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POSTOPERATIVE COMPLICATION OF THYROIDECTOMY, AN INTERVENTIONAL STUDY WITH LITERATURE REVIEW OF PREVIOUS STUDIES

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ABSTRACT

Postoperative complication of thyroidectomy is a serious problem and should be perfectly managed to decrease the intraoperative and postoperative morbidity and mortality of thyroid surgery. This is an interventional study on 100 postoperative thyroidectomiesd patients were done to them at ALYARMOUK TEACHING HOSPITAL from the period of JANUARY 2016 TO DECEMBER 2017, with literature review of previous studies. Ten patients (10%) unilateral lobectomy with isthmuctomy were done to them, 65 patients (65%) subtotal thyroidectomy were done to them, 20 patients (20%) near total thyroidectomy were done to them and 5 patients (5%) total thyroidectomy were done to them. Ten patients (10%) have single nodule in one lobe, 5 patients (5%) have difrrentiated thyroid cancer, 70 patients (70%) have non toxic multinodular goiter, and 15 patients (15%) have toxic goiter. Sixty eight patients were female (68%) and 32 were male (32%). The female to male ratio was (2.125: 1), The age ranged from (11 to 60) years, with a mean age (30 years + 5 years), the majority being in the 4th decade of life, constituting 33 patients (33%). Sterile surgical arenas, general anesthesia, and improved surgical techniques have made morbidity and mortality from thyroid surgery extremely rare today.

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INTRODUCTION

The thyroid is an endocrine gland with an extremely rich blood supply, its isthmus lies across the 3rd, 4th and 5th rings of the trachea and the lobes lie on either side, reaching up as far as the 'pocket' under the attachment of sternothyroid to the thyroid cartilage, it is enclosed in the thin pretracheal fascia and also has its own fibrous capsule, when the gland is enlarged, the strap muscles are stretched tightly over it and the carotid sheath is displaced laterally, an important diagnostic feature is that swellings of the thyroid move on swallowing (Omar Faiz and David Moffat, 2002). The arteries to the thyroid gland are the superior thyroid artery, the inferior thyroid artery, and sometimes the thyroidea ima, the arteries anastomose profusely with one another over the surface of the gland (Richrd S.Snell, 2007). Three pairs of thyroid veins usually form a thyroid plexus of veins on the anterior surface of the thyroid gland and anterior to the trachea , the superior thyroid veins accompany the superior thyroid arteries, they drain the superior poles of the thyroid gland, the middle thyroid veins do not accompany but run essentially parallel courses with the inferior thyroid arteries, they drain the middle of the lobes, the usually independent inferior thyroid veins drain the inferior poles, the superior and middle thyroid veins drain into the Internal Jugular Veins, the inferior thyroid veins drain into the brachiocephalic veins posterior to the manubrium (Keith L. Moore, Arthur F. Dalley and Anne M. R. Agur, 2014). Lymphatic drainage of the thyroid gland is to nodes beside the trachea (paratracheal nodes) and to deep cervical nodes inferior to the omohyoid muscle along the internal jugular vein (Richard L. Drake, A.Wayne Vogl and Adam W. M. Mitchell, 2014). The nerves of the thyroid gland are derived from the superior, middle, and inferior cervical (sympathetic) ganglia, they reach the gland through the cardiac and superior and inferior thyroid peri-arterial plexuses that accompany the thyroid arteries, these fibers are vasomotor, not secretomotor, they cause constriction of blood vessels (Keith L. Moore, Arthur F. Dalley and Anne M. R. Agur, 2014). The thyroid gland arises as a median outgrowth from the floor of the pharynx near the base of the tongue, the foramen cecum of the tongue indicates the site of origin and the thyroglossal duct

