phase contrast MR demonstrate significant variation in Fontan hemodynamics with respiration, as well as smaller cardiac pulsatility (Hjortdal, et al. Circulation, 2003). In this work, we demonstrate that the effects of respiration and exercise cause significant hemodynamic disturbances and energy loss. Time-dependent, 3-D blood flow simulations are performed using a custom finite element solver and patient specific geometry. A respiration model has been developed to provide accurate inflow boundary conditions that capture respiratory modulation of IVC flow. Blood flow features, pressure, and energy losses are analyzed at rest and with increasing IVC flow rates to simulate graded exercise conditions. Resistance boundary conditions representing pulmonary vascular resistance are enforced at the pulmonary artery outlets and decreased by up to 20% during exercise (Kullik, et al. Br. Heart J., 1983). Energy efficiency is high at rest but drops substantially with maximal exercise. Flow vortices increase in intensity with respiration and exercise, explaining higher energy dissipation when compared to rest. Trends in efficiency across multiple patient-specific models are examined. We conclude that the effects of respiration and exercise should be incorporated in computational models to provide realistic evaluations of TCPG performance, and for future work in optimizing TCPG geometry.

14.5.2. Atherosclerosis and Aneurysms

Atherosclerosis and aortic aneurysms

C.K. Zarins, Stanford University, Stanford, CA

Hemodynamic factors play an important role in atherosclerotic plaque localization as well as in artery wall responses. The unique susceptibility of the human abdominal aorta to aneurysm formation may be due to a combination of aortic structural architecture, nutrition, and biology as well as to biomechanical forces acting over time. We have studied experimental and human plaque formation and adaptive aortic enlargement in relation to aortic wall strain and medial lamellar architecture.

In-vivo magnetic resonance imaging of the aorta in experimental animals and humans reveals that aortic wall motion and cyclic strain varies about the circumference of the aorta with the point of maximum wall motion spiraling down the aorta. The anterior aorta, which has the greatest degree of motion, also has greater medial thickness and more medial lamellae than the posterior aorta which moves very little. It is not clear whether this asymmetry in wall motion is due to atherosclerotic plaque localization, but exposure to oscillatory wall motion, inhibits plaque deposition. Differences in aortic wall strain patterns may also play a role in aneurysmal degeneration. Plaque deposition is accompanied by compensatory enlargement of the aorta with thinning of the media underlying the plaque. Under these circumstances the plaque may provide structural support to the aortic wall. Subsequent plaque atrophy may leave an enlarged aorta with a thinned wall unable to support wall tension. Understanding the biomechanical forces acting on the aortic wall may provide new clues regarding the pathogenesis and risk of rupture of aneurysms and lead to improvements in endovascular devices such as stents and stent grafts which significantly alter aortic wall motion and cyclic strain.

References


5614 We, 08:45-09:00 (P28)

Towards the integration of heterogeneous data: computational fluid dynamics as part of a processing chain in the context of risk assessment for cerebral aneurysms

A. Frangi1, R. Hese2, J. Ceaobla3, D. Rufenacht4, 1University Pompeu Fabra, Barcelona, Spain, 2Medical Physics, University of Sheffield, UK, 3George Mason University, USA, 4University Hospital Geneva, Switzerland

This paper will describe the development of an image processing and computation chain for assessment of haemodynamic characteristics of cerebral aneurysms of individual patients, and will outline how these data might be integrated with heterogeneous data from other sources (including the patient record) to improve the management of cerebral aneurysms. It is the primary thesis of this paper that the process of disease diagnosis, treatment planning and treatment development is compromised by the fragmentation of the data that should underpin it. The paper will represent computational fluid dynamics as one part of a ‘complex information processing’ strategy that can evaluate indices or parameters that cannot be deduced by direct measurement. Other parameters are derived from morphometric, mechanobiological and patient level or systemic data, and an overview of the processing operations for these data will also be presented.

The paper will focus on the challenges associated with the integration of heterogeneous data to complement the clinical decision-making process. The processing chain for haemodynamic characterization, including the image processing steps and the boundary condition specification, will be discussed in detail. The process of selection of important haemodynamic indices for cerebral aneurysm will be discussed. Results will be presented for a number of cases.

