# Editorial

The overview by Tominaga from Nagoya, Japan, deals with an important issue in the management of advanced renal failure. His article is based on his great experience in this field. Tominaga is working in a department where they are responsible for both dialysis treatment and parathyroid surgery – a unique combination from our point of view. I have had the privilege to visit their unit a few times and I am impressed by their clinical work and research activity.

Secondary hyperparathyroidism (2HPT) is a common and serious complication of chronic renal insufficiency. After several years of dialysis the proportion of patients that have to undergo parathyroidectomy for 2HPT is high and increases with the duration of dialysis. In Japan, where they have a low rate of kidney transplantation, the number of dialysis patients is large which means that the number of patients undergoing surgery for 2HPT is also large – after 20 years of dialysis this proportion is about 30 percent. In the department where the author is working, almost two thousand patients have undergone parathyroidectomy for renal hyperparathyroidism during the last three decades – a highly impressive figure.

The author describes the most important factors in chronic renal failure that lead to an imbalance in the metabolism of calcium, phosphate, vitamin D and parathyroid hormone (PTH). He also emphasises the importance of preventing the development of 2HPT, as high serum levels of calcium, phosphorus, calcium-phosphorus product and PTH are associated with an increased risk of mortality (1). In his article Tominaga therefore discusses the current strategies in the medical treatment of 2HPT. He mentions new medicines that have been tried – also in their own department in Nagoya – in attempts to suppress PTH secretion by acting on the calcium-sensing receptor. However, it would have been of value if the author had discussed at somewhat greater length how the dialysate calcium concentration should be adjusted in relation to the patient´s serum calcium to achieve an optimal effect of the calcium component (2).

A long duration of renal insufficiency contributes to chronic stimulation of the parathyroid gland and development of parathyroid hyperplasia. PTH plays a central role in mineral metabolism, and high- and low-turnover bone disease seems to be attributable to high and low serum PTH (3). Kidney transplantation is the best treatment for renal HPT and in most cases it leads to normalisation of serum calcium and PTH. However, the degree of parathyroid hyperplasia is the main limiting factor for the ability of parathyroid glands to involute after kidney transplantation. Parathyroidectomy is therefore one therapeutic option for advanced HPT and in this overview the author gives his guiding principles for surgical indications. His hypothesis is that when a gland becomes heavier its pattern is transformed from diffuse to nodular hyperplasia. The parathyroid cells are thereby transformed from polyclonal to monoclonal cells and the author has also found that cells from nodules have a lower expression of calcium sensing receptors (4). On the basis of their

own results the author states that when a patient has at least one nodular gland, 2HPT may be resistant to both medical treatment and kidney transplantation.

Concerning surgical treatment, both total and subtotal parathyroidectomy are considered standard procedures for 2HPT. The author recommends total ectomy with autotransplantation of parathyroid tissue, and this is in agreement with the general opinion today. The single one randomised study in which subtotal and total parathyroidectomy have been compared also favours the more radical method (5) – in the event of recurrent disease reoperation at the autograft in the forearm is easier to carry out than re-exploration of the neck. The author also emphasises that subtotal parathyroidectomy seems to involve a certain risk of inducing parathyromatosis (a condition where multiple foci of hyperfunctioning tissue are left behind), as capsular rupture of a gland contributes to a potential risk of "spilling" cells. Tominaga pays great attention to the surgical technique and the preoperative gland detection. The article contains a number of operative and postoperative details which are of great value, not at least for surgeons working in this surgical field. The author also points out that the surgeons should be familiar with the embryology and anatomy of the parathyroid gland, and as Edward D Churchill stated as early as in 1931, "the success of parathyroid surgery must lie in the ability of the surgeon to know a parathyroid gland when he sees it, to know the distribution of the glands, where they hide, and also to be delicate enough in technique to be able to make use of this knowledge" (6).

I will conclude by saying that the overview by Tominaga is based on his great experience and the impressive results by his group in surgical treatment of 2HPT due to chronic kidney disease. The review addresses, however, not only the surgical treatment but also the current medical strategies for patients with 2HPT.

## References

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Henry Johansson, MD
Department of Surgical Sciences
University Hospital
751 85 Uppsala
Sweden