losclerosis, and one with membranoproliferative glomerulonephritis). The other seven patients were clinically diagnosed with chronic renal failure. Twelve urine and serum samples were collected from three patients with ischemic acute renal failure. As VEGF concentration in the urine did not change after 24 hours' incubation at room temperature, those urine samples were stored at −30°C until use and submitted to colorimetric enzyme immunoassay to quantitate VEGF. Renal function was evaluated by 24-hour creatinine clearance (C_{Cr}).

There was an inverse relationship between urinary excretion of VEGF and C_{Cr} (Fig. 1A). VEGF excretion in urine did not correlate with its serum (Fig. 1B) or plasma level (data not shown). In patients with acute renal failure, no correlation was observed between urinary excretion of VEGF and C_{Cr} (Fig. 1C).

We examined patients with a wide range of renal functions to focus on the relationship between urinary VEGF and renal function. In patients with chronic renal failure, VEGF excretion in urine increased as renal function decreased. Since excretion was independent of the serum level of VEGF, urinary VEGF appears to be derived from the kidney. Therefore, increased excretion of VEGF suggests its increased secretion in residual nephrons under diffuse and continuous hypoxia. In patients with acute renal failure, urinary VEGF excretion was independent of renal function, probably because excretion of VEGF from the kidney did not change much since the ischemic period was transient and the area was limited. Although further studies are needed, urinary VEGF might be a unique indicator of renal hypoxia.

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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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Relationship between birth weight, glomerular number, and glomerular size

To the Editor: In a recent issue of *Kidney International* Mañalich et al confirm previous reports of a positive relationship between birth weight and glomerular number, and describe an inverse relationship between birth weight and glomerular size, a novel observation [1]. It is regrettable, however, that they chose the arbitrary value of 2500 g to distinguish "normal" from "low" birth weights since the gestational age of the infants ranged from 36 to 41 weeks. A weight of 2500 g is in approximately the 25th percentile at 36 weeks but well below the 3rd percentile at 41 weeks. This is important because other studies indicate that intrauterine growth retardation (IUGR), not low birth weight per se, is associated with oligonephronia in humans [2] (abstract; Leroy et al, *Ped Nephrol* 6:C21, 1992) as well as in rats [3]. It would be interesting to know if the difference in nephron numbers remained the same if the infants were divided according to whether their birth weights were above or below the 10th percentile for gestational age, a widely accepted criterion for IUGR. It would also be interesting to know whether there was a relationship between the presence or absence of IUGR and kidney weight, corrected for gestational age, as has been reported previously. The choice of symbols used in the figures was confusing. Infants were described as black, white, or at a gestational age younger than 38 weeks. Presumably those born before 38 weeks were also either black or white. Why separate the more mature infants by race but not these?

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Reply from the authors

We used the value of 2500 g to divide "low" from "normal" weight at birth in our infants, regardless of whether the gestation was 36 or 41 weeks, following the guidelines of the World Health Organization [1, 2]. Only four of our infants with "normal" weight at birth could be considered to have intrauterine growth retardation (IUGR), as defined in the tables presently used in our country [3]. The issue of the relationship between IUGR and nephron number and size is important and not directly addressed in our paper. However, our results indicate a significant correlation between birth weight and the number of nephrons, as shown by others, and a significant inverse correlation between the number and the size of glomeruli, a novel finding, as correctly noted by Dr. Haycock.

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