THE EFFECT OF WALL REACTION RATE ON COMPUTATIONALLY SIMULATED PLATELET ADHESION IN A 2D EXPANSION TUBE

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INTRODUCTION

The formation of thrombi pose one of the greatest threats to modern human life. Their negative effects can be seen in the atherosclerotic diseases: coronary artery disease, peripheral artery disease, and stroke and many other problems including those induced by the use of prosthetic surfaces, such as artificial heart valves and aortic grafts. The first step in the formation of a thrombus is the adhesion of platelets. The dependence of platelet adhesion on the interactions of blood flow, diffusivity and biochemical reaction, has long been studied, by many groups. However, the influence of complex flow, found to occur at the sites of arterial branching and stenoses in the above listed conditions, characterised by recirculation zones and stagnation points, although acknowledged, has yet to be presented as a determinant of reaction mechanism, modeled numerically. This model has been developed to investigate the effect of shear dependent reaction rate mechanisms within complex flows on platelet adhesion. The results are compared to previously published experimental and analytical work [1,2,3].

METHOD

In order to model representative complex flows the flow through an expansion tube was simulated (inlet height = 0.917 mm, outlet height = 1.5 mm), in a similar manner to previous experimental work [1]. This was implemented on an NT PC using the commercial finite volume code FLUENT 5.5 (FLUENT Inc.). The equations for conservation of mass, momentum and species were solved using an implicit segregated solver, in such a manner that the fluid flow was solved initially and then used to generate the species solution. The fluid flow boundary conditions were a parabolic inlet velocity (U_{max} = 0.06 ms^{-1}), and an outflow outlet, Re = 37.5. Platelets were modeled as a continuum species in a bulk flow (mass fraction = 0.0035), which adhere to the surface, downstream of the expansion, via a specified wall reaction mechanism. A linear shear dependent wall reaction rate was implemented through a user defined subroutine, equation 1 [2].

$$K = k_0 + \alpha\tau \quad (1)$$

Where K is the wall reaction rate, τ is the shear stress at the wall, k_0 is a constant (0.001 s^{-1}) necessary to maintain adhesion at the stagnation point and α is shear dependent weighting, α was varied from 0 - 1 1/Pa s.

The convergence criterion was set to 10^{-6} for all residuals.

RESULTS AND DISCUSSION

The fluid solution for the flow through the expansion tube is illustrated by the streamlines in figure 1, (top). This clearly illustrates a recirculation zone downstream of the expansion, terminated in a stagnation point after which the flow returns to Poiseuille flow. The corresponding wall shear stress magnitude, on the wall downstream of the expansion, is given in figure 2. This shows a peak in wall shear stress within the recirculation zone with zero wall shear stress at the stagnation point, SP, on figure 2. This then rises to a constant downstream value.

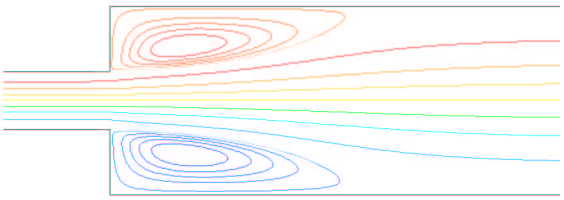

Figure 1. Streamlines for flow through the expansion tube, Re = 37.5

The rate of platelet adhesion to the downstream wall as a function of wall reaction mechanism is illustrated in figure 3. For the reaction linearly dependent on shear stress the curves show a peak in platelet adhesion within the recirculation region, followed by a minimum at

the stagnation point, followed by a second peak in adhesion downstream of the stagnation point. Therefore maximum platelet adhesion does not occur at the stagnation point, but occurs a short distance at either side. This result agrees well with experimental studies of platelet adhesion in expansion tubes [1]. When the shear weighting was reduced to zero, such that the reaction became first order the minimum in adhesion at the stagnation point disappeared, in contradiction of experimental results [1, 3]. This suggests therefore that platelet adhesion is not governed by a simple first order reaction mechanism.

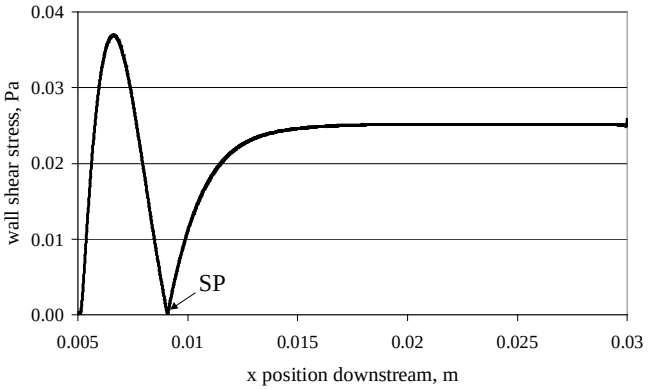

Figure 2. Wall shear stress downstream of expansion, Re = 37.5, showing stagnation point.

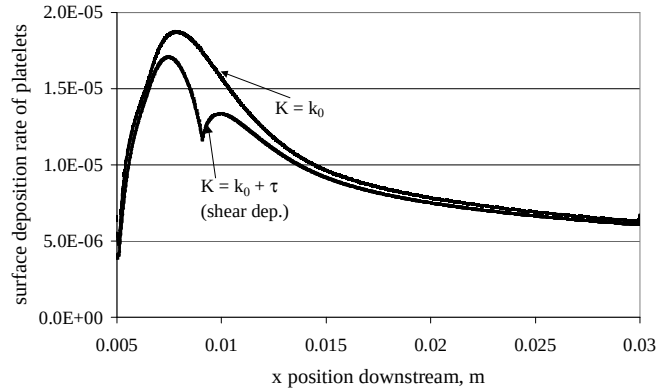

Figure 3. Rate of platelet adhesion downstream of expansion tube

CONCLUSIONS

The adhesion of platelets in complex flows has been modeled numerically and found to agree well with previously published experimental and analytical results [1,2,3]. The first order reaction rate did not mimic the form of adhesion expected. In order to achieve this a reaction rate dependent upon shear stress proved to be necessary. A linear dependence produced good results, but further studies need to be undertaken to identify the exact relationship. It is hoped that this computational model may now form a platform on to which higher degrees of complexity may be built, more closely simulating the altered vascular geometries encountered in diseased arteries and regions proximate to prosthetic devices. This would allow problem areas to be identified more swiftly and optimum geometries for prosthetic devices and surgical procedures, such as bypass grafts, to be designed.

REFERENCES

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