Letters to the Editor

The general population has been demonstrated for body size [2] and serum cholesterol [3] and has been postulated to be due to malnutrition [3]. We agree with Canavese et al that a great deal of research is still necessary regarding the biological mechanisms of homocysteine metabolism in patients with ESRD and their relationship to cardiovascular risk.

The finding of differing TT genotype prevalence over years of dialysis reported in these two studies is not irreconcilable. There may be other differences in the design of the studies that might explain variable risk estimates, including clinical status, sample size, measurement issues, and race/ethnicity composition. We intend to examine follow-up data from our study and agree that experience reported from other centers is greatly needed.

Routine screening of either homocysteine or genetics in not routinely recommended. A professional organization weighing the merits of such a clinical policy should take into consideration the cost-effectiveness of the test and associated interventions. In our clinic, the costs of such tests are significantly lower than those mentioned by Canavese et al.

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Daily hemodialysis and nutritional status

To the Editor: A switch from conventional hemodialysis (CHD) to daily hemodialysis (DHD) has been found to improve nutritional status of hemodialysis patients [1]. This observation by Galland et al needed to be confirmed. We, too, evaluated the effect of a switch from CHD (3 × 4 hours/week) to DHD (6 × 2 hours/week), using bioelectrical impedance analysis (BIA); we also observed a dramatic improvement in patients’ lean body mass.

Fourteen volunteer patients were evaluated during 3 months on CHD and 12 months on DHD. The lean body mass was evaluated at −3, 0, +6 and +12 months, respectively. Five patients were excluded (renal transplantation and aortic valve replacement, respectively). Patients. Am J Kidney Dis 31:997–1006, 1998

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ACKNOWLEDGMENTS

The authors thank Dr. Ingrid Ledebo from Gambro, Sweden, for her support, and Professor M. Jadoul for his comments.

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To the Editor: In uremic patients, hyperhomocysteinemia was found to be unrelated to measurements of oxidative stress [1]. The data imply that hyperhomocysteinemia is no primary risk factor for vascular lesions and thrombotic events in chronic renal failure.

Moderate hyperhomocysteinemia is considered to develop when dietary supplementation of folate is insufficient. Accordingly, folate supplementation can easily correct hyperhomocysteinemia, and an inverse relationship between homocysteine and folate concentrations usually exists, which was also observed in the study by Mezzano et al [1]. Hyperhomocysteinemia often coincides with elevated neopterin levels, which indicate immune activation and oxidative stress emerging from activated macrophages [2], and, in patients with uremia, increased neopterin concentrations are associated with protein oxidation products [3]. Also in the study of Mezzano et al, oxidation products correlated with markers of immune activation [1]. 5,6,7,8-Tetrahydrofolic acid, the biologically active cofactor, is very susceptible to oxidation, which may become relevant under oxidative stress conditions. Immune system-derived oxidative stress seems to be crucial for folate depletion, resulting in hyperhomocysteinemia even when dietary folate is within the recommended range [2]. In coronary artery disease, folate supplementation was found to improve endothelial function earlier than changes of homocysteine concentrations became apparent [4], and also the data of Mezzano et al support the view that hyperhomocysteinemia is an indirect consequence of hyperconsumption of antioxidant vitamins during prolonged states of immune activation. Then, hyperhomocysteinemia is not primary in the pathogenesis of vascular lesions and thrombotic events. Nevertheless, hyperhomocysteinemia still could contribute to accelerate the underlying pathogenic process.

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