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REFERENCES

- MAGNUSSON P, SHARP CA, MAGNUSSON M, *et al*: Effect of chronic renal failure on bone turnover and bone alkaline phosphatase isoforms. *Kidney Int* 60:257–265, 2001
- MAGNUSSON P, LARSSON L, MAGNUSSON M, *et al*: Isoforms of bone alkaline phosphatase: Characterization and origin in human trabecular and cortical bone. *J Bone Miner Res* 14:1926–1933, 1999
- UREÑA P, HRUBY M, FERREIRA A, *et al*: Plasma total versus bone alkaline phosphatase as markers of bone turnover in hemodialysis patients. *J Am Soc Nephrol* 7:506–512, 1996
- GARNERO P, DELMAS PD: Assessment of the serum levels of bone alkaline phosphatase with a new immunoradiometric assay in patients with metabolic disease. *J Clin Endocrinol Metab* 77:1046–1053, 1993

Reply from the authors

We welcome the letter from Dr. Ureña Torres as it offers us an opportunity to clarify and emphasize several aspects of our measurements of the different bone alkaline phosphatase (BALP) isoforms in patients with chronic renal failure (CRF) [1]. In reply to his queries regarding questions 1 and 2, total ALP was significantly increased; however, the majority of the patients had activities within the reference interval for healthy adults. We agree with the suggestion that the increase of BALP isoform B2 in CRF patients may be clinically useful. None of the patients in this study had any biochemical or clinical evidence of hepatic disorder. To answer the third question, we used the previously reported reference intervals for all three BALP immunoassay kits (Alk-phase-B, Tandem-R Ostase and Tandem-MP Ostase) and refer him to Figure 2 [1]. To respond to his fourth question, discordant findings between different studies are not uncommon, which probably reflects the heterogeneity of bone disorders in CRF patients. We did, however, find a significant correlation between the novel BALP isoform B1x and PTH, which might contribute to the positive correlations previously reported [2].

We suggested that B1x should be further evaluated as a marker of adynamic bone disease. This will indeed require a classification of patients by bone histomorphometry, which was not obtained in this study. Although adynamic bone disease is usually associated with relatively low parathyroid hormone (PTH) levels, PTH may fail to discriminate between adynamic and moderate hyperparathyroid states and even high PTH levels may occur [3]. Another important point, discussed in our paper [1], is that PTH was analyzed using a commercial assay originally reported to detect only the intact (1-84 PTH) circulating molecule. However, it has recently been demonstrated that a fragment (most likely the 7-84 PTH) interferes with this assay [4]. Thus, the PTH values reported in our study (and other studies) might well be

higher than the true circulating levels of intact 1-84 PTH. Bone mineral density was not assessed and we prefer not to speculate as to whether these patients were osteopenic or osteoporotic.

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REFERENCES

- MAGNUSSON P, SHARP CA, MAGNUSSON M, *et al*: Effect of chronic renal failure on bone turnover and bone alkaline phosphatase isoforms. *Kidney Int* 60:257–265, 2001
- UREÑA P, HRUBY M, FERREIRA A, *et al*: Plasma total versus bone alkaline phosphatase as markers of bone turnover in hemodialysis patients. *J Am Soc Nephrol* 7:506–512, 1996
- MONIER-FAUGERE M-C, MALLUCHE HH: Calcitriol pulse therapy in patients with end-stage renal failure. *Curr Opin Nephrol Hypertens* 3:615–619, 1994
- SLATOPOLSKY E, FINCH J, CLAY P, *et al*: A novel mechanism for skeletal resistance in uremia. *Kidney Int* 58:753–761, 2000

Prediction of hypertension in hemodialysis patients

To the Editor: I have read with much interest the paper by Agarwal and Lewis [1] on prediction of hypertension in chronic hemodialysis patients. In the introduction, the authors focus on the fact that it is still uncertain which blood pressure measurement the clinician has to adopt to define hypertension in these patients. There is no question that in the general population 24-hour ambulatory monitoring is a better measure than the office measure. It is well-documented that the ambulatory estimate is superior to the office estimate for predicting incident cardiovascular complications, as well as left ventricular hypertrophy (LVH) [2], which is a valid surrogate end point. Whether or not 24-hour ambulatory monitoring predicts survival and cardiovascular complications in the dialysis population still remains to be proved. This is important mostly because two surveys have shown that routine pre-dialysis blood pressure and 24-hour ambulatory monitoring explain to a similar degree the variance in left ventricular mass. Both the paper by Conlon *et al* [3] and our study based on multivariate modelling [4] have clearly shown the strength of the association between 24-hour ambulatory monitoring and left ventricular mass is not superior to that of pre-dialysis blood