letters to the editor

etorre guidedi
milan, italy

correspondence to ettore guidedi, m.d., clinical research center in nephrology and arterial hypertension, ospedale niguarda-ca' granda, piazza ospedale maggiove 3, 20162 milano, italy.

e-mail: etguid@tin.it

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reply from the author

i read with interest dr. guidedi’s comments about our recently published work in kidney international [1]. dr. guidedi postulates that the donor kidney may determine both the risk of hypertension post-transplant and the risk for rejection. consistent with the first portion of this hypothesis, we showed previously that the blood pressure levels in pairs of individuals who receive kidneys from the same donor are highly correlated [2]. the pathogenic link between donor graft and recipient’s blood pressure post-transplant may be related to (1) graft damage during preservation; (2) preexisting graft vascular damage in the donor prior to donation and due to hypertension; or (3) other genetic characteristics of the donor graft in patients with familial hypertension [3]. the data presented in our recent paper [1] are at least partially consistent with the postulate that the donor graft also determines, in part, the risk of rejection. however, it is much more difficult for us to postulate a plausible pathogenic mechanism for this association, unless it is the hypertension itself, causing endothelial changes, that predisposes to immune phenomena.

fernando g. cosio

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preventing dialysis hypotension: a comparison of usual protective maneuvers

to the editor: we read with great interest the article of dheenan and henrich [1], in which the effects of sodium modeling, high sodium dialysis, cool temperature dialysis, and isolated ultrafiltration on intradialytic hypotension were compared. the authors suggest that sodium modeling and cool temperature dialysis should be used as a first-line step in the prevention of intradialytic hypotension with a higher dialysate sodium concentration as a reasonable alternative.

indeed, we fully agree with the authors that isolated ultrafiltration followed by isovolemic dialysis will certainly have a disadvantage over cool temperature dialysis, which is understandable. the initial ultrafiltration rate was much

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higher during isolated ultrafiltration compared to cool temperature dialysis, whereas vascular reactivity is comparable between these two treatment modalities [2].

We would, however, like to add a word of caution with regard to the use of sodium modeling and high sodium dialysate. Data with regard to sodium fluxes during different dialysate sodium concentrations are scarce. Nevertheless, in a pilot study including 11 dialysis patients, in which conductivity measurements were used to assess ionic dialysance [3], we observed a mean ionic dialysance of 321 mmol during dialysis with a dialysate sodium concentration of 140 mmol/L, compared to 231 mmol with a dialysate sodium concentration of 144 mmol/L despite a similar ultrafiltration volume. This means a reduction in sodium removal of 90 mmol during high compared to standard sodium dialysis, which corresponds to 2100 mg sodium (the recommended daily sodium intake of a dialysis patient). With regard to sodium modeling, no data on ionic mass balance are as yet available. However, the fact that dialysate sodium was reduced from 152 to 140 mmol in the protocol used by Dheenan and Henrich will undoubtedly have led to reduced sodium removal compared to the cool dialysis treatment in which a dialysate sodium concentration of 140 mmol was used. In view of the very small and nonsignificant hemodynamic advantage of sodium modeling over cool dialysis, and regarding the relation between salt loading, hypertension, and left ventricular hypertrophy [4], we suggest that cool dialysis should be used as the single first-line step in the prevention of hypotensive episodes during dialysis. More data with regard to sodium fluxes during sodium modeling are needed before it can be recommended as a standard treatment maneuver in the prevention of hemodynamic instability in hemodialysis patients.

Jeroen P. Kooman, Karin Moret, Frank M. van der Sande, Paul G.G. Gerlag, A. Warmold van den Wall Bake, and Karel M.L. Leunissen
Maastricht, The Netherlands

Reply from the author

Kooman et al make two key points in their comments. First, the isolated ultrafiltration followed by isovolemic dialysis protocol had a higher initial ultrafiltration rate than the other protocols and this design placed the procedure at a comparative disadvantage. We agree with this point and included a caveat about the different ultrafiltration rate in our paper. In designing the study, we wanted to replicate usual clinical practice, and, in most units, this is similar to how isolated ultrafiltration is applied. We agree that the use of isolated ultrafiltration may be better tolerated in patients who have lower ultrafiltration requirements.

Second, Kooman et al mention the issue of sodium removal during sodium-modeling protocols. We agree that, over time, a cumulative positive sodium balance could be a consequence of sodium modeling. This, in turn, would lead to hypertension in some patients, and this occurrence, although not a uniform finding, has been observed previously [1, 2]. Kooman et al argue that cool temperature dialysis may therefore be, on balance, a more preferred approach to episodic intradialytic hypertension. There are two aspects of this suggestion to keep in mind. First, cooler temperature dialysis is not tolerated by all patients; in our experience, about 20% of patients are unable to tolerate it. Second, the procedure is effective via the release of catecholamines, sympathetic nervous system activation, and an increase in left ventricular contractility [3, 4]. Patients with silent coronary disease (with a critical stenosis) may, therefore, come to clinical attention with the therapy. In sum, our paper suggests that sodium modeling and cooler temperature dialysis are the most effective, although not perfect therapies for this problem, and we agree with Professor Kooman and colleagues that individual tailoring of these therapies to meet specific patient needs is warranted.

Sunita Dheenan and William L. Henrich
Toledo, Ohio, and Baltimore, Maryland

Correspondence to William L. Henrich, M.D., Department of Medicine, University of Maryland School of Medicine, 22 S. Greene Street (N3W42), Baltimore, Maryland 21201, USA.

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