tion of renal replacement therapy, cardiac performance may change as a function of PTH level but not as the effect of duration of therapy. Such a possibility is suggested by the data presented in Table 3. Results given in the second and third columns show that cardiac mass, volume and function indexes deteriorate in some patients but improve or do not change in others. The differences may depend on mode of dialysis and PTH concentration.

Therapy with EPO leads to the regression of LVH in dialysis patients. The mean hemoglobin level in the study group presented in the first column of Table 4 is 8.6 g/dl (range, 8.1–9.2), but the data plotted in Figure 1 reveal that as many as 9 HD patients had a hemoglobin level below 8 g/dl. We are curious as to whether these patients, particularly those with hemoglobin levels of 6–7 g/dl, were given EPO. If yes, it should be mentioned, because this hormone affects cardiac function regardless of renal replacement therapy. If EPO was not given to these patients, one wonders why the authors did not address the severe anemia.

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REFERENCES

1. Foley RN, Parfrey PS, Kent GM, Harnett JD, Murray DC, Barre PE: Long-term evolution of cardiomyopathy in dialysis patients. *Kidney Int* 54:1720–1725, 1998
2. Cohen JL, Barooah B, Segal KR, Batuman V: Two-dimensional echocardiographic findings in patients on hemodialysis for more than six months. *Am J Cardiol* 60:743–745, 1987
3. Leenen FH, Smith DL, Khanna R, Oreopoulos DG: Changes in left ventricular hypertrophy and function in hypertensive patients on continuous ambulatory peritoneal dialysis. *Am Heart J* 110:102–106, 1985
4. Harnett JD, Parfrey PS, Griffiths SM, Gault MH, Barre P, Guttmann RD: Left ventricular hypertrophy in end-stage renal disease. *Nephron* 48:107–115, 1988
5. Hara S, Ubara Y, Arizono K, Ikeguchi H, Katori H, Yamada A, Ogura Y, Murata H, Mimura N: Relation between parathyroid hormone and cardiac function in long-term hemodialysis patients. *Miner Electrolyte Matab* 21:72–76, 1995

To the Editor: We would like to comment on the study of Foley et al [1]. In our opinion it is not correct to speak about the "natural history of cardiomyopathy" because the development of this cardiomyopathy is highly dependent on the way patients are treated, particularly the extent to which the treating team succeeds in controlling "volume."

The fatalistic attitude that unfortunately prevails in many dialysis centres is supported by the conclusion that "progressive left ventricular dilatation with compensatory hypertrophy is the major long-time evolutionary pattern" and that "intervention beyond one year may be relatively ineffective."

The fact that better results were obtained with peritoneal dialysis may simply be due to better volume control in those patients. Other studies [2] have shown that this is not always the case. The absence of a relationship with blood pressure could be related to the confounding effect of antihypertensive drugs, but probably also to an independent effect of volume retention.

We recently showed [3] that prolonged strict volume control can cause regression of dilatation and hypertrophy of the left ventricle while abolishing the need for antihypertensive drugs. An extreme example of a patient in whom volume control caused complete regression of severe cardiomyopathy without hypertension [4] illustrates the importance of hypervolemia as an independent risk factor. Clearly (as Foley et al stressed in earlier publications) only intervention studies can definitely resolve the complicated problems of dialysis morbidity.

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REFERENCES

1. Foley RN, Parfrey PS, Kent GM, Harnett JD, Murray DC, Barre PE: Long-term evolution of cardiomyopathy in dialysis patients. *Kidney Int* 54:1720–1725, 1998
2. Lameire N, Vanholder RC, Van Loo A, Lambert MC, Vijt D, Van Bockstaele, Vogeleere P, Ringoir SM: Cardiovascular diseases in peritoneal dialysis patients: the size of the problem. *Kidney Int Suppl* 56:S28–S36, 1996
3. Özkahya M, Ok E, Cirit M, Aydin Ş, Akçiçek F, Başçi A, Dorhout Mees EJ: Regression of left ventricular hypertrophy in haemodialysis patients by ultrafiltration and reduced salt intake without antihypertensive drugs. *Nephrol Dial Transplant* 13:1489–1493, 1998
4. Töz H, Özerkan F, Ünsal A, Soyda C, Dorhout Mees EJ: Dilated uremic cardiomyopathy in a dialysis patient cured by persistent ultrafiltration. *Am J Kidney Dis* 32:664–668, 1998

Reply from the authors

To the Editor: We would like to thank all the correspondents for their interest and helpful queries and comments.

Dr. Erturk makes several reasonable points. It certainly would have been of interest to look at the potential role of post-dialysis blood pressures in the hemodialysis patients. The short answer is that we did not record them, partly reflecting the fact that the study began in the early 1980s. It is hardly fair to say that post-dialysis blood pressure is a better *predictor* of LV mass index than pre-dialysis blood pressure. We have previously shown, in a prospective cohort study of over a thousand