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marks the path of migration of the thyroid gland to its final adult location, the thyroglossal duct usually disappears early in development, but remnants may persist as a cyst or as a connection to the foramen cecum, i.e. a fistula , there may also be functional thyroid gland, associated with the tongue (a lingual thyroid), anywhere along the path of migration of the thyroid gland, or extending upward from the gland along the path of the thyroglossal duct (a pyramidal lobe) (Richard L. Drake, A.Wayne Vogl and Adam W. M. Mitchell, 2014). The normal thyroid gland weighs (20–25 g), the functioning unit is the lobule supplied by a single arteriole and consisting of (24–40) follicles lined with cuboidal epithelium, the follicle contain colloid in which thyroglobulin is stored , the hormones tri-iodothyronine (T3) and L-thyroxine (T4) are bound to thyroglobulin within the colloid ,when hormones are required, the complex is resorbed into the cell and thyroglobulin is broken down, T3 and T4 are liberated and enter the blood, where they are bound to serum proteins, albumin, thyroxine-binding globulin (TBG) and thyroxine- binding prealbumin (TBPA), the small amount of hormone that remains free in the serum is biologically active , the metabolic effects of the thyroid hormones are due to unbound free T4 and T3 (0.03% and 0.3%) of the total circulating hormones respectively, T3 is the more important physiological hormone and is also produced in the periphery by conversion from T4. T3 is quick acting (within a few hours), whereas T4 acts more slowly (4–14 days) , the synthesis and liberation of thyroid hormones from the thyroid is controlled by thyroid-stimulating hormone (TSH) from the anterior pituitary, secretion of TSH depends upon the level of circulating thyroid hormones and is modified in a classic negative feed back manner, in hyperthyroidism, when hormone levels in the blood are high, TSH production is suppressed where as in hypothyroidism it is stimulated, regulation of TSH secretion also results from the action of thyrotrophin- releasing hormone (TRH) produced in the hypothalamus (Zygmunt H. Krukowski, 2008). The parathyroids are four endocrine glands (sometimes three or five) about the size of split peas which usually lie in two pairs behind the lateral lobes of thyroid glands, the superior parathyroids arise from the fourth branchial pouch and owing to their short migration can usually be found posterior to the upper two-thirds of the thyroid (Herald Ellis, Sir Roy Calne and Christopher Watson, 2010). The inferior parathyroids arise from the third pouch in association with the developing thymus, so may lie almost any where in the neck or superior mediastinum although the majority lie within 1cm of the lower thyroid pole, the glands secrete the (84) amino acid peptide parathyroid hormone (PTH) which controls the level of the serum calcium in extracellular fluid, PTH is released in response to a low serum calcium or high serum magnesium level, PTH activates osteoclasts to resorb bone and increase calcium reabsorption from urin and renal activation of vitamin D which subsequent increased gut absorption of calcium (Zygmunt H. Krukowski, 2013).

PATIENTS AND METHODS

This is an interventional study on 100 patients were thyroidectomy were done to them at ALYARMOUK TEACHING HOSPITAL from the period of JANUARY 2016 TO DECEMBER 2017, with literature review of previous studies. Ten patients (10%) unilateral lobectomy with isthmuctomy were done to them, 65 patients (65%) subtotal thyroidectomy were done to them, 20 patients (20%) near total thyroidectomy were done to them and 5 patients (5%) total thyroidectomy were done to them as showed in table number one. Ten patients (10%) have single nodule in one lobe, 5 patients (5%) have diffrentiated thyroid cancer, 70 patients (70%) have non toxic multinodular goiter, and 15 patients (15%) have toxic goiter as showed in table number 2.

Table 1. Types of operation

Type of operation	No of patients	%
Unilateral lobectomy with isthmuctomy	10	10%
Subtotal thyroidectomy	65	65%
Near total thyroidectomy	20	20%
Total thyroidectomy	5	5%
Total	100	100%

Table 2. Types of disease

Type of disease	No of patients	%
Single nodule in on lobe	10	10%
Diffrentiated thyroid cancer	5	5%
Non toxic multinodular goiter	70	70%
Toxic goiter	15	15%
Total	100	100%

RESULTS

Sixty eight patients were female (68%) and 32 were male (32%) as showed in table number 3.The female to male ratio was (2.125: 1), the age ranged from (11 to 60) years, with a mean age (30 years $\pm$ 5 years), the majority being in the 4th decade of life, constituting 33 patients (33%) as showed in Table 3. Table number 4 showed the postoperative complication.

Table 3. Age & Sex distribution of patients

Age group (Years)	No of female	No of male	Total	%
11 – 20	5	2	7	7%
21 -30	20	7	27	27%
31 - 40	25	8	33	33%
41 – 50	10	13	23	23%
51- 60	8	2	10	10%
Total	68	32	100	100%

Table 4. Postoperative complication

Postoperative complication	No of patients	%
Subcutaneous seroma	4	4%
Deep bleeding	3	3%
Recurrent laryngeal nerve injury	1	1%
Superior laryngeal nerve injury	3	3%
Superficial cellulitis	5	5%
Pus collection	2	2%
Hypoparathyroidism	10	10%
Hypothyroidism	25	25%
Hypertrophic scar	7	7%
TOTAL	60	60%

