CORRESPONDENCE

Hormone Replacement After Thyroid and Parathyroid Surgery

by Prof. Dr. med. Andreas Schäffler in volume 47/2010

Additional Iodide Therapy Not Required

I have a slightly different approach regarding two issues, and my clinical practice therefore differs slightly.

- Target value for the concentration of thyroid stimulating hormone (TSH): I choose a measurement of 1–2 mU/L to compensate for autoimmune hypothyroidism. Since in patients who have had surgery one objective is to avoid repeated surgery, I believe that the TSH concentration and growth stimulus should be lower. I therefore select a low-normal target range of 0.5–1 mU/L. In patients in whom dosage adjustment proves difficult, in whom the required dosage is high, and in patients with cardiac risk I would rather accept a normal TSH value that is slightly on the high end of the acceptable range than one that is on the lower end; an actual low value is never acceptable.) After correcting the dosage I will measure the TSH concentration 6 weeks later.

- Combination with iodide: In view of the still prevailing but less severe iodine deficiency, I don't see the need for additional medication with iodide in thyroid volumes below 6 ml and substitution of more than 50% of the thyroid hormone requirement, especially as during the transformation of T4 into T3, iodide is split off and released. In larger residual volumes—for example after hemithyroidectomy—I combine a residual volume of more than 8 ml with iodide.

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Importance of Postoperative Hypocalcemia

In his review the author describes the hormone replacement therapy after thyroid surgery, as well as the therapeutic options in hypocalcemia following thyroid or parathyroid surgery. Important issues in this context are not mentioned. The different medicinal options are described in detail, but their practical implementation and the consequences for the affected patients are not

explained. Whereas permanent hypocalcemia is rare, even transient hypothyroidism occurring in 10–25% is a serious complication. Further to the demanded intra-operative exposure of all 4 parathyroid glands, also their immediate autotransplantation in the strap muscles in case of visible or expected ischemia to prevent permanent hypoparathyroidism has to be mentioned. Clinically manifest hypocalcemia puts a severe strain on patients and may affect their quality of life substantially. For this reason it is important to explain to patients with postoperative hypocalcemia—even if this is considered transient—in detail the symptoms and prognosis and provide them and the general practitioners practical guidelines. In our hospital, we determine serum calcium and parathyroid hormone before and after surgery in all thyroid and parathyroid patients. In case of postoperative hypoparathyroidism (<15 pg/mL) we substitute even in patients without symptoms 3×1,000 mg calcium/d plus 2×0.5 µg 1.25-dihydroxy-cholecalciferol/d for at least 2–3 weeks, since these patients are usually discharged after 2 days. The substitution treatment is eventually completed after examinations by the general practitioner or endocrinologist. In case of reoperations of the parathyroid glands or multiple gland disease, the facility of cryopreservation of parathyroid tissue for possible later reimplantation should be available.

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Target Range Was Missed

As an addendum to the article, which details the use of hormone replacement therapy after thyroid surgery, we wish to emphasize the importance of continuous therapeutic monitoring. As a target range for L-thyroxine substitution, a TSH serum concentration of 1–2 mU/L was recommended. Studies from the US (1) and the UK (2) give rise to the suspicion that a substantial proportion of patients receiving drug treatment for thyroid disorders are not adequately monitored in terms of their metabolic function. The results of one of our own studies from Germany (3), based on data from the population-based Study of Health in Pomerania (SHIP) showed that of 266 participants who took thyroid medication, 31% had TSH serum concentrations outside the local, study specific reference range and therefore missed the target range mentioned above.

In addition to unsatisfactory patient compliance, inappropriate dosage adjustment will result in mistreatment of thyroid disorders, which may be accompanied by adverse effects and constitutes a crucial reason for poor therapeutic results. We therefore recommend establishing therapeutic target ranges and placing more importance on monitoring drug treatment.

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In Reply:

We thank Dr Otto for his remarks, which do not contradict our own recommendations. The TSH target ranges recommended by specialty societies for hormone replacement therapy for primary hypothyroidism cited the “lower standard value for TSH,” which can obviously be interpreted in various ways. Since all sources agree with regard to the upper threshold of 2.0 mU/L, the values vary only with regard to the lower limit by source (country, type of assay) from 0.5 mU/L (1) to 0.4 mU/L (2) and to 0.3 mU/L (3). There are no

systematic data about which range is more appropriate within the lower threshold (0.5–1 mU/L or 1–2 mU/L). Rather, the factors mentioned by Dr Otto, which relate to the individual patient and are based on his or her individual situation, are important for the selected target range. In the article, 1–2 mU/L was recommended, since a primary target value of 0.5 mU/L entails a risk of subclinical hypothyroidism, which has been proved to be associated with complications including atrial fibrillation, osteoporosis, and diastolic cardiac dysfunction. If more than 6 ml of functional tissue remains this may be sufficient in some patients to maintain autochthonous euthyroidism. This residual tissue is susceptible to recurrent development of goiter, so iodine supplementation is a given in this setting (1). There is no reason to assume that, if additional L-thyroxine substitution is required (especially in patients at risk of iodine deficiency and in young patients), the residual tissue is not at all times, even for several decades, subject to recurrent goiter development. However, controlled data to refute or confirm this do not exist.

We thank Professor Goretzki and his colleagues for their correct additional points. Owing to reasons of space and the topical focus on hormone replacement therapy we were not able to discuss intraoperative surgical aspects, such as autotransplantation or cryopreservation. We cannot recommend treating every postoperative patient with hypoparathyroidism identically, with high doses of calcium and calcitriol, since hypercalcemia and overdosage of the $\text{Ca} \times \text{phosphate}$ product should be avoided and since individual patients respond to very different degrees. In such a scenario, close monitoring by the treating physician is recommended postoperatively.

We thank Hannemann, Nauck, and Wallaschofski for their comments, which we can only underline. For reasons of space we were not able to discuss the cited references any further.

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Conflict of interest statement

The authors of all contributions declare that no conflict of interest exists according to the guidelines of the International Committee of Medical Journal Editors.