

EFFECT OF AGEING AND ARTERIAL STENOSIS ON VENTRICULAR-ARTERIAL COUPLING: A COMPUTATIONAL MODEL STUDY

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INTRODUCTION

The performance of the left ventricle (LV) is determined directly by its intrinsic properties (myocardial contractility and heart rate), and is regulated indirectly by the preload, and the afterload. In particular, the coupling between the left ventricle and the arterial system (VA coupling) was considered to have considerable influence both on the mechanical performance and on the biological remodeling of the coupled VA system. The VA coupling problems are most frequently pointed to in the context of ageing and hypertension. Ageing and hypertension lead to degenerated arterial remodeling featured by stiffness, which results in later and higher systolic pressure and wider pulse pressure that might in turn stimulate the remodeling of the LV. Typically, the LV adapts to confront higher and later systolic pressure by both hypertrophy and ventricular systolic stiffening (David A. Kass, 2005). As a result of the remodeling in both, AV coupling will be further altered to impair the cardiovascular reserve function.

VA coupling may also be affected by arterial stenoses which induce changes both in the magnitude of the afterload and in the pattern of wave propagation in the arterial system. So far, stenoses have generally been considered to have significant influences on local and downstream hemodynamics; the effects on far upstream hemodynamics and VA coupling were rarely addressed.

In this study, we develop a multi-scale model of the CVS, by coupling a one-dimensional (1D) model of the arterial system with a lumped parameter model (0D model) of the remainder. The multi-scale model is in a closed-loop form, which affords us a useful tool for investigating the interrelationship between cardiac indices and preload/afterload. With the model, we investigate the effects of ageing and arterial stenoses on VA coupling.

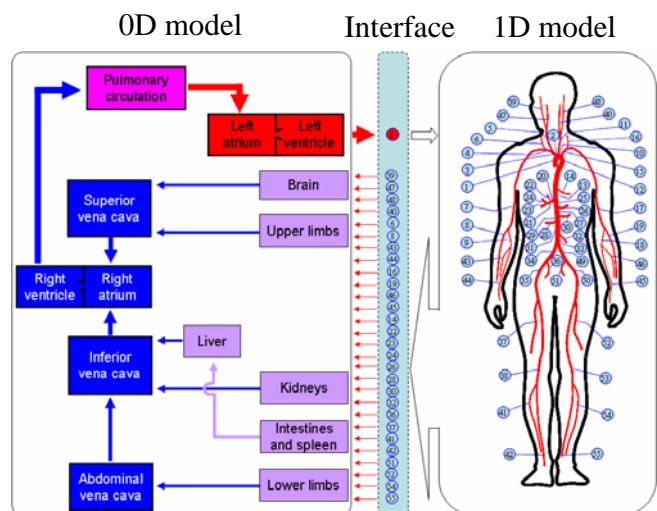


Figure 1. Schematic description of multi-scale modeling of the cardiovascular system

METHODS

The 55 largest arteries in the arterial system are described by a 1D model, and the remaining peripheral vascular systems, the heart and the pulmonary circulation are represented by a 0D model. The two models

are then coupled numerically to form a closed-loop system as shown in figure 1.

0-1D coupling computation is implemented at the aortic inlet and the distal ends of the 27 arteries through numerical iteration to reach continuity of mass and pressure between the two models.

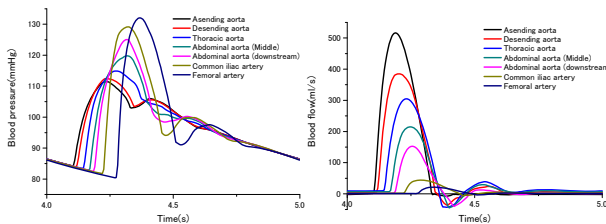


Figure 2. Simulated arterial pressure (left), and flow (right) waveforms

RESULTS

Figure 2 shows the simulated pressure and flow waveforms at several typical locations in the arterial system of a young adult. The simulations well reproduce the essential characteristics of arterial wave transmission in the arterial system. The systolic pressure increases away from the heart, towards the periphery, while the mean pressure decreases. Differently from the pressure waves, the flow waves show decreasing peaks and mean values along the aorta. In particular, in early diastole, strong retrograde flow appears downstream of the abdominal aorta.

The effects of ageing are studied for three representative ages: 20, 55 and 70 years old, respectively. Figure 3 shows the effects of ageing on the ascending aortic pressure and on the P-V relationship of the LV. It is evident that the systolic pressures of the elderly are much higher than the young adult, which is considered to result from the early arrival of the reflected waves at the LV due to the increased wave propagation speed in the arterial system in the elderly with arterial stiffening. The P-V relationships of the LV

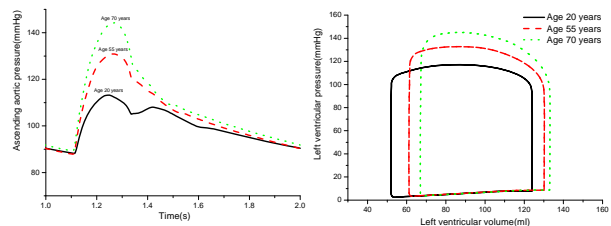


Figure 3. Effects of ageing on aortic pressure (left) and on P-V relationship of the LV

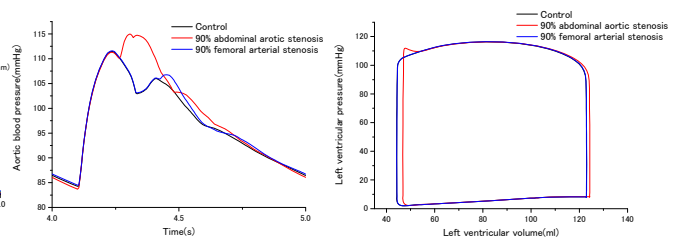


Figure 4. Effects of stenoses on aortic pressure (left) and on P-V relationship of the LV (right). indicate the higher oxygen consumption while smaller stroke volume in the elderly than in the young adult.

The effects of stenoses located in the abdominal aorta and the femoral artery are compared. The ascending aortic pressures and the P-V relationships of the LV simulated for the two 90% (occlusion ratio) stenoses are plotted in figure 4. The femoral arterial stenosis seems to only affect the diastolic pressure in the ascending aorta with no significant influence on the P-V relationship, while the aortic stenosis induces considerable changes in the ascending aortic pressure over a cardiac duration and deterioration of the LV performance characterized by early closure of the aortic valve and abrupt increase in LV pressure in late systole.

REFERENCES

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ACKNOWLEDGEMENTS

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