DISCUSSION

During the 1800s, the mortality rate from thyroid surgery was approximately 40%. Most deaths were caused by infection and hemorrhage. Sterile surgical arenas, general anesthesia, and improved surgical techniques have made death from thyroid surgery extremely rare today. Theodor Kocher, Theodor Billroth, and William S. Halsted are just a few of the names intimately associated with the development and refinement of

thyroid surgery. Their contributions helped to make thyroid surgery less feared and better understood than it once was. Although the complication rate of thyroid surgery has certainly decreased, surgeons must nevertheless maintain a healthy respect for the possibility of complications. Patients must be appropriately and preoperatively counseled regarding potential complications. All must be well aware of the surgical risks they are undertaking. By developing a thorough understanding of the anatomy and of the ways to prevent each complication, the surgeon can minimize each patient's risk. The surgeon's experience is a significant contributor to various complications during thyroid surgery. At the same time, several reports have pointed out the safety of thyroid procedures performed at residency-training centers, where surgeries are performed under the supervision of an experienced surgeon. Meltzer *et al* developed a model to predict the risk of 30-day complications from thyroid surgery, with the presence of thyroid cancer being the most significant variable. Among patients with thyroid cancer, the presence of coronary artery disease and the use of central neck dissection also predict increased risk, according to the model, while in patients without thyroid cancer, complications are predicted by coronary artery disease, dyspnea, complete thyroidectomy, and lobe size (Meltzer C, Klau M, Gurushanthaiah D, *et al* 2016). A study indicated that economic and social factors influence the outcomes of thyroidectomies in the United States, the study, which involved data from 14,220 in patients and 7215 out patients thyroidectomies, found that persons from high–health-risk communities undergoing thyroidectomy were more likely to be women and African Americans and that they had a higher likelihood of being operated on by low-volume surgeons. They also had a greater risk of suffering postoperative complications and of being readmitted to the hospital and also tended to have longer hospital stays (Al-Qurayshi Z, Randolph GW, Srivastav S, *et al*, 2016). Nonetheless, a retrospective study by Ayala and Yencha indicated that hospitals with a low surgical volume can safely perform out patients thyroid surgery.

The study, involving 160 patients who underwent either total thyroidectomy (61 patients) or hemithyroidectomy (99 patients) at a low-surgical volume facility, found that the hospital discharged 109 patients on the day of surgery, while 43 patients were admitted to the hospital for 23 hour observation and eight patients were admitted for longer than 24 hours. Among the surgical complications encountered were temporary recurrent laryngeal nerve (RLN) injury (four patients), permanent RLN injury (one patient), temporary bilateral RLN injury (one patient), delayed hematoma (two patients), and transient hypocalcemia (eight patients) (Ayala MA, Yencha MW, 2015). By understanding the presentation and treatment of each complication, the surgeon can handle complications expediently and avoid worsening consequences. The complication of our study were managed perfectly and death rate was zero. The bleeding cases were treated by re exploration with ligation of the slipped vessel individually (in one case inferior thyroid artery, the other superior thyroid artery and the last case is middle thyroid vein). The case of injured one side of RLN, tracheostomy was done to the patient and after week removed. The cases of SLN injuries were treated conservatively. Hypothyrodism cases treated by long life thyroxin tablet. Hypoparathyrodism cases all are temporarily and treated conservatively. The superficial infection treated by antibiotics and the cases with pus collection was drained and send for culture and sensitivity followed by proper antibiotics treatment. The cases with hypertrophic scar treated conservatively and no one need surgery. In literature review of previous studies divided the complications of thyroid surgery into minor, rare, and major complication.

Minor complications

Postoperative surgical site seromas may occur. These may be followed clinically and allowed to resorb if small and asymptomatic. Large seromas may be aspirated under sterile conditions, and repeated aspirations may be necessary. Poor scar formation is another frequently preventable complication. Ideally, the surgeon will design as small an incision as reasonable in a natural skin crease over the thyroid gland. The neck should be flexed to determine the location of the natural skin creases. This incision should provide adequate exposure and yet minimize the resultant scar. The surgeon should avoid excessive retraction which may damage the skin edges. If significant damage is present at the end of the surgery, the skin edges can be resected prior to closure.

Rare complications: such as damage to the sympathetic trunk, are occasionally reported. Most of these are uncommon and can usually be avoided if the surgeon has good knowledge of the anatomy and sound operative technique.

