The Concept of Preload and Afterload Applied to Renal Hemodynamics

To the Editor:

I have enjoyed reading the fine review of de Jong et al (1) on the therapeutic strategies to lower proteinuria in glomerular disease and on the possible influence of these treatments on the progression of renal disease. However, I want to take issue with their proposal to borrow the terms “preload” and “afterload” from cardiology in describing the events following changes in preglomerular and postglomerular vascular resistance. In the Cecil Textbook of Medicine, preload is defined, in its strictest sense, as the stretch of myocardial fibers at the end of the diastole, whereas afterload is the resistance that the ventricle must overcome during systole in order to eject a stroke volume (2). This concept thus implies that the heart is a pump that actively delivers work (as in lifting a load). It is difficult to visualize such a function for the glomerular tuft.

In clinical practice, the term preload is often used as a synonym for the venous return to the heart. With this in mind, one could make a case to use a similar terminology in describing the preglomerular arterial flow. However, the analogy becomes rather confusing when the postglomerular vascular changes are described in terms of afterload, because the glomerular tuft does not contract against an efferent resistance.

The terminology, as proposed by the authors, certainly has its attractiveness in that it attempts to describe complicated phenomena in a concise way. It might seem an acceptable jargon for the knowledgeable nephrologist, but it will tend to confuse those who feel less well at home in the field of renal hemodynamics. Therefore, I would like to make a plea to stick to the more precise description of these phenomena in terms of the glomerular capillary hydraulic pressure gradient, influenced by systemic arterial pressure and by the changes in afferent and efferent vascular resistance.

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REFERENCES


Response:

We thank Professor Koene for his comments on our concept of glomerular preload and glomerular afterload reduction. Indeed, he is obviously correct in that the function of the glomerular capillary loop cannot be compared with that of the heart, the first being a specialized structure that affords the filtration of water and solutes, whereas the latter is an active pump.

We however used the concept of preload and afterload reduction not to directly compare the function of the glomerulus with that of the heart, but to compare the function of the vessels to and from the glomerulus with the function of the vessels to and from the heart. By modulation of the resistances of these vessels, the function of the organ positioned intermediate between the vessels can be influenced greatly. This holds true both for the heart and for the glomerulus. The function of the heart may be improved by either cardiac preload or afterload reduction or by the combination of both. Similarly, a glomerular preload reduction (afferent vasoconstriction), a glomerular afterload reduction (efferent vasodilation), or a combination of both can influence the filtration properties of the glomerulus.

We argue for this concept because it draws attention, as it has been done in cardiology, to the fact that the function of the organ may be influenced in different ways. One should not rely only upon the treatments that influence glomerular afterload (such as angiotensin-converting enzyme inhibitors) but also look for treatments that lower glomerular preload (such as low-protein diets). If clinically feasible, the combination of both can, in some instances, be the treatment of choice. In that respect, it is noteworthy that it has recently been shown that the antiproteinuric effect of an angiotensin-converting enzyme inhibitor adds to that of a low-protein diet (1,2).