data, but three main aortic branches as well as other small branches were neglected. The total length of the aorta was 25 cm. As viewed from the top of the aortic arch, the angle between the ascending aortic arch and the descending aortic arch was 144°. The cross-section perpendicular to the centerline of the aorta was assumed to be a circle with its radius of 1.3 cm uniformly from the inlet to the outlet. The LV model was also modeled based on clinical data. LV was assumed to be symmetric with respect to a long-axis plane. The mitral valve (MV) and the aortic valve (AV) were both circular with radius of 1.3 cm. The deformation of LV was calculated by prescribing weighting functions of ventricular wall movement and the amount of volume change at every time step. It was assumed that the ventricular surface moved inwards or outwards in a normal direction independently of internal blood pressure. A time variation of the volume of LV was set based on MRI-derived data [4]. The length of systole and diastole were 0.413 s and 0.337 s. A stroke volume over a cardiac cycle was 70 ml.

It was assumed that flow was laminar, and blood was an incompressible Newtonian fluid. The flow simulations were performed under the moving boundary condition using a finite element method, as implemented in ANSYS-FLOTRAN ver. 7.0 (distributed by Cybernet System Ltd., Tokyo). The simulation was commenced from diastole, at the onset of which a quiescent flow state was assumed. In a diastolic flow simulation, the MV model [5] was used, and only intraventricular flow was calculated. During systole, flow in LV and aorta was calculated in an integrated manner.

RESULTS

Figure 1 shows streamlines of blood flow in mid diastole ($t = 0.250$ s), early ($t = 0.426$ s), mid ($t = 0.573$ s) and end systole ($t = 0.749$ s). The images are viewed from the left of the body with the septum (anterior) side towards the left. Once LV started to expand, blood flowed through the open MV into the ventricular cavity. In mid diastole, blood inflow gave rise to recirculating flow beneath AV, redirecting blood inflow preferentially towards the outflow tract (Fig. 1a). Later, this recirculating flow extended circumferentially to the posterior side, developing into an annular vortex that encircled blood inflow along the LV long axis. When LV started to contract, intraventricular blood flow was ejected to the aorta (Fig. 1b). It was accommodated by the recirculating flow under the aortic valve. Until mid systole, blood in the aorta was axially directed to the outlet along the curve of the aortic arch (Fig. 1c). However, with progress of systole, a helical flow developed. It firstly appeared in an ascending aorta, and extended to a descending aorta. By the end of systole, secondary helical flows dominated the whole part of the aorta as seen in Fig. 1d.

In Fig. 2, vector plots of secondary flow at the inlet section of the aorta at the same moments as Fig. 1b, 1c and 1d are illustrated, on which contour plots of the magnitude of flow velocity normal to this plane are superimposed. The image was viewed from the distal side, with the ventricular septum towards the up and with MV towards the bottom. In the beginning of systole, velocity profile was fairly flat (Fig. 2a). As LV contraction progressed, velocity profile was skewed to the MV side and a pair of swirls developed across the symmetric plane of LV (Fig. 2b). With the swirls, high
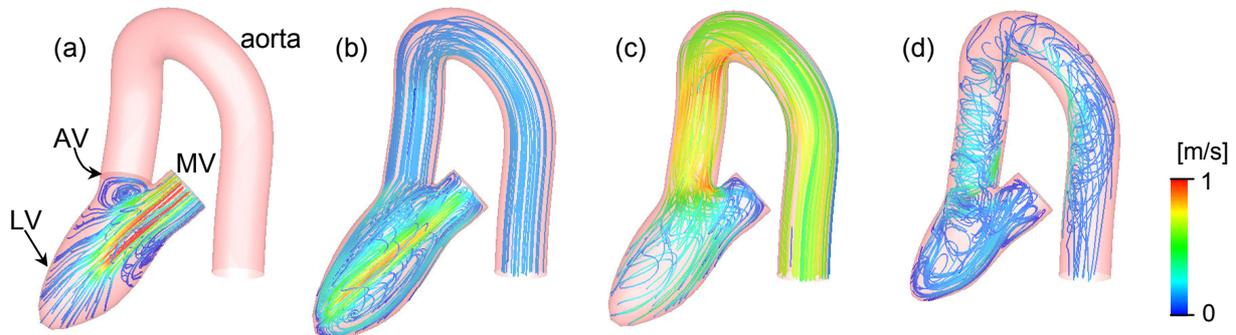
Fig. 1 Streamlines of blood flow in (a) mid diastole, (b) early, (c) mid, (d), end systole. Images are viewed from the left of the body with its septum (anterior) side towards the left. LV: left ventricle, AV: aortic valve. MV: mitral valve.
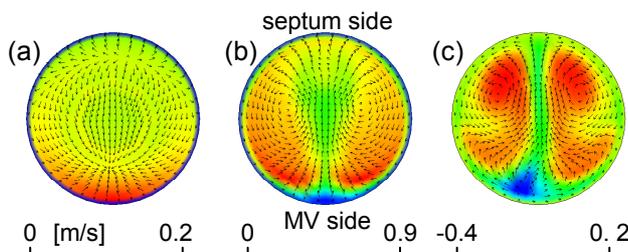

Fig. 2 Vector and counter plots of the flow at the plane at the aortic valve constructed from the simulation results. (a) early, (b) mid, (c), end systole.
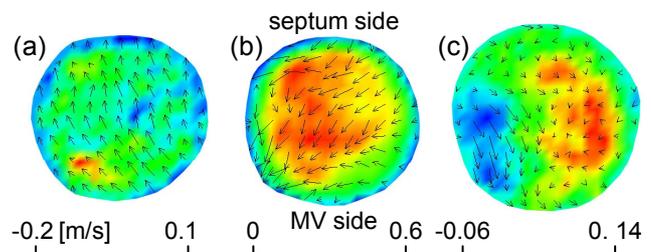
Fig. 3 Vector and counter plots of the flow at the plane 1.3 cm above from the aortic valve constructed from MR data. (a) early, (b) mid, (c), end systole.

velocity portion of the flow moved to the septum side, and flow through AV became faster close to the ventricular septum than beside MV (Fig. 2c).

DISCUSSION AND CONCLUSIONS

Flow patterns, especially generations of recirculating flows in LV and the helical flow in the aorta, simulated here were overall similar to *in vivo* results measured by Kilner et al [6][7]. It was found that the formation of recirculating flows beneath AV in the LV during diastole accommodated ventricular blood ejection to the aorta during systole.

It was observed that swirl secondary flows developed and the velocity profile at AV changed dynamically during systole. For comparison, a velocity profile at the cross-section about 1.3 cm above AV was measured with magnetic resonance phase-contrast mapping. Figure 3 shows examples of measurement results in early, mid and end systole plotted in the same manner as Fig. 2. They demonstrate that the skewness of the velocity profile started during mid systole and persisted to the end while a region of high velocity moved from the MV side to the septum side, as being consistent with our results. However, different subjects showed different patterns. Therefore, it was not possible to evaluate our results solely from these data. A large variation of the velocity profile at AV suggested that various factors such as a joint angle between LV and the aorta, LV motion and configuration of the aorta, could affect it.

At this moment, the fluidic mechanism that promotes the secondary flows at AV was unclear. However, it is at least likely that they affect the aortic hemodynamics including wall shear stress, particularly in the ascending aorta. If so, it would be possible that a change in a LV function is related to the occurrence of vascular diseases in the aorta. Thus, it is important to take into account intraventricular flow dynamics when analyzing pathophysiological significance with respect to pathogenesis of the vascular diseases.

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