sence of hyperglobulinemias and disproteinemias (New J Med 281:1428, 1969). Our simplified and practical proach to the diagnosis, surveillance and treatment of the ole spectrum of cases of consumption coagulopathy has n described elsewhere.[5,6] No single test is universally use- in the diagnosis and management of consumption coagu- pathy. We have not yet uncovered all the secrets that the T may yield to us. In our view this test of Breen and llis deserves the most serious attention from those inter- ed in problems of consumption coagulopathy.

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IDIOPATHIC HYPERCALCIURIA

To the Editor: The paper by Finn et al. in the December 24, 1970, issue of the *Journal*, entitled "Transplantation of a Kidney from a Patient with Idiopathic Hypercalciuria," purports to show that idiopathic hypercalciuria is caused by excessive gastrointestinal absorption of calcium rather than by an inability of the kidney to conserve calcium. The kidney of a donor with idiopathic hypercalciuria was transplanted into a recipient who did not go on to acquire idiopathic hypercalciuria himself. The donor, however, persisted in having this abnormality.

The problem with the conclusion reached from this retrospective case is that the recipient was, of course, placed on steroids after the transplant, and steroids are known to decrease intestinal absorption of calcium, as indicated by Harrison and Harrison, in their article entitled "Transfer of Ca^{45} across Intestinal Wall in Vitro in Relation to Action of Vitamin D and Cortisol." (Amer J Physiol 199:265, 1960). This could have affected his urinary excretion of calcium also.

If one wanted to use this case to demonstrate the primary role of increased gastrointestinal absorption of calcium in idiopathic hypercalciuria, one would have had to place the donor with idiopathic hypercalciuria on steroids, or a dietary calcium of only 150 mg per day, before the nephrectomy and transplantation. If the donor had responded to this program by decreasing his urinary calcium excretion to normal levels before transplantation, the post-transplant data presented in their paper would have been unnecessary. However, if the donor's urinary calcium excretion had continued to be high under these circumstances, the recipient's normal urinary calcium excretion after transplantation would have been important, indicating a nonrenal origin for the condition.

If this study had been carried out before transplantation, we would have more meaningful data. I agree with the authors' basic contention, but believe that they missed a chance to prove it. Transplantation can only help us with this question if we take a patient as donor whose calcium excretion in the urine does not decrease to normal despite the severest calcium restriction in the diet, or if steroids are not given after operation to the recipient.

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The above letter was referred to the authors of the article in question, who offer the following reply:

To the Editor: The study was not a retrospective one, and calcium balance studies carried out in the donor before nephrectomy demonstrated hypercalciuria on a diet of both 300 mg and 1 g of calcium. Hypercalciuria disappeared immediately in the transplanted kidney, but balance studies were carried out one year later to avoid the effect that chronic uremia might have had on calcium metabolism in the early post-transplantation period. Contrary to Dr. Silber's opinion, studies of patients with idiopathic hypercalciuria have demonstrated that although urinary calcium decreases with calcium restriction, negative calcium balance occurs with inappropriately high urinary calcium for 10 to 18 days. Whether negative calcium balance would persist during a longer period of severe restriction of calcium intake is unknown. Reducing calcium intake for a brief period, however, does not differentiate two populations of patients, one with a primary renal leak of calcium and another with excessive gastrointestinal absorption.

Regarding Dr. Silber's objection that steroids were not given to the donor before nephrectomy, as we pointed out in our article, all studies of idiopathic hypercalciuria in which steroids have been administered demonstrate further increase in urinary excretion,[1-3] and we did not consider it justifiable to subject the donor to this potential risk.

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