Dear Sir,

In reply to my criticism of their work, Dr. Kaminsky et al. [1] introduce a number of potentially confusing ideas which seem to derive from their denial of the physical properties of blood. As early as in 1967, Nashat and Portal [2] pointed out that the difference in afferent and efferent arteriolar resistance is magnified by the fact that owing to the removal of glomerular filtrate, the viscosity of the blood traversing the efferent vessel is always greater than that traversing the afferent. In claiming that macroglobulinaemia is the prototype of hyperviscosity, Dr. Kaminsky et al. [1] fail to realise that blood viscosity is governed by a variety of factors. The most important determinant of blood viscosity is the red cell number, and polycythaemia is the simplest model of hyperviscosity. Although they dismissed the work of Loute et al. [3], it should be noted that there are other similar reports such as that of De Jong et al. [4] who noted that gradual reduction of haematocrit was associated with an increase in GFR and reductions in filtration fraction and protein excretion. Eight weeks after phlebotomy, it was recorded that there was a general movement of variables towards pre-phlebotomy levels.

In quoting our 1983 paper [5], Dr. Kaminsky et al. [1] reinforce the idea that they do not realise that red cell deformability is another factor contributing to whole blood viscosity. Thus, while Loute et al. [3] published a case report in which the viscosity effects arose from the increase in red cell numbers, our paper referred to the reduced deformability of hyperproteinaemia-induced echinocytes. In an associated paper [6], we reported '...the results of a small experiment designed to ascertain whether or not blood viscosity, increased because of the stiffening and deformation of the erythrocyte membrane, could be the cause of hyperproteinaemic proteinuria'.

Perhaps the cause of the problem facing Dr. Kaminsky et al. [1] is that they are attempting to evaluate a model of intraglomerular haemodynamics which takes no cognizance of the haemorheological determinants of blood flow. Because of the lack of similar rheological properties in whole blood, renal perfusion with non-blood fluids cannot produce information of physiological relevance.

References

