STRESS ANALYSIS ON 3D CORONARY ARTERIAL TREE AND PLAQUE SUBJECT TO CYCLIC FLEXION

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INTRODUCTION

Plaque rupture with superimposed thrombosis is the main cause of the acute coronary syndromes of unstable angina myocardial infarction, and sudden death. Previous studies have shown that endothelial disruption may relate to mechanical fatigue associated with the number of cyclic flexion on plaques [1,2]. A novel method is proposed to analyze and quantify local stress on *in vivo* patient-specific coronary vascular structures and plaque reconstructed from a pair of cine angiograms subject to cardiac motion. We hypothesize that mechanical stress on coronary arterial wall and plaque depends on the magnitude of deformation and the artery geometry in terms of vessel diameter, narrowing percentage, and lesion length. This work is of significance in facilitating evaluation of a moving coronary arterial tree attached to the myocardium and assessment of arterial mechanical stress.

METHODS

The major processes include two main tasks: (a) Coronary artery reconstruction and (b) Strain and stress analysis by using FEM (ANSYS 6.0). A previously validated method [3] was used for dynamic three-dimensional (3-D) reconstruction of the moving coronary arterial tree from a pair of routine cine angiograms acquired at any two arbitrary viewing angles. The *in-vivo* solid dynamic analyses can then be performed to assess the motion pattern of the moving coronary arterial tree or plaque throughout the cardiac cycle [4]. A simulation study was performed by using a finite-element analysis (FEA) to investigate the local stress on the normal and diseased moving arterial wall with lipid pool and plaque cap (n=48) subject to the cardiac cyclic flexion. The diseased arterial segments consist of vessel wall, plaque cap, and lipid pool as shown in Figure 1. To study the effect of lesion geometry relative to the stress magnitude, various types of lesion characterized by (a) site location, (b) length, and (c) degree of stenosis (narrowing ratio in diameter) are employed.

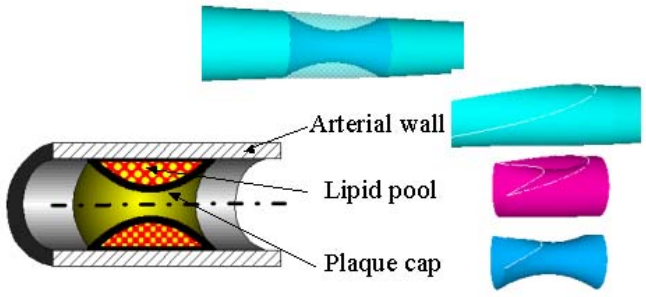

Figure 1: Components of a modeled lesion in FEM simulations.

RESULTS

A typical example of moving coronary arterial tree is shown in Figure 2. According to the simulations, the diseased vessel with smaller vessel diameter, higher narrowing percentage and/or larger lesion size result in higher elastic stress on the plaque cap, in which the vessel diameter is the dominant variable as indicated in the Table 1. Figure 3 shows the effective stress distribution on the lumen surfaces of a left coronary arterial tree calculated from end diastole (ED) to end systole (ES). The maximum stress is 6.1*E5 dyne/cm^2. A large value of stress gradient was found at bifurcation regions indicating that higher stress occurs at junction between the proceeding and branching vessels during the cyclic flexion. Additionally, the regions closer to the bifurcations underwent a larger deformation yielding a higher stress than those further to the bifurcations. These findings agree with the phenomenon that plaque rupture frequently occurs in the location where the geometry of artery shape is subject to considerable transforming, which has been reported in previous research [5].

Figure 2: Superimposition of the reconstructed left coronary trees from a pair of cine angiograms acquired from end-diastole to end-systole.

Factors of lesion geometry	Maximum of effective stress	Stress distribution
Lesion length ↑	Increase ↑	Uniform stress concentration
Narrowing percentage ↑	Increase ↑	Maximal stress at the middle of plaque
Diseased artery diameter ↑	Decrease ↓	Stress gradient decreases

Table 1: Effects of lesion geometry on maximum stress and distribution.

