Pulsatile calculations show that the introduction of the micro-catheter increases significantly the wall shear stress and pressure at the parent artery, aneurysm wall and neck, while the flow and loads in the sac are reduced. Complex haemodynamic patterns are observed and the characterisation of loads on the catheter, local flow patterns induced by the catheter and blood flow lines are studied. Comparisons with the catheter-free case illustrate the consequences of catheterisation. Such insight is expected to lead to an improved understanding of the treatment and the development of new techniques and devices.

7143

Three dimensional measurements of cerebral aneurysms and vessel size
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Our goal is to find the range of size of cerebral aneurysms, neck sizes, and vessel diameters with use of 3D rotational angiography. Measurement of 1st(A), 2nd, and 3rd axes of aneurismal cavity, the maximum and the minimum of diameter of neck (N), and the diameter of parent vessel (P) were obtained from 43 cerebral aneurysms in 30 patients with use of 3D-RA (Philips Medical Systems, INTEGRIS 3D-RA version 4.1 and model BV 3000/6000). The average frequent place where cerebral aneurysms were found in the 43 is ICA (9) and AcomA (9). The next is IPC (7), MCA (6), and IC Tip (5). One aneurysm was found on the A1, V, M2, PCA, PICA, and PcomA. The long diameter of neck is the almost same as the short one or 1.75-fold longer that of shorter neck. The form of aneurysm cavity was an ellipsoid which allowed calculating the volume with the three axes. The 1st axis is 0.85-fold shorter than the 2nd axis and is 0.74-fold shorter than the 3rd length. All aneurysms on any places keep these ratios. The (P) is gradually decreased from ICA to MCA via IPC while (A) increased. (N) is almost same in any places. The range of (P) on all places is narrower than (N) or (A). Therefore, the ratio of (P)/(A) and (P)/(N) decreases along the vessel pathways, whereas (N)/(A) is unchanged. These results are used as the definition of characterizing aneurysm, neck and parent vessel.

6187

Fluid–structure interaction finite element analysis of middle cerebral artery aneurysm
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Hemodynamic factors such as the wall shear stress are believed to affect a number of cardiovascular diseases including atherosclerosis and aneurysm. Since resuling phenomena in a living human body is currently beyond the capabilities of in vivo measurement techniques, computer modeling is expected to play an important role in obtaining a better understanding of the relationship between the cardiovascular diseases and the hemodynamic factors. In order to investigate this cardiovascular hemodynamics, we have developed a modeling and simulation system in which the patient-specific 3D geometry of an artery can be obtained from clinical data, and analyzed. The simulation program is based on Fluid–Structure Interaction Finite Element method. The Newtonian fluid analysis is based on the deforming-spatial-domain/stabilized space–time (DSD/SST) formulation. The structure can be modeled as the elastic or hyperelastic material. In the simulations of cerebral artery aneurysm, the flow behavior with compliant arterial walls is different from what we see with rigid arterial walls. Consequently, the distribution of the wall shear stress on the compliant arterial walls is significantly different from that on the rigid arterial walls. With the idea that precise vessel wall modeling is indispensable in order to reveal the mechanism of growth and rupture of aneurysm, we have incorporated hyperelastic vessel wall and aneurysm model in the FSI analysis. The effects of factors that are believed to affect vessel wall dynamics, such as wall thickness and stiffness distribution, on the deformation of aneurysm and hemodynamics were investigated.

References

6254

Biomechanical study of structural changes of human cerebral arteries
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Introduction: Human cerebral arteries may develop aneurysms, as a result of geometrical and structural modifications in the arterial wall resulting from a variety of genetic, epigenetic and biomechanical factors. Aneurysms exhibit a markedly different wall composition and structure, characterized by relatively low elastin content. The goal of the study is to characterize the biomechanical properties of human cerebral arteries, post mortem, and to assess the effects of further degradation of elastin by elastase treatment.

Methods: Six left posterior common arteries (PCAL) and 6 right posterior common arteries (PCAR) were collected from cadavers (mean age of 74 years) earlier than 24 hours post mortem. After excision, arteries were carefully cleaned from their surrounding tissues and a small ring (2 mm) was cut off for the zero state load measurement. Arteries were subjected to elastase-induced for a period of 30 minutes before washed with a solution of PBS containing elastase inhibitor, aprotinin. Pressure-diameter curves were then recorded for later biomechanical analysis. A small ring was cut radially to measure the open angle and define the zero stress state.

Results: The sections stained with Hemuslu-Eosine and Aldhyde-Fuchsine put into evidence the elastin sheet degradation in elastase treated arteries as compared to normal arteries. Pressure-diameter curves were obtained from which structural and elastic properties were calculated. Elastase shifts the pressure-diameter curves up showing an effective dilation of the artery after elastase treatment. Both p-d curves, however, exhibit similar shapes and slopes, especially at the high-pressure range, suggesting that the collagen engagement profile is unaffected. This is reflected also on the compliance-pressure curves, which are remarkably similar.

Discussion and Conclusion: Degradation of elastin sheets of aged human cerebral arterial wall did not markedly modify the biomechanical aspect of the arterial wall. At high pressures, collagen, which was unaffected from the elastase treatment came in and limited arterial extension in a similar fashion in both control and treated arteries. Elderly cerebral arteries revealed to be fairly non-compliant at physiological pressure. This is consistent with the fact that in aged cerebral arteries elastin was already fragmented and not properly distributed, as confirmed by histology.