


Discussion

COLIN G. THOMAS, JR. (Chapel Hill, North Carolina): President Wells, Secretary Copeland, Members, and Guests. Based upon a very rigorous and critical review of the manuscript, which I received well in advance, and after hearing the presentation, I am compelled to conclude that this is a very fine piece of work, and Dr. Diethelm and his colleagues fully deserve our congratulations. This is a remarkable experience and noteworthy in several respects.

With a data base of over 4000 transplant recipients, 29 years of observation, less than 1% of patients required parathyroidectomy for continuing hyperparathyroidism. The clinical results are excellent with minimal morbidity. Their recommendations are well-founded and should be valuable to all of us in the management of these challenging problems. However, I do have a few questions.

The reasons for persistent hyperparathyroidism have been stated to be related to a variety of factors such as impaired renal graft function, autonomy of parathyroid function, persistence of high calcium set point for setting off the parathyroid insufficient calcitriol secretion. Do the authors have an opinion as to the mechanism that best explains the persistent hyper function of these parathyroid glands?

In the asymptomatic patient, a calcium level of 12 mg per dL constitutes an indication for parathyroidectomy. How is this figure derived? It is higher than that recommended at a recent NIH conference for patients with primary asymptomatic hyperparathyroidism. Yet, their patients with normal renal function might be considered to be in the same category.

Immunoparathyroid hormone levels were not mentioned as a factor influencing a decision for reoperation. With patients being on Prednisone and an elevated immunoparathyroid hormone level, are you not concerned about the continued loss of bone density? Perhaps it should be monitored.

Eleven percent of the patients undergoing total parathyroidectomy developed permanent hypoparathyroidism. Does cryopreservation provide a mechanism for recovery? Or should subtotal parathyroidectomy be carried out more frequently?

I commend to this audience the published article for serious thought and predict that, unlike the Magna Carta which is more frequently quoted than read, this paper will be read and widely quoted.

Thank you. [Applause]

DR. SAMUEL A. WELLS, JR. (St. Louis, Missouri): I wanted to discuss this paper also, and I thought that it contained a great deal of useful information. I was interested in the one patient that you presented that had a parathyroid adenoma and wondered whether that patient and, perhaps, other patients, as we have seen occasionally, actually had primary hyperparathyroidism, developed renal disease and a subsequent, secondary hyperparathyroidism as a complication. These patients, when operated on, often have minimally elevated parathyroid glands, minimally elevated in size, except for the one large presumed parathyroid adenoma. I would be interested in knowing your thoughts about that one case.

I, too, was concerned about the decision point of 12 mg per dL. John Potts, who is a mineral metabolism expert at the Massachusetts General Hospital chaired the committee on asymptomatic hyperparathyroidism at the NIH, and he did recommend — or the committee did — a lower point of intervention. These patients are at risk for bone disease, especially this group of patients who have preexisting bone disease. Are they placed at greater risk if you choose not to intervene surgically? I would be interested in knowing your thoughts about these two questions. [Applause]

DR. JEFFREY D. KERBY (Closing Discussion): President Wells, Secretary Copeland, Members, and Guests. It is indeed an honor for me to be invited by Dr. Diethelm to close the discussion on this paper. I would like to thank Dr. Thomas and Dr. Wells for their comments and for their questions.

Dr. Thomas asked a number of questions, the first as to a mechanism for persistent hyperparathyroidism in these patients with normal renal function. Currently, we don't have a good answer for that question. We know that 30% of patients posttransplant develop hypercalcemia. Close to 60% to 70% of those patients resolve their hypercalcemia within the first year. And it is very difficult to predict which patients will resolve and which patients will not, which patients will develop symptoms long term and which patients will not. So I don’t have a clear answer as far as a mechanism.

As far as how we derived the calcium level of 12.0 as our set point for recommending parathyroidectomy: There is some evidence that patients that have calcium levels over 12 do develop some of the metabolic complications of hypercalcemia, bone problems, pancreatitis, and some soft tissue calcification. We have followed patients with persistent borderline hypercalcemia who are asymptomatic for a number of years, and have found them not to have any of the complications. It is true that, certainly, this level is higher than some have recommended, and we should take a look at these patients that we are following long term to more closely examine this level.

As far as examining PTH levels in these patients, we have followed PTH levels in some of these patients. We used varying assays over the 29-year period of the study, and we found it very difficult to include this in our results.

There is some evidence that up to 50% of posttransplant patients have elevations of PTH without concurrent elevation of serum calcium, so we find it kind of difficult to use PTH levels as a means of determining when to suggest parathyroidectomy.

Three of the patients in our study were hyperparathyroid after their parathyroidectomy. The question was whether cryopreservation or a more liberal use of subtotal parathyroidectomy would be recommended. As Dr. Diethelm stated in the presentation, it had been our experience to put back about 30 mg to 60 mg of tissue
during our transplantation of parathyroid. It is now our recommendation to use a more liberal amount, 80 mg to 100 mg, and this seems to have presented the problems associated with hyperparathyroidism after the parathyroidectomy. We have used cryopreservation in the past. It is not currently our recommendation to do so or practice to do so.

Dr. Wells asked about the one patient with the adenoma, whether this represented primary hyperparathyroidism and not secondary. We would agree with that statement, this probably represented a sporadic case of primary hyperparathyroidism in our transplant population and was not probably related to the renal disease.

I would like to thank the Association for allowing us to present these findings and for the privilege of the floor. [Applause]