

LETTER TO THE EDITOR

Blood Flow, Slip, and Viscometry

Dear Sir:

In a recent paper by Nubar (1) it was proposed that the anomalous viscous behavior of blood is largely due to slip. We believe that this is just one possible explanation; others of equal or even greater justifiability are ignored in that paper.

(a) Blood viscosity may not in general be independent of the magnitude, or even the direction, of the rate of shear. The viscous property of fluids is a consequence of molecular interaction. For a given fluid, the coefficient of viscosity is in fact a fourth-order *tensor* which reduces to a constant scalar only when it is assumed that the rate of shear is small and the fluid is statistically isotropic (2). In the extreme conditions under which blood exhibits variable viscosity, neither of these assumptions can be substantiated.

(b) The results of viscometry may be based on the wrong equations of motion. The Navier-Stokes equations, on which these results are based, assume that the fluid can be treated as a *continuum* and that the shear stress and the rate of shear are related *linearly*. The latter assumption is well-known to be a first approximation, valid only for small rates of shear (2). Again, in the extreme conditions under which blood exhibits variable viscosity, neither of these assumptions can be readily justified.

(c) The "no-slip" boundary condition, on which the results of viscometry are based, may not be valid in these extreme circumstances; however, the vast body of experimental evidence in support of this condition (3) can hardly be dismissed by the type of curve-fitting argument presented by Nubar (1). Although he uses two major examples to justify his hypothesis of slip, his interpretation of them is questionable. First, in the case of a low density gas, which can no longer be treated as a continuum, the no-slip condition loses its meaning rather than its validity since this concept is only meaningful in the context of continuous media. Second, the described "skidding" of red cells is clearly due to lubrication of the wall with plasma as was proposed by Fitz-Gerald (4). This implies slip no more than the skidding of a solid body over an oily surface implies the slip of *oil* over that surface.

If the idea of slip is to be pursued rigorously, then its far-reaching implications, in both fluid and hemodynamics, must be carefully examined. Thus when two adjacent layers of fluid are forced to shear against each other, an opposing shear force is set up at the interface to oppose this departure from equilibrium. The magnitude of this "viscous" force is, roughly speaking, inversely proportional to the freedom of molecules to migrate from one point to another. If this shearing motion occurred at a blood-glass interface, one would expect this violent departure from equilibrium to be opposed by an equally violent viscous force that would tend to restore equilibrium by diminishing this velocity difference at the wall. The slip hypothesis, however, implies exactly the opposite, i.e., that the magnitude of this opposing viscous stress at a fluid-solid interface is lower than that at a fluid-fluid interface. We believe that there is no experimental or theoretical evidence to support this.

If slip does occur at a fluid-solid interface then the local velocity gradient (which measures the rate of shear of a fluid element in contact with the solid boundary) is undefined and, hence, the shear force at that interface is undefined. Nubar's expression for the shear stress at the wall, in the presence of slip, does not represent the shear interface very close to the

wall. These have quite different implications biologically, since blood-wall interactions may be important for the formation of thrombi or atherosclerotic plaques (5), while high shear forces within the blood itself would be more apt to cause hemolysis. Furthermore, if slip did occur at the walls of blood vessels, its obvious biological purpose would be to reduce the shear force at the wall of arteries with a consequent reduction in the after-load seen by the heart. Mathematically it is the wall-fluid interaction which must be inserted into the equations of motion. Thus Nubar's hypothesis that "the shear stress at the wall is independent of slip" is both mathematically incorrect and biophysically unlikely.

Finally, slip at an interface between two media depends on the nature of the interface as well as on the properties of *both* media. Therefore, even if slip is demonstrated at blood-solid interfaces in viscometers, a similar degree of slip need not occur at blood-vessel interfaces.

Since the constancy of blood viscosity is only an approximation, some variation (particularly under extreme conditions) does not constitute an anomaly. On the other hand, the no-slip condition is so well established experimentally that its contradiction would constitute a very serious anomaly. We thus believe that there is no justification for accepting slip as more than just one of the above three possibilities. In fact, we feel that of the three possibilities there is more evidence in support of the first two.

Dr. Zamir is supported by the National Research Council of Canada.

Dr. Roach is supported in part by the Ontario Heart Foundation.

Received for publication 29 December 1971.

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