

SPECIAL ARTICLE

In situ preservation of parathyroid glands: advanced surgical tips for prevention of permanent hypoparathyroidism in thyroid surgery

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Summary

Hypoparathyroidism (HPT) is one of the most frequent and severe complications of thyroid surgery. It is caused by intraoperative damage, devascularization or accidental removal of the parathyroid glands (PTGs). The incidence of postoperative HPT is directly proportional to surgery extent and surgeon's experience.

After 40 years of experience in thyroid surgery, the first author summarizes the already known surgical steps in thyroid surgery and adds some useful practical tips for in situ preservation of PTGs.

Our surgical technique focuses on meticulous capsular dissection and preservation of the middle thyroid, Kocher's vein trunk, as well as vein branches that accompany the posterior branch of the superior thyroid artery and inferior thyroid artery trunk. Ligation of all blood vessels should be as close as possible to the thyroid capsule. Identified PTGs should be de-attached from the thyroid capsule on the vascular pedicle

without significant dislocation. PTGs preservation during central neck dissection (CND) can be facilitated by using methylene blue dye for sentinel lymph nodes biopsy. PTGs are not colored in blue, unlike central lymph nodes, which facilitates central neck dissection and reduces the possibility of accidental removal of PTGs.

After several thousands of preserved PTGs using this original technique, a total prevalence of permanent HPT in the first author's series is less than 0.5%.

Following given key points and recommendations to surgical in situ preservation of PTGs, a surgeon can provide good outcome for patients after total thyroidectomy (with or without central neck dissection), regarding HPT as one of the most severe complications of thyroid surgery.

Key words: hypoparathyroidism, parathyroid, surgery, venous arterial pedicle

HPT is one of the most frequent and severe complications of thyroid surgery. It is caused by intraoperative damage, devascularization or accidental removal of the PTGs. A meta-analysis from 2014 reported highest prevalence of transient and permanent HPT of 38% and 3%, respectively, although both are probably underestimated [1]. It is known that the incidence of postoperative HPT is directly proportional to surgery extent, being highest in patients with malignancy that undergo

total thyroidectomy and CND. Giordano et al. [2] in 2012 reported incidence of 52% for transient and 16% for permanent HPT in patients with bilateral CND. Locally advanced thyroid carcinomas and reoperations carry significantly higher risk for HPT. However, the surgeon is the most important factor of prognosis, both for outcome and postoperative complications [3]. Inexperience and "rough" surgical technique can lead to damage, devascularization, or removal of unrecognized PTGs along with

thyroid gland or dissected lymph nodes. If arterial blood supply is interrupted, PTG will suffer from ischemia, while disruption of venous drainage causes stasis, resulting in PTGs infarction. Both arterial, as well as venous vascularization, are important for proper parathyroid function and prevention of HPT.

Although surgical technique has shown significant progress over decades [4], due to better understanding of anatomy and physiology of PTGs, complications like HPT still occur, representing a life-long problem that reduces significantly the quality of life for these patients. There is no synthetic substitute for parathyroid hormone, thus, preservation of adequate PTG vascular supply is crucial. This does not exclude the possibility of autotransplantation of PTGs due to vascular or oncological reasons, but reported success of autotransplantation differs [5,6].

Based on 40-year experience in thyroid surgery, the first author summarized the already known surgical steps in total thyroidectomy, with or without CND, and added some useful practical tips for *in situ* preservation of PTGs that have been used for decades in author's every-day-practice [7].

Thompson et al. in 1973 introduced capsular dissection [8] as the prerequisite for approaching the tertiary arterial branches, PTGs and recurrent laryngeal nerves. Exact surgical technique of *in situ* preservation of PTGs on arterial pedicles using magnification was described in 1975 by Attie and Khafif [9]. Later, in 1987, Schwartz and Friedman pointed out the importance of PTG capsule preservation and bloodless surgical technique, with no necessary use of magnification [10]. In 1980, Dunlop et al. showed the significance of middle thyroid Kocher's vein by venous sampling in parathyroid adenomas [11].

In 1993, the first author published preservation of the Kocher's vein trunk, as well as preservation of vein branches that accompany the posterior branch of superior thyroid artery and inferior thyroid artery trunk [7]. Back then, in the available literature, the importance of vein preservation was not apostrophized, neither is in recent publications.

