sure and left ventricular mass in stable hemodialysis patients. *J Am Soc Nephrol* 7:2658–2663, 1996
7. Agarwal R, Lewis RR: Prediction of hypertension in chronic hemodialysis patients. *Kidney Int* 60:1982–1989, 2001
8. Peixoto AJ, Santos SF, Mendes RB, *et al*: Reproducibility of ambulatory blood pressure monitoring in hemodialysis patients. *Am J Kidney Dis* 36:983–990, 2000

The value of the (1-84) PTH/C-PTH ratio for the diagnosis of low bone turnover in dialysis patients

To the Editor: In a recent issue of *Kidney International*, Monier-Faugere et al [1] clearly demonstrated the superiority of the parathyroid hormone (1-84) PTH/C-PTH fragment ratio in predicting bone turnover. However, we wonder why the authors were only interested by the diagnosis of low bone turnover since their patients had normal plasma calcium while taking calcium carbonate ($CaCO_3$) but no calcitriol and had not been exposed to aluminum phosphate binder. Indeed, in such patients, low bone turnover is not an actual bone disease, in contrast to what occurs in patients with previous aluminum overload [2]. In the present study, the major clinical concern is hyperphosphatemia, and this abnormality was worse in the group with normal or high bone turnover since it was 7.1 mg/dL, that is, above 6.5, the threshold for which an increased mortality risk has been observed [3]. It would have been more clinically relevant to know whether hyperphosphatemia was associated with high bone turnover and to show the superiority of their ratio for this latter diagnosis. With the Biosource RIA, using the Bouillon antibodies that also measures (1-84) PTH at the exclusion of (7-84) PTH, we reported in 1991 that in 23 hemodialysis patients never exposed to aluminum, the nine patients with osteitis fibrosa had levels above 1.7, the upper limit of normal, whereas all the six patients with low turnover had levels below this limit [4]. Out of the eight patients with normal turnover, three had levels between 1.0 and 1.7 this limit, four within the normal range and one below this range.

Albert Fournier, Roxana Oprisiu, Delia Bunea, and Bahloul Hedi
Amiens, France

Correspondence to Albert Fournier, Hôpital Sud – Service de Néphrologie – 80054 AMIENS CEDEX – France.
E-mail: Fournier.Albert@chu-amiens.fr

REFERENCES

1. Monier-Faugere MC, Geng Z, Mawad H, *et al*: Improved assessment of bone turnover by the PTH-(1-84)/large C-PTH fragments ratio in ESRD patients. *Kidney Int* 60:1460–1468, 2001
2. Fournier A, Said S, Ghazali A, *et al*: The clinical significance of adynamic bone disease in uremia, in *Advances in Nephrology Year Book*, edited by Grünfeld JP, Mosby, St Louis, 1997, Vol 27, pp 131–166
3. Block G, Hubert-Shearon T, Levin N, Port F: Association of serum phosphorus and calcium phosphorus product with mortality risk in chronic hemodialysis patients. A national study. *Am J Kidney Dis* 31:607–617, 1998
4. Fournier A, Solal ME, Oprisiu R, *et al*: Optimal range of plasma concentration of true 1-84 parathyroid hormone in patients on maintenance dialysis. *J Clin Endocrinol Metab* 86:1840–1842, 2001

Reply from the authors

We thank Dr. Fournier and his colleagues for their interest in our recently published study [1] and would like to offer the following answers to their comments.

Nephrologists face the challenge of differentiating low bone turnover from normal-high bone turnover because of the immediate therapeutic ramifications, that is, normal-high bone turnover should be treated with vitamin D while low bone turnover is not amenable to vitamin D treatment. Vitamin D therapy in patients with adynamic bone disease is associated with elevation in calcium phosphate product, secondary to improved intestinal calcium absorption with inability to maintain normal bone calcium accretion [2]. It is generally accepted that uncontrolled calcium phosphate product is associated with increased cardiovascular calcifications and mortality. It is not justified to assume absence of disease merely because of presence of normal serum calcium.

The described diagnostic approach for low bone turnover inherently allows the recognition of normal-high bone turnover by exclusion.

We are sorry that Dr. Fourner et al are disappointed that serum phosphorus levels are not diagnostic for bone turnover but our data simply support statistically a well-known clinical observation.

Marie-Claude Monier-Faugere and Hartmut H. Malluche, for the authors
Lexington, Kentucky

Correspondence to: Hartmut Malluche, M.D., Division of Nephrology, Bone and Mineral Metabolism, University of Kentucky Medical Center, Room MN 564, Lexington, KY 40536-0084, USA.
E-mail: hhmall@uky.edu

REFERENCES

1. Monier-Faugere MC, Geng Z, Mawad H, *et al*: Improved assessment of bone turnover by the PTH-(1-84)/large C-PTH fragments ratio in ESRD patients. *Kidney Int* 60:1460–1468, 2001
2. Kurz P, Monier-Faugere MC, Bognar B, *et al*: Evidence for abnormal calcium homeostasis in patients with adynamic bone disease. *Kidney Int* 46:855–861, 1994