Dear Sir,

Previous reports concerning the hemodialysis-induced change in plasma antithrombin III (AT-III) concentration have been controversial. Turney et al. [7] have found subnormal levels of the substance in uremic patients which were restored to normal by hemodialysis. Contrary to this, Jorgensen and Stoffersen [4] and Brandt et al. [1] observed a small but significant decrease in circulating AT-III after hemodialysis. We [2] have recently reported slight but not unequivocal changes in plasma AT-III in uremic patients after dialysis with either Cuprophan or polyacrylonitrile membrane; no correlation with the respective changes in platelet counts was found. It has been questioned whether the different methodology of analysis (i.e., radial immunodiffusion or functional assay) could account at least in part for the contradictory results. But the good correlation between the functionally and immunologically determined plasma AT-III levels in uremic patients before as well as after dialysis [1] shows that both methods are applicable in this clinical setting. In view of the effect of intravenous heparin administration on circulating AT-III [6], we agree that difference in the heparinization schedule largely account for this discrepancy in AT-III levels after dialysis in the different reports. In our study [2], each patient was given a total dose of 2,000 IU of heparin during hemodialysis.

Two recent reports [3, 8] dealing with the levels of plasma AT-III after dialysis add further confusion to this already complicated issue. They both showed a significant postdialysis increase of the substance and speculated on the possible responsible mechanism(s). Woo et al. [8] theorized that this elevation could be due to the release of AT-III by damaged platelets within the dialysis circuit, as perviously suggested [5], and/or to the removal of hypothetical dialyzable toxins which suppress liver as well as endothelial production and release of the substance. However, the authors failed to evaluate either serum proteins or hematocrit before and after dialysis in their patients. Without this information, the possibility that the observed postdialysis elevation of AT-III merely reflects fluid removal during hemodialysis cannot be excluded. Conversely, Jorgensen et al. [3], who indeed attributed the postdialysis elevation of AT-III in their patients to hemoconcentration, did not report the change, if any, in platelet counts after dialysis. But, as stressed above, the possible platelet damage related to dialysis may have a sometimes important role in this respect. In our opinion, these studies, while demonstrating an unequivocal increase in plasma AT-III after dialysis, do not provide any plausible explanation for this because of the above mentioned methodological flaws.
References