Letters to the Editor

progression of renal disease. Also, as we discuss in our article, it could be the focus of therapeutic interventions to slow progression of renal disease.

In light of the above, we suggest that the technical concerns of Mr. Basgen and Dr. Mauer do not invalidate the conclusions of our article. Nevertheless, their concerns are highly appropriate. Indeed, we endorse their proposal that scientific journals should adopt the high standards proposed by Nyengaard for morphometric studies. Our endorsement, however, is modified in two respects. First, Nyengaard’s article is too technical. Investigators may be dissuaded from doing morphometric studies if they will be judged by the principles contained in his article. Second, we suggest that a much simpler, applied version of Nyengaard’s principles should be published. Dr. Mauer and his group would be ideal candidates to execute such work. Dr. Mauer is a bellwether investigator in this field and is noted for his lucid exposition of data.

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Different response of urinary excretion of VEGF in patients with chronic and acute renal failure

To the Editor: Vascular endothelial growth factor (VEGF) is constitutively expressed by epithelial cells of a nephron from embryonic to adult kidneys. In renal disease, VEGF appears to be involved in a repair of the glomerulus [1]. It is excreted in urine [2] and is suspected to be secreted from tubular cells by hypoxic stimulation in vitro [3, 4] and in vivo [4]. Thus, because the amount of urinary VEGF might reflect renal hypoxia, we measured it in patients with renal diseases and various renal functions.

A total of 29 urine and 26 serum and plasma samples were collected from 22 non-diabetic non-nephrotic patients without serious complications. Fifteen patients had a renal biopsy (11 with IgA nephropathy, two with membranous nephropathy, one with focal segmental glomeru-

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criteria for calcimimetic agent in the treatment of more severe secondary hyperparathyroidism

To the Editor: The prevention and treatment of secondary hyperparathyroidism still are challenging for the nephrologist. Therefore, the recent work of Goodman et al. is very interesting and promising [1]. The goal of their study was to assess the safety and efficacy of a calcimimetic agent in dialysis patients with modest to severe secondary hyperparathyroidism. However, their study appears to focus only on a single criterion for modest or severe secondary hyperparathyroidism, the level of intact parathyroid hormone (PTH). It is well known that secondary hyperparathyroidism is a state of increased synthesis and secretion of PTH, but also a state of increased hypertrophy and particularly hyperplasia of the parathyroid cells [2]. There are two forms of parathyroid gland hyperplasia, diffuse and nodular. Parathyroid glands with nodular hyperplasia are larger. In 90% of parathyroid glands weighing 0.5 g nodular hyperplasia was found. Such nodules are made of cells with significantly reduced numbers of vitamin D receptors, calcium-sensing receptors (CaSR), and a higher calcium set point for PTH secretion. From a clinical point of view, it is important to note that patients with nodular parathyroid gland hyperplasia are often resistant to calcitriol therapy.

Today, high-resolution sonography is the best technique to determine the shape and size of abnormal parathyroid glands [3]. Therefore, it would be more appropriate to use not only a level of PTH for more severe hyperparathyroidism, but also the size of the parathyroid glands as criteria. As large parathyroid glands with nodular hyperplasia have reduced numbers of CaSR, it would be interesting to see the effect of a calcimimetic agent in dialysis patients with large parathyroid glands (that is, suspected nodular hyperplasia).

Despite these remarks, we hope that a new calcimimetic agent, as well as new phosphate binders and new vitamin D analogues, will be useful in the treatment of dialysis patients.

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REFERENCES


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