Atherosclerotic plaques affect resting myocardial blood flow quantification using contrast-enhanced magnetic resonance perfusion imaging

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INTRODUCTION Coronary artery disease (CAD) is the most frequent cause of death in industrial nations[1]. It results in a narrowing of the coronary arteries, which may lead to reduced myocardial perfusion. Quantitative MRI-based measurement of myocardial blood flow (MBF) is a promising technique for early diagnosis of myocardial perfusion deficits[2]. This method requires accurate knowledge of the arterial input function (AIF), i.e. the amount of contrast agent (CA) entering the myocardial tissue, which is usally obtained from the MRI signal in the left ventricle. Between this point of measurement and the myocardium dispersion of the CA bolus may occur[3]. The influence of atherosclerotic plaques on this dispersion, however, has not yet been studied in detail. In this contribution, we hence use computational fluid dynamics (CFD) simulations in a high-detailed coronary vascular model to study the impact of both stenosis grade and morphology on CA bolus dispersion and, consequently, on MBF quantification.

METHODS A high-detailed model of the left anterior descending (LAD) coronary artery was reconstructed based on a computed tomography scan of a coronary corrosion cast (Fig. 1). At the beginning of the main branch, several concentric stenoses with varying degrees of area reduction (50%, 70%, 90%, 93%, 95%) as well as four 93%-grade stenoses with different shapes were inserted. The morphologies of these obstructions were based on the classification by Meuwissen et al.[4] and included two eccentric, an asymmetric and a complex shape (cf. Fig. 1). Hexahedral O-grid type meshes containing between 3.1 and 3.6 million elements were created for all models. Simulation of blood flow and CA transport was carried out by solving the incompressible Navier-Stokes equations and the advection-diffusion equation using the open-source software package OpenFOAM (OpenFOAM 2.2.2, ESI Group, Bracknell, United Kingdom). Possible transitions to turbulent flow were accounted for by including a large eddy simulation turbulence model[5]. A realistic pressure profile was assigned at the inlet to include reductions of inflow due to the inserted stenoses. The resistance of the downstream vascular tree was included by applying resistance outlet boundary conditions based on the structured tree model[6] including arteriolar vasodilation for the high-grade stenoses. Blood was modeled as an incompressible non-Newtonian fluid with density $\rho = 1.06\ \mathrm{g/cm^3}$ using the generalized power-law model[7]. For the transport simulations, we employed a gamma-variate function at the inlet[3] and a diffusion constant of $D = 2.98 \cdot 10^{-10}\mathrm{m^2 s^{-1}}$. All simulations were carried out on the High Performance Cluster Elwetritsch (Elwetritsch, RHRK, TU Kaiserslautern, Germany) using 128 cores. The error that arises if dispersion is neglected was determined as described earlier[8], with the reference MBF value adjusted by the reduction of mean inflow in case of stenosis.

Fig. 1. The 3D model of the LAD artery. The numbers indicate the outlets that were evaluated, the small figures on the right show the employed stenosis shapes: concentric, eccentric I, and eccentric II, asymmetric, complex (top to bottom).

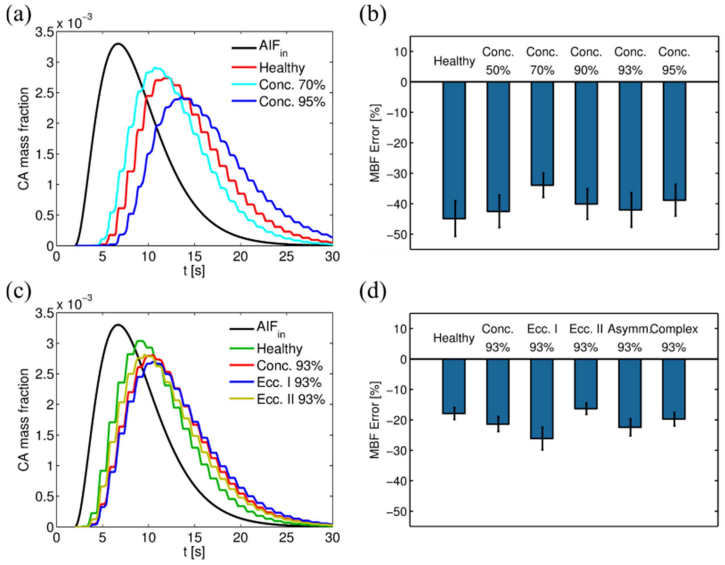

Fig. 2. The concentration-time curves that were observed at outlet 1 (a) and outlet 2 (c) in the stenosis-free model and in a part of the models with stenoses as indicated in the legend. The resulting MBF errors are depicted in (b) and (d), respectively.

RESULTS Marked dispersion was observed at all outlets in all models, but with a varying degree of magnitude. While mild stenosis reduced dispersion in some cases (cf. Fig. 2a), more severe dispersion was observed with increasing stenosis severity for stenosis grades higher than 90%. The relationship between stenosis morphology and CA dispersion was found to be very complex. Fig. 2b shows the concentration-time curves at outlet 2, where large differences due to stenosis morphology were observed. The difference between the two eccentric stenoses was comparable to the difference between the stenosis-free and concentric stenosis model. MBF errors due to CA dispersion varied strongly between outlets, and were heavily influenced by both stenosis grade and morphology (Fig. 2b,d). In general, however, no clear relationship between dispersion and MBF underestimation was found.

DISCUSSION Our analysis reveals that atherosclerotic plaques have a substantial impact on CA bolus disperison in coronary arteries. The presented results indicate that a complex relationship exists between local hemodynamics and bolus dispersion. The magnitudes of the resulting MBF errors suggest that the accuracy of quantitative MBF measurements using contrast-enhanced MRI may be impaired by these vascular transport effects. The impact of dispersion on the MMID4 analysis seems to be affected by the magnitude of the MBF value itself. It may be speculated that other quantification techniques such as the frequently used Fermi model[2] are subject to similar effects.

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