Vascular device interaction with the endothelium

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Cerebral stents and Intra Aortic Balloon Pumps (IABP) are examples of mechanical devices that are inserted into arteries to restore flows to clinically healthy states. The stent and the IABP 'correct' the arterial flow by static dilation and by cyclical occlusion respectively. As this presentation shows, these functions are effectively modelled by current engineering practice. As interventions however, by their very nature they involve physical contact between a non-biological structure and the sensitive endothelial surface. The possible damage to the endothelium is not currently well addressed and we also consider this issue.

Cerebral stents generally have two primary clinical objectives: to mechanically dilate a stenosed artery and to have minimal detrimental impact upon local blood flow characteristics. These objectives are well served at the arterial scale as these devices are evidently effective in opening up diseased arteries and restoring vital flows. However, at the near-wall micro-scale the picture is less satisfactory, as thin stent wires apply stresses to the endothelium and glycocalyx and the local flow is disturbed rather than being ideally streamlined. This causes further interaction with this endothelium topography. Wall Shear Stress (WSS) is the measure commonly used to indicate the interaction between fluid and wall but it is a broad brush approach that loses fidelity close to the wall. We will present simulation results of blood flow through a stented cerebral saccular aneurysm under these limitations of WSS.

The Intra Aortic Balloon Pump (IABP) is a widely used temporary cardiac assist device. The balloon is usually inserted from the iliac artery, advanced in the aorta until it reaches the desired position; with its base just above the renal bifurcation and the tip approximately 10cm away from the aortic valve. The balloon is inflated and deflated every- (1:1), every other- (1:2) or every second (1:3) cardiac cycle. Balloon inflation, which takes place during early diastole, causes an increase in the pressure of the aortic root which leads to an increase in coronary flow. Balloon deflation which takes place during late diastole achieves one of the main IABP therapeutic effects by reducing left ventricular afterload.

Unavoidably, the balloon contacts the inner wall of the aorta with every inflation/deflation cycle. This repeated event and possible contact with atherosclerotic plaque have been reported to be responsible for balloon rupture. However, there has not been a methodical study to investigate the mechanical effects of balloon-wall interaction. For example, during inflation the balloon approaches the endothelium as it displaces a volume of blood proximally and distally. This squeezing process generates shear stresses, which hasn't yet been quantified. Similarly, when the balloon moves away from the endothelium during deflation, it generates micro pressure differences that may impose stretching (pulling) stresses on the endothelium cells.

Both of the above cases indicate that a very high spatial resolution is required in order to fully understand the process of interaction between device and endothelium, and to interpret the effects at the cellular level.

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