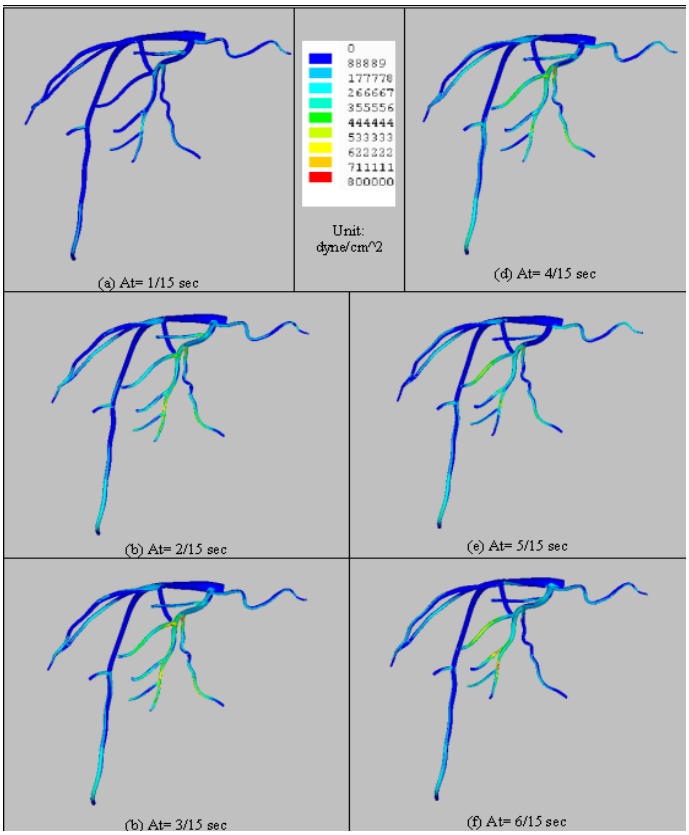

Figure 3: Stress distribution and variation with time.

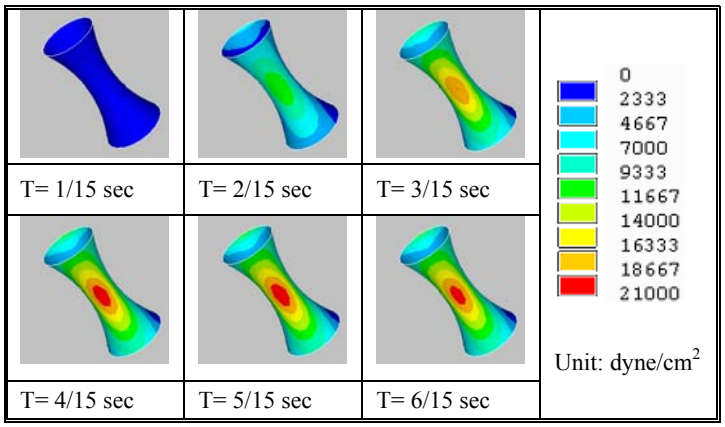

Figure 4: Cap surface stress distribution on a narrowed vessel with 1.5mm diameter, 3mm lesion length, and 50% narrowing.

CONCLUSIONS

The impact of shear flow stress on vessel wall has been elucidated in recent research [6]. There are still lack of methods to explore and quantify the mechanism of plaque atherogenesis, plaque composition and plaque rupture subject to cyclic stress generated by heart muscle contraction. In this study, a technique is proposed to estimate the elastic stress on vessel wall of patient-specific coronary arterial tree and study the relationship between the lesion geometry and the stress distribution on plaque cap. The combined technologies of 3-D coronary tree reconstruction and FEA modeling can facilitate creation of patient-specific lesion profiles for clinical evaluation, potential prediction of future events, and optimization of treatment.

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