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**Comment on “Prosthetic heart valves’
mechanical loading of red blood cells in
patients with hereditary membrane defects”,
Grigioni *et al.*, Journal of Biomechanics
38:1557-1565**

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Grigioni *et al.* (2005) have proposed a model for loading of blood cells in turbulent flow. At each point on the surface of a geometrically simplified cell, the Reynolds stress tensor is resolved into normal and tangential components. The magnitude of the tangential component is then integrated over the whole of the cell surface to give a single scalar parameter, which is presented as an expression of the overall shear loading on a cell. Grigioni *et al.* have suggested that their results explain differences in observed hemolytic state among patients with prosthetic heart valves and abnormal erythrocyte morphology, based on a clinical sample of two patients.

The analysis neglects the physical nature of Reynolds stress, which is not simply analogous to stress in solid mechanics. Reynolds “stresses” are in fact products of density and turbulent velocity fluctuations. In the Reynolds-average view of turbulent flow, a velocity component $u_i(x, y, z, t)$ is decomposed into a time-average component $\bar{u}_i(x, y, z)$ and a fluctuating component $u'_i(x, y, z, t)$. The Reynolds stress tensor is then defined by $\tau_{ij}^R = \rho \overline{u'_i u'_j}$, where ρ is fluid density. Thus, the Reynolds stresses are momentum fluxes which must be added to viscous stresses in order to give the correct acceleration of fluid particles *in a time-average sense* (see, for example, Bradshaw, 1971).

The Reynolds stress is a useful analytical tool when it is desirable to view the complex, unsteady turbulent flow at long time scales and large length scales, as in many engineering applications. However it cannot be interpreted directly as the flow-induced stress on a surface – in fact, because of the no-slip boundary condition, the Reynolds stress is zero at any surface exposed to flow (in a reference frame fixed to the surface). The problem with over-reliance on the Reynolds stress can be highlighted by considering the importance of time

scale. Low-frequency turbulent fluctuations in blood velocity (corresponding to large eddies) simply transport a cell, causing low acceleration and low stress. In high frequency fluctuations (small eddies), because of a cell's inertia, it will be subject to higher slip velocity relative to plasma, and higher velocity relative to neighbouring cells, which experience different local flow conditions in different eddies. However, these effects are ignored if Reynolds stress is the sole descriptor of turbulence, since fluctuations at all frequencies are weighted equally in the Reynolds stress.

The work of Grigioni *et al.* is a welcome step towards understanding the effects of turbulence on blood cells at a fundamental level. However, it is an oversimplification to suggest that the Reynolds stress is an adequate or complete description of the loading on a suspended cell due to turbulence.

References

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