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PERIPHERAL BYPASS GRAFT ATHEROGENISIS STUDY USING NEAR-WALL CONVECTIVE TRANSPORT NORMAL TO THE WALL, GIVEN BY A SERIES EXPANSION OF LAGRANGIAN DYNAMICS.

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ABSTRACT

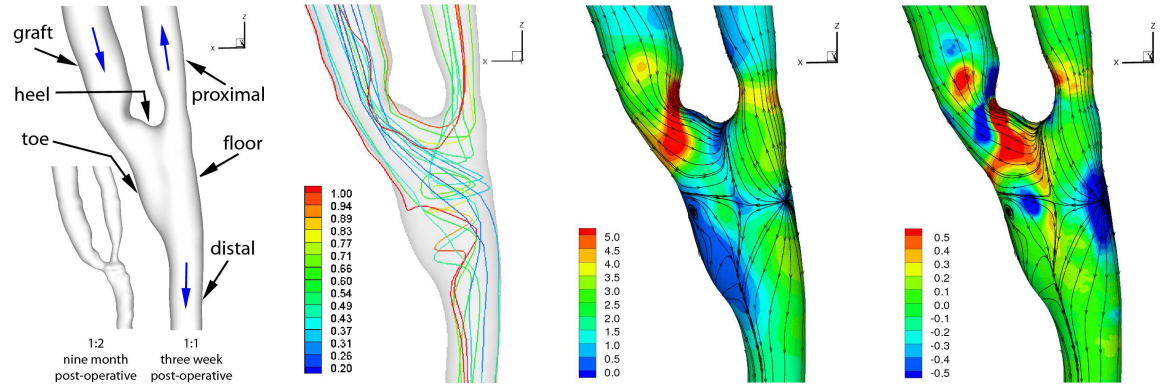
This patient specific study of a peripheral bypass graft geometry explores measures, based on the *wall shear stress* (WSS), to identify regions at risk from atherogenisis. Follow-up scans are used to study the post-operative vascular remodelling, in this case consisting in re-stenosis, resulting in re-operation to insert a jump graft, which also failed. The study represents an extreme case which is prone to disease. This work has a broad scope in physiological flows as well other applications where interactions between flow and wall is of importance.

One manifestation of cardiovascular disease is *peripheral arterial disease* and arises through atherogenesis (the development of early atherosclerotic lesions) [1], which are located preferentially at bifurcations at the toe, heel and floor as shown in Figure 1(a). It has been postulated since the work of [2] that atheroma in man is predisposed to occur in regions of low WSS; furthermore most intimal thickening is found in regions where the average WSS is less than 10 dynes/cm^2 (1 Pa = 10 dynes/cm^2) [3]. Large near-wall residence times, which in itself is linked to the WSS, has also been thought as a possible correlation with atherogenesis due to the poor oxygenation of the lumen wall.

Near-wall transport to and from the wall is of importance in arterial haemodynamics from the stand point of exchange processes and interactions between flow and wall. In this study of the bypass graft this transport may be considered beneficial when considering oxygen and nutrients being supplied to the wall and harmful when considering excessive low density lipids (LDL) transport to the wall.

The near-wall convective transport normal to the wall is derived by considering a series expansion of Lagrangian dynamics for a Newtonian fluid. From this expansion, up to second order terms and assuming steady flow (reasonable simplification as discussed for the same patient case in [4]), the transport normal to the wall is given by

$$\phi = -\frac{1}{2\mu}(\delta t \cdot \delta z^2)\left(\frac{\partial \tau_x}{\partial x} + \frac{\partial \tau_y}{\partial y}\right) \tag{1}$$



(a) (b) (c) (d)

Figure 1: (a) Bypass geometry taken three weeks and nine months post-operatively with blue arrows indicating direction of flow. Regions termed toe, heel and floor are of typical arteriosclerosis locations. (b) Massless particle tracks coloured by time [s] from their release at the graft inlet, highlight features in the flow field. CFD results of (c) wall shear stress (WSS) magnitude [Pa], and (d) transport to and from the wall [mm s^{-1}] (with -ve transport is to and +ve from the wall) with shear lines.

where x and y locally define the geometry wall tangent plane and z is perpendicular to the tangent plane, δt and δz are small increments. The convective transport normal to the wall is therefore proportional to the spatial gradients of WSS. Plotting the surface shear lines, which are aligned to the tangential component of the viscous traction exerted by the flow on the wall, regions of transport to the wall are further noted by divergence of the shear lines and transport from the wall as coalescing.

In terms of the detailed haemodynamics, the regions of separation at the toe and heel of the anastomosis, and the stagnation point at the floor due to the impact of the jet coming from the graft are clearly evident. From Figure 1(c) it is noticeable that the toe, heel and floor of the anastomosis are regions stipulated to be at risk with WSS$<$1 Pa; in fact over 50% of the anastomosis has WSS$<$1 Pa, representing an extreme case.

Figure 1 shows the geometrical post-operative evolution in the first nine months. The vascular remodelling reduces the areas of recirculating fluid, with the anastomosis narrowing around the areas of faster flow, tending to reduce the spatial gradients of the wall shear stress which yield the convective transport normal to the wall. The reduction of conduit patency occurs mostly at the toe of the anastomosis, where the WSS predicted by the steady computations is low, as well as at the the start of the graft and proximal artery despite being regions of respectively elevated WSS and not expeceted to remodel and reduce conduit patency.

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