Wall Shear Stress and Arterial Performance: two approaches based on engineering

Michael W Collins, Carola S Koenig* & Mark A Atherton
School of Engineering and Design,
**Brunel Institute of Bioengineering,*
*Brunel University, Uxbridge UB8 3PH, UKAddres*s
collinmw@hotmail.com

This crucially important subject generates a very wide literature and the recent authoritative 'in vivo' review of Reneman *et al* [1] (& [2]), with Vennemann *et al* [3], are taken as seminal. In this paper we use approaches based on conventional engineering to address two key issues raised in [1].

The first is that of basic theory. *To what extent can underlying fluid flow theory complement the in vivo understanding of wall shear stress (WSS)?* In [1], which is sub-titled *Discrepancies with Theory'*, Poiseuille's Law is used, extended to Murray's Law in [2]. But they do 'not hold in vivo' [2] because 'we are dealing with non-Newtonian fluid, distensible vessels, unsteady flows, and too short entrance lengths' [1].This comment coincides with the four factors Xu and Collins identified in their early Review of numerical analysis for bifurcations [4]. Subsequently they addressed these factors, with an engineering-based rationale of comparing predictions of Computational Fluid Dynamics (CFD) with Womersley theory, *in vitro* and in *vivo* data. This rationale has yet to be widely adopted, possibly due to computing complexities and the wide boundary condition data needed. This is despite uncertainties in current *in vivo* WSS [2].

Secondly, [1] and [2] focus on endothelial function. WSS is an 'important determinant of arterial diameter' and 'mean (M)WSS is regulated locally'. One pointer is the possible importance of the glycocalyx, so that 'endothelial cells are not seeing WSS' and which 'may be involved in the regulation of the total blood flow' [3]. A typical glycocalyx is shown in [3]. Such a model should focus on adaptation of arterial diameter by 'nitric oxide and prostaglandins' [1]. *So, using an engineering approach, can we construct a model for local regulation of MWSS?* Again, remarks from [1]-[3] resonate with the conclusions of a review of nanoscale physiological flows [5] undertaken as part of an early Nanotechnology Initiative of the UK's EPSRC. In [5] is illustrated the fractal nature of the intestinal villi-glycocalyx geometry, together with an engineering-style control loop for nitric oxide release and arterial diameter-flow rate control.

Within our discussion we report two studies to obtain CFD predictive data very close to the endothelial surface. In both cases we compared two independent codes, respectively two CFD codes, and CFD and Lattice Boltzmann solvers. We also give an updated version of the endothelium control loop.

[1] Reneman R.S. *et al,* J Vasc Res 2006; 43: 251-269
[2] Reneman R S. *et al*, Med Biol Eng Comput 2008; 46: 499-507
[3] Vennemann P *et al*, Exp Fluids 2007; 42: 495-511
[4] Xu X.Y. & Collins M.W. Proc Inst Mech Eng (Part H) 1990:204: 205-216
[5] Ciofalo M. et al, Med Eng Phys 1996; 18, 6: 437-451