4450 We, 09:00-09:15 (P28)

Wall thickening and its relation to local haemodynamic parameters in peripheral bypass grafts

A. Augit1, M. Jackson2, S. Zhao1, A.D. Hughes2, S.A. Thorn2, X.Y. Xu1, 1Department of Chemical Engineering, 2NHIL, International Centre for Circulatory Health, St Mary’s Hospital, Imperial College London, UK

Atherosclerosis is characterised by accumulation of soft lipid material within hardened fibrous elements called plaques. It affects human arteries, leading to heart attacks, strokes, other circulatory problems. Several systemic risk factors are implicated in its development and have been incorporated within the “reaction to injury” hypothesis [1]. Plaques, however, have a multi-local distribution where systemic risk factors cannot account for. Lesions are predisposed near branch points and regions of marked or complex curvature, where disturbed flow may occur, resulting in local haemodynamic abnormalities. Altered haemodynamic forces (such as low and oscillating shear stress) may be critical in the localisation of plaque. Studies have observed flow abnormalities at sites commonly afflicted by plaque formation. To correlate underlying haemodynamics with plaque characteristics, proximal atherosclerotic plaque easting sucks is result due to its insidious development over many decades. However the autologous vein-graft is a convenient model where accelerated development of thickening is well described [2]. Using computer simulations, applied to graft geometrical and flow data, we have mapped these forces and prospectively observed for the development of wall thickening in the corresponding anatomical locations. 5 Subjects were scanned using MRI to obtain geometry from which computational fluid dynamics models were created. Calculated haemodynamic parameters were set in relation to intima-media thickness (IMT) and lumen diameter (LD) obtained from ultrasound scans 6–12 months after surgery. The results indicate possible correlations between haemodynamic parameters and IMT or LD in some but not all cases. Larger data sets should be analysed to expand possibilities of this promising approach.

References


5514 We, 09:15-09:30 (P28)

Association of biophysical forces with impaired remodeling mechanisms of the abdominal aortic aneurysm wall

D.A. Yorpp1,2, D.W. Chew1,2, N. Sakamoto1,3, M. Sakamoto1,3, M.S. Makaroun1, 1Department of Surgery and 2Department of Bioengineering, and 3McGowan Institute for Regenerative Medicine, University of Pittsburgh, Pittsburgh, PA, USA

Abdominal aortic aneurysm (AAA) rupture occurs when the wall stresses acting on it exceed the wall strength. While much attention has been placed on AAA wall stresses, relatively little work has been performed to understand the mechanisms of AAA wall weakening, which represents an imbalance between extracellular matrix synthesis and degradation. The goal of this work was to investigate the hypothesis that the wall of a AAA exhibits discordant repair/remodeling mechanisms, and that this is mediated by the local biophysical environment; namely, mechanical wall stress and hypoxia. Pairs of AAA wall samples were harvested at the time of elective surgical repair. Each pair was selected based on disparate levels of either mechanical wall stress, estimated computationally, or mural oxygen tension, predicted by the thickness of adjacent intraluminal thrombus. That is, each pair consisted of either “high stress” and “low stress”, or “hypoxic” and “normoxia” wall specimens. Using TaqMan® PCR, we computed the differential expression of genes selected based on their known association with remodeling. We also explored the response of macrophages to mechanical stimulation in vitro. Peripheral blood mononuclear cells were isolated from buffy coats obtained from blood bank, and cultured on collagen I or fibrin, in RPMI with 10% human serum and Granulocyte-Macrophage Colony Stimulating Factor. C3 (part of the complement system of the immune system) and P-Selctin were both elevated in high stress AAA tissue samples, while endothelin-1 and collagen IV were reduced. MMP-2, MMP-9, uPA, tPA, CD68, heme oxygenase-1, oxygen-regulated protein, ICAM-1, IL-12>p40, a nitric oxide synthase and VEGF were upregulated in “hypoxic” versus “normoxic” AAA tissue, while collagen IV was reduced. The expression of monocyte marker CD162L on cultured macrophages decreased after exposure to 10% cyclic stretch for 24 hours, suggesting that cyclic