After several thousands of preserved PTGs using this original technique, with autotransplantation only if necessary, the total prevalence of permanent HPT in the first author's series is less than 0.5%. Also, in the recently published study by Lee et al. [12], patients with CND, who had inferior thyroid veins preserved bilaterally, had lower incidence of hypocalcemia, as well as faster recovery of ionized calcium levels after surgery,

compared to those who were treated with less vascular-sparing technique.

To summarize, there are several key points in PTG preservation: (1) meticulous, atraumatic surgical technique; (2) bloodless surgical field, without suction; (3) capsular dissection and ligation of all blood vessels as close as possible to the thyroid capsule; (4) preservation of middle thyroid, i.e. Kocher's vein trunk, with ligation of its branches; (5) identification of PTGs, de-attachment from the thyroid capsule on vascular pedicle, without significant dislocation; (6) ligation of posterior branch of superior thyroid artery and the accompanying vein distal to branching of the superior parathyroid vascular pedicle; (7) ligation of inferior thyroid artery tertiary branches (instead of trunk ligation) and preservation of the inferior thyroid veins trunk.

At the end of operation, viability of all PTGs should be checked visually (Figure 1). Properly vascularized PTGs have shiny capsule and typical yellowish-brown color. If venous stasis is noticed, decompression with needle, scalpel or fine scissors can provide quick recovery of PTG. If ischemia is suspected, needle or scalper puncturing, with bleeding as a result, can suggest PTG is with good vascularization [7]. However, in case arterial or venous devascularization occurs, tissue should be minced and transplanted into sternocleidomastoid muscle.

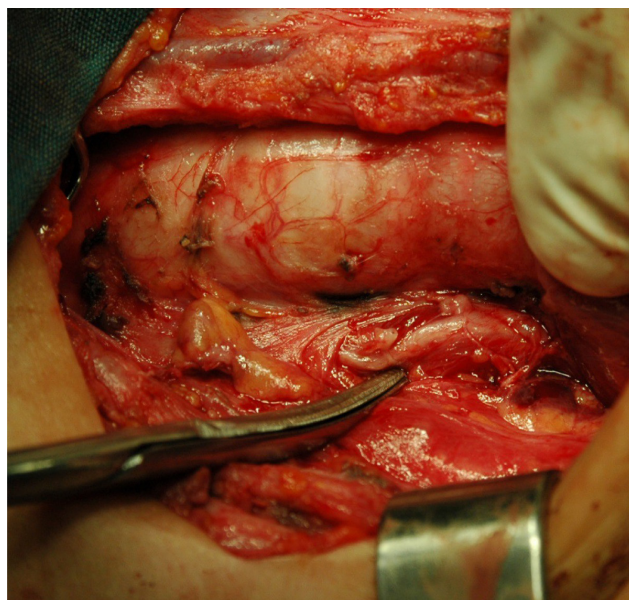


Figure 1. Intraoperative photo of *in situ* preserved parathyroid glands.

Further, the authors would like to share personal viewpoint regarding facilitating *in situ* preservation of PTGs and prevention of their acciden-

tal removal, especially in CND. For over a decade, as a standard procedure, we use methylene blue dye for mapping the sentinel lymph nodes of the jugulo-carotid chains [13-15]. PTGs are not colored in blue, unlike central lymph nodes, so distinction can be made, which facilitates CND and reduces the possibility of accidental removal of PTGs. This is especially useful for less experienced surgeons.

Nowadays, preservation of PTGs is facilitated by utilization of Harmonic scalpel, Ligasure or bipolar pincette. However, the authors recommend the least use of these to avoid thermal damage. Instead, direct ligation or fine suture should be enough, without placing hemostats that could

lead to over-traction or PTG abruption. Dissection is safer using 2.5 magnification lenses.

Following given key points and recommendations to surgical *in situ* preservation of PTGs, a surgeon can provide good outcome for patients after total thyroidectomy (with or without CND), regarding HPT as one of the most severe complications of thyroid surgery. Nevertheless, stem cells are possibly the key solution to the problem of postoperative HPT [16].

Conflict of interests

The authors declare no conflict of interests.

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