Major complications

Bleeding: Intraoperative bleeding stains the tissues and obscures important structures. Moreover, intraoperative bleeding increases the risk of other anatomic complications. Deliberate dissection and fastidious hemostasis are essential to prevent this complication, postoperative bleeding can be a devastating complication of thyroid surgery. An unrecognized or rapidly expanding hematoma can cause airway compromise and asphyxiation. The incidence of hemorrhage after thyroid surgery is low (0.3-1%), but the surgeon must be aware of this potentially fatal complication, patients with postoperative bleeding present with neck swelling, neck pain, and/or signs and symptoms of airway obstruction (eg, dyspnea, stridor, hypoxia). Immediately examine such patients for evidence of hematoma. Imaging studies are of no benefit in the initial stage of evaluation. Physical examination is the critical step in evaluation. Not waste time with imaging studies when active bleeding is possible. All bandaging should be removed and the neck examined for possible swelling indicating a wound hematoma. Imaging studies such as CT scanning and ultrasonography may be useful in cases of mild neck swelling without airway compromise. The surgeon must carefully assess the airway before transferring a patient for radiologic studies. Fiberoptic laryngoscopy may be warranted in patients with airway issues without apparent wound hematoma to assess vocal fold function. Sound surgical technique is essential. The thyroid is a highly vascular organ and bleeds copiously. Therefore, care must be taken to avoid traumatizing the thyroid tissue during the procedure. The prevention of postoperative bleeding depends on good intraoperative hemostasis. Hemostasis in thyroid surgery is achieved by means of clamp and tie, surgical clips, diathermy, ultrasonic coagulating-dissection such as a harmonic scalpel (HS) or electrothermal bipolar vessel sealing systems (EBVSS). Before closing the wound, irrigate it well and address all residual bleeding. Finally, avoid the use of neck dressings. A dressing that covers the wound may mask hematoma formation, delaying its recognition. Controversy still surrounds the use of

drains after thyroid surgery either (active or passive). Results of several prospective studies have disputed the usefulness of drains. Randomly assigned 200 patients undergoing thyroidectomy into 2 groups, One group received a suction drain at the time of surgery, whereas the other did not, hematomas occurred in (5%) and (7%), respectively. Two (2%) patients without drains and 1 (1%) with a drain required exploration for vascular ligation. Neither of these differences was statistically significant. In study reported that the postoperative hospitalization time was longer in the patients with drains (3.4 vs 1.6 d), as was the duration of postoperative pain (Schoretsanitis G, Melissas J, Sanidas E, 1998). Prospectively evaluated the usefulness of drains after thyroidectomy in a randomized trial for 100 patients (Debry C, Renou G, Fingerhut A, 1999). Four patients, all of whom received drains, developed hematomas. No definitive evidence suggests that drains prevent hematoma or seroma formation. If a drain is placed, its use should not substitute for intraoperative hemostasis. Non suction drainage is not recommended because it increases the infection risk and the need for neck dressings. These dressings obstruct the view of the neck and may delay diagnosis of a hematoma.

Treatment: If a neck hematoma is compromising the patient's airway, open the surgical incision at the bedside to release the collection of blood, and immediately transfer the patient to the operating room. In the case of a hematoma without impending airway obstruction, transfer the patient to the operating room as soon as is practical. Remain with the patient and be prepared to assist with airway management. In the operating room, open the surgical incision, explore the wound, irrigate it, control all bleeding sites, and close the wound.

Injury to the Recurrent Laryngeal Nerve: The recurrent laryngeal nerve (RLN) innervates all of the intrinsic muscles of the larynx with the exception of the cricothyroid muscle, which is innervated by the superior laryngeal nerve (SLN). Mechanisms of injury to the RLN include complete or partial transection, traction, contusion, crush, burn, misplaced ligature, and compromised blood supply. The consequence of an RLN injury is true vocal-fold paresis or paralysis. Patients with unilateral vocal fold paralysis present with postoperative hoarseness or breathiness. The presentation is often subacute. At first, the vocal fold usually remains in the paramedian position, creating a fairly normal voice. Definite vocal changes may not manifest for days to weeks. The paralyzed vocal fold atrophies, causing the voice to worsen. Other potential sequelae of unilateral vocal-fold paralysis are dysphagia and aspiration. Bilateral vocal-fold paralysis may occur after total thyroidectomy, and it usually manifests immediately after extubation. Both vocal folds remain in the paramedian position, causing partial airway obstruction. Patients with bilateral vocal-fold paralysis may present with biphasic stridor, respiratory distress, or both. On occasion, the airway is sufficient in the immediate postoperative period despite the paralyzed vocal folds. At follow-up, such patients may present with dyspnea or stridor with exertion.

Evaluation: Techniques for assessing vocal fold mobility include indirect and fiberoptic laryngoscopy. Documentation of vocal fold mobility should be a routine part of the preoperative physical examination of any patient presenting with a thyroid mass. Postoperative visualization should also be performed, as these patients may be asymptomatic, especially at first. Laryngeal electromyography (EMG) may be useful to distinguish vocal fold paralysis from injury to the cricoarytenoid joint secondary to intubation. Furthermore, EMG may yield information concerning prognosis of the patient with RLN injury. Study performed laryngeal EMG in 24 patients with vocal fold paralysis due to numerous etiologies (eg, idiopathic causes, surgery, tumor, trauma, neurologic diseases) (Parnes SM, Satya-Murti S, 1985). No patient in whom EMG revealed an absence of motor unit potentials or fibrillation potentials regained movement of the true vocal fold. Of 14 patients who had normal or polyphasic action potentials, 11 regained function. However, most of the tests were performed more than 6 months after the onset of paralysis; therefore, this study revealed little regarding the usefulness of early EMG testing. The patient with bilateral paralysis of the true vocal folds who presents with airway obstruction after extubation likely requires emergency re intubation or tracheotomy. Fiberoptic laryngoscopy may be performed to confirm the diagnosis if the patient is clinically stable.

Prevention: Deliberate identification of the RLN minimizes the risk of injury. When the nerve is identified and dissected, the reported RLN injury rate during thyroidectomy is (0.2-1%). This rate is reportedly higher if surgery is repeated (2-12%) or if the nerve is not clearly identified (4.6-6%). Intraoperative hemostasis and a thorough understanding of the anatomy are essential for identifying and preserving the nerve. The course of the RLN differs on the right and left sides of the neck. The left RLN branches from the vagus at the level of the aortic arch. It then passes below the arch and reverses its course to continue superiorly, posterior to the aortic arch and into the visceral compartment of the neck. It travels near or in the tracheoesophageal groove until it enters the larynx just behind the cricothyroid articulation. The right RLN branches from the vagus more superiorly than does the left, at the level of the subclavian artery. It loops behind the right subclavian artery and ascends superomedially toward the tracheoesophageal groove. It then continues superiorly until entering the larynx behind the cricothyroid articulation. Classic descriptions of the RLNs hold that they ascend in the tracheoesophageal groove, however, they may in fact be lateral to it. Low in the neck, the course of the right RLN is relatively oblique and lateral and, probably, more prone to injury than the left RLN. The nerve may branch several times before entering the larynx. Take care to identify and preserve each branch. In approximately 5 of 1000 patients, a non recurrent laryngeal nerve is found on the right side. This arrangement occurs when a retroesophageal right subclavian artery arises from the dorsal side of the aortic arch. The non recurrent laryngeal nerve branches from the vagus at approximately the level of the cricoid cartilage and directly enters the larynx without looping around the subclavian artery. A left-sided non recurrent laryngeal nerve RLN can occur only when a right-sided aortic arch and ligamentum arteriosum are concurrent with a left retroesophageal subclavian artery. The inferior thyroid artery has been described as an important landmark for identifying the RLN. However, its relationship to the nerve is subject to variation . Numerous descriptions and attempts to quantify the percentages of each relationship of the nerve to the artery have been put forth. Percentages differ on the right and left sides. On the right, the nerve runs between branches of the artery in approximately (50%) of patients. The nerve is anterior to the artery in (25%) and posterior in (25%). On the left, the nerve courses posteriorly to the artery in (50%) of patients; in approximately (35%), the nerve runs between

branches. In only (15%) is it anterior to the artery. In summary, the nerve is always near the artery, but the exact relationship cannot be determined with certainty. Therefore, the inferior thyroid artery is not a dependable landmark for identifying the nerve. Several approaches are used to identify and preserve the RLN. The authors' preferred approach involves finding the nerve at its point of laryngeal entry, which is approximately (0.5 cm) below the inferior cornu of the thyroid cartilage. Thyroid tissue in the region of the ligament of Berry is meticulously dissected from the trachea by carefully ligating traversing vessels. This technique exposes the RLN as it enters the larynx. It also minimizes the compromise of blood supply to the parathyroids and limits the extent of dissection involving the nerve. Thick connective tissue called the Berry ligament attaches the thyroid to the trachea at the level of the second or third tracheal ring. This is the most common site of injury to the RLN The nerve may run deep to the ligament, pass through it, or even penetrate the gland a short distance at this level. Be extremely careful in this area during surgery. Retraction of the thyroid lobe may result in traction injury and make the nerve susceptible to transection. The path of the nerve must be clearly identified. Continuous electrophysiologic monitoring of the RLN during thyroid surgery is easily performed.

Two EMG devices include an endotracheal- tube electrode (Xomed-Treace, Jacksonville, FL) and an RLN- postcricoid-laryngeal surface electrode. Both provide useful EMG information and help to reveal the location of the RLN. Use of EMG is controversial and has not been recommended for routine thyroid surgery given the low rate of RLN injury. The authors know of no randomized studies that have been performed to compare the rate of postoperative RLN palsy in visual versus electrophysiologic RLN detection in thyroid surgery. A literature review indicated that adjunctive use of intraoperative electrophysiologic neuromonitoring may be no more effective than visual nerve identification alone for avoiding RLN injury during thyroid surgery, no significant difference in the incidence of RLN injuries was found in a comparison of both monitoring methods (Deniwar A, Bhatia P, Kandil E, 2015). The additional information EMG provides may be beneficial in patients undergoing revision thyroid surgery, in patients with previously radiated necks, in patients with large masses, or in patients with contralateral nerve palsy. In the setting of unilateral vocal- fold paralysis, management of the contralateral thyroid is controversial. In the authors' experience, given the low rate of RLN injury, the best approach is not changing appropriate oncologic management and proceeding with total thyroidectomy. Exceptions may include a young patient who presents with a low (< 6) multifactor activated immune cell (MAIC) score. For such a patient, non operative treatment may be considered, with appropriate care coordinated with an endocrinologist. Treatment: Not perform corrective procedures for unilateral vocal- fold paralysis until at least (6) months after thyroidectomy because a reversible injury improves by that time. If the nerve was definitely transected during surgery, treatment for the paralyzed fold may be performed sooner than this. Two surgical treatment options are available for patients with unilateral vocal- fold paralysis, medialization and reinnervation. Medialization is most commonly performed. However, the authors' knowledge, no investigator has compared the efficacy of these 2 procedures. Medialization of the impaired vocal fold improves contact with the contralateral mobile fold. It may be accomplished with injection laryngoplasty or laryngeal framework surgery. Type I thyroplasty is probably the most common procedure. A window in the thyroid cartilage is created at the level of the true vocal fold. An implant is then placed to push the vocal fold medially. Medialization with an injection of absorbable gelatin sponge (Gelfoam; Pharmacia & Upjohn Company, Kalamazoo, MI) may be performed before (6) months if the patient desires it or if he or she has is aspirating. The gelatin sponge resorbs over time and is, therefore, a temporary treatment. An implant made of silicone or polytetrafluoroethylene (PTFE, Gore-Tex; W. L. Gore & Associates, Inc; Newark, DE) is considered permanent. However, most authorities agree that no negative consequences occur if nerve recovers function after a type I thyroplasty. In addition, the implant may be removed, though this requires another surgical procedure. A number of reinnervation procedures have been described for addressing the permanently injured RLN. These procedures maintain or restore tone to the intrinsic laryngeal musculature. When the true vocal fold atrophies after denervation, it loses contact with the contralateral fold and the voice weakens. By preventing atrophy, reinnervation procedures may help maintain or improve the patient's voice. Primary neurorrhaphy may be used to immediately repair the transected RLN. This procedure typically results in synkinesis because of nonselective reinnervation of abductor and adductor muscles.

Reinnervation procedures have been described by using the phrenic nerve, ansa cervicalis, and preganglionic sympathetic neurons. Although animal models demonstrated EMG and histologic evidence of reinnervation, as well as restored movement of the vocal fold, experience in humans has not been as impressive as this. Improvement in phonation quality has been documented in humans after reinnervation with the ansa cervicalis, but no movement is observed. Transfer of neuromuscular pedicles have been described and reportedly restore movement of the vocal fold. However, these reports are limited, and success is not universal. In bilateral vocal- cord paralysis, initial treatment involves obtaining an adequate airway. Emergency tracheotomy may be required. If possible, first perform endotracheal intubation. Consider exploring the neck to ensure that no reversible causes of nerve injury (eg, misplaced ligature) are present. When good preservation of the RLNs is ascertained, a trial of extubation may be performed after several days. Intravenous steroids may be beneficial in this situation. Remove the tube over a Cook catheter and in a controlled setting in case reintubation is necessary. Be ready to perform emergent tracheotomy. If nerve function has not recovered after a second trial of extubation, tracheostomy is certainly warranted. The principal goal for surgery in bilateral vocal-fold paralysis is to improve airway patency. Cordotomy and arytenoidectomy are the most common procedures. These procedures enlarge the airway and may permit decannulation of a tracheostomy. However, the patient must be counseled that his or her voice will likely worsen after surgery. Transfer of a neuromuscular pedicle is reported to improve the airway in cases of bilateral true vocal- fold paralysis. However, again, these reports are limited, and this treatment is not a widely accepted.

Hypoparathyroidism; Hypoparathyroidism is another feared complication of thyroid surgery. Inadequate production of PTH leads to hypocalcemia. Hypoparathyroidism, and the resulting hypocalcemia, may be permanent or transient. The rate of permanent hypoparathyroidism is (0.4-13.8%). The

condition may be due to direct trauma to the parathyroid glands, devascularization of the glands, or removal of the glands during surgery. The rate of temporary hypocalcemia is reportedly (2-53%). The cause of transient hypocalcemia after surgery is not clearly understood. It may be attributable to temporary hypoparathyroidism caused by reversible ischemia to the parathyroid glands, hypothermia to the glands, or release of Endothelin-1. Endothelin-1 is an acute-phase reactant known to suppress PTH production, and levels have been elevated in patients with transient hypoparathyroidism. Other hypotheses have been put forth to account for transient hypocalcemia not caused by hypoparathyroidism. These include calcitonin release and hungry-bone syndrome. Calcitonin is produced by the thyroid and inhibits bone breakdown while stimulating renal excretion of calcium. Its effects on calcium metabolism oppose those of PTH. Hungry-bone syndrome occurs in patients with preoperative hyperthyroidism. These patients have increased bone breakdown in their hyperthyroid state. When a patient's thyroid hormone level drops acutely after surgery, his or her stimulus to break down bone is removed. The bones, now "hungry" for calcium, remove calcium from the plasma, decreasing serum calcium levels. Risk factors for hypocalcemia after thyroidectomy include Graves disease and malignancy. The type of procedure performed (total thyroidectomy, thyroidectomy with neck dissection, repeat thyroidectomy, subtotal thyroidectomy, near- total thyroidectomy) also affects the risk in transient hypocalcemia after surgery. As more of the parathyroid gland inadvertently removed, the risk of hypocalcemia rises.

Presentation: Most patients who are hypocalcemic after thyroidectomy are initially asymptomatic. Symptoms and signs of hypocalcemia include circumoral paresthesias, mental status changes, tetany, carpopedal spasm, laryngospasm, seizures, QT prolongation on ECG, and cardiac arrest. Patients having undergone a procedure where all parathyroid glands have been placed at risk for injury (total thyroidectomy, subtotal thyroidectomy, or completion thyroidectomy) should undergo evaluation for iatrogenic hypoparathyroidism. An effective method of evaluation of parathyroid function is to follow ionized calcium (or total calcium and albumin) levels in the perioperative period. If iatrogenic hypoparathyroidism is a concern, close follow-up care is warranted until calcium levels demonstrate that parathyroid function is intact. Alternatively, a normal postoperative PTH level can accurately predict normocalcemia after thyroid surgery. Identification of at risk patients with low PTH levels will facilitate prompt calcium replacement therapy and safe early discharge from hospital. Wong, *et al* found that the combination of immediate postoperative PTH levels (< 1.5 pmol/l) and morning serum calcium (< 2.0 mmol/l) would accurately identify patients at risk of hypocalcemia following total thyroidectomy (Wong C, Price S, Scott-Coombes D, 2006). Hypocalcemia may also be indicated by presence of the Chvostek sign or the Trousseau sign. The Chvostek sign is elicited by tapping the region of the facial nerve in the preauricular area resulting in facial contractions. The Trousseau sign is carpal spasm that may be elicited by inflation of a blood pressure cuff on the upper arm. In the setting of hypocalcemia, other causes (renal failure, hypomagnesemia, medications) may be considered. Serum phosphorous levels are elevated in patients with hypoparathyroidism secondary to decreased renal excretion, this difference may help in distinguishing low PTH levels due to other etiologies of hypocalcemia (eg, hungry bone syndrome). Regardless of the etiology, the management is unchanged. If uncertainty remains 6 months after surgery, PTH levels may be checked evaluate recovery of parathyroid gland function. The best way to preserve parathyroid gland function is to identify the glands and to maintain their blood supply. A large cadaveric study to identify the most common positions of the parathyroid glands demonstrated that (77%) of superior parathyroid glands were at the cricothyroid junction and intimately associated with the RLN. About (22%) of the superior parathyroid glands were on the posterior surface of the upper lobe of the thyroid. Approximately (1%) of the superior glands were behind the junction of the hypopharynx and upper esophagus. The study demonstrated that the location of the inferior parathyroid glands was variable. Forty-two percent were on the anterior or lateral surfaces of the lower lobe of the thyroid, often hidden by vessels or creases in the thyroid. Thirty-nine percent were located within the superior tongue of the thymus. Fifteen percent were extrathyroidal and lateral to the lower lobe. Two percent were in the mediastinal thymus, and another (2%) were in other ectopic positions, such as the carotid sheath. The ectopic inferior parathyroid glands were consistently associated with remnant thymus tissue. The inferior parathyroid glands and the thymus both develop from the third branchial pouch, a finding that explains the close association of these structures. The inferior parathyroid glands receive their blood supply from the inferior thyroid artery. The superior parathyroids also usually receive their blood supply from the inferior thyroid artery. However, in some cases, the superior parathyroids receive their vascular supply from the superior thyroid artery, the anastomotic loop between the inferior and superior thyroid arteries, or direct branches off the thyroid gland. The key to parathyroid preservation is identifying the parathyroids and preserving their blood supply by ligating all vessels distal to them. Ligate the vessels as close to the thyroid gland as possible. Recognition of the parathyroid glands, which appear in various shapes and which have a caramel-like color, is critical. When they lose their blood supply, they often darken in appearance. The surgical wound and the excised thyroid gland should be carefully examined for parathyroid tissue. The devascularized gland (pathologically confirmed with frozen-section analysis), should be removed, cut into (1 to 2 mm) pieces, and reimplanted into a pocket created in the sternocleidomastoid muscle or strap muscle. The location may be marked with a permanent suture or a metallic hemoclip for easier identification in any future surgeries.

Treatment: Patients who have asymptomatic hypocalcemia in the early postoperative period should not be treated with supplemental calcium. The hypocalcemic state may stimulate the stunned parathyroid glands to produce PTH. Patients who have symptomatic hypocalcemia in the early postoperative period or whose calcium levels continue to fall rapidly require treatment. In symptomatic patients, replace calcium with intravenous calcium gluconate. Ten milliliters of (10%) solution (1 g) may be administered over (10) minutes. A calcium infusion may be started at a rate of 1-2 mg/kg/h if symptoms do not resolve. Titrate the infusion to the patient's symptoms and calcium levels. Typically, patients that begin to have symptoms can be started on oral calcium. One to two grams of elemental oral calcium should be supplied each day. Calcium carbonate (1250 mg) provides (500 mg) of elemental calcium, therefore, the patient should take (2500-5000 mg/day). The patient needs concomitant replacement of vitamin D with calcitriol (Rocaltrol) (0.25-1 mcg/d). The calcium supplementation should be divided (4 – 5) times per

day rather than in a single dose to maximize absorption by the GI tract. Endocrinology consultation may provide assistance in monitoring of calcium levels and the medical management of the sequelae of hypoparathyroidism. In (1-2) months, an attempt to wean the patient off oral calcium may be made to reveal if the hypoparathyroidism is temporary. Dependence on calcium supplementation for longer than (6) months usually indicates permanent hypoparathyroidism.

Thyrotoxic Storm: Thyrotoxic storm is an unusual complication of thyroid surgery. This condition may result from manipulation of the thyroid gland during surgery in the patients with hyperthyroidism. It can develop preoperatively, intraoperatively, or postoperatively. Surgery is generally recommended only when patients have Graves disease and other treatment strategies fail or when underlying thyroid cancer is suspected. Thyrotoxic storm is potentially lethal and must be dealt with astutely. Signs of thyrotoxic storm in the anesthetized patient include evidence of increased sympathetic output, such as tachycardia and hyperthermia. Other symptoms and signs in the awake patient include nausea, tremor, and altered mental status. Cardiac arrhythmias may also occur. If treatment is not given, the patient may progress to coma. Preoperative awareness of the hyperthyroid patient and appropriate medical treatment are the keys preventing thyrotoxic storm. Patients undergoing thyroidectomy for persistent thyrotoxicosis require treatment based on the time available and the severity of symptoms. The goal is to restore a state as close to euthyroid as possible before surgery. Medical management is directed at targets of the thyroid hormone synthetic, secretory, and peripheral pathways. These include thioamides (methimazole, propylthiouracil [PTU]), which affect synthesis. PTU also inhibits peripheral deiodination (thyroxine [T4] to triiodothyronine [T3]). Iodine used at supraphysiologic doses decreases synthesis of new thyroid hormone (the Wolff-Chaikov effect), and it has an onset of action within (24 hours) and a maximum effect at (10 days). Beta-blockers should be given to every thyrotoxic patient unless contraindicated (eg, congestive heart failure [CHF]). High doses of glucocorticosteroids impair peripheral conversion of T4 to T3 and are used for when thyrotoxicosis is severe and when rapid management is necessary. Morbidity and mortality rates in adequately prepared patient are low.

Intraoperative management: The first step in managing a thyrotoxic crisis during thyroidectomy is to stop the procedure. Intravenous beta-blockers, PTU, sodium iodine, and steroids are administered to control sympathetic activity, the release of thyroid hormone, and hyperthermia. Use cooling blankets and cooled intravenous fluids to reduce the patient's body temperature. Carefully monitor oxygenation, because oxygen demands increase dramatically during a thyroid storm.

Postoperative management: Removal of the thyroid gland does not immediately relieve thyrotoxicosis because the half-life of circulating T4 is **(7-8 days)**. As thyroid hormone levels decrease and as symptoms resolve, medications should be gradually weaned over the weeks after surgery. An endocrinologist should be consulted to assist in this process.

Injury to the Superior Laryngeal Nerve: The SLN has (2) divisions: internal and external. The internal branch provides sensory innervation to the larynx. It enters the larynx through the thyrohyoid membrane and, therefore, should not be at risk during thyroidectomy. The external branch provides motor function to the cricothyroid muscle and is at risk during thyroidectomy. This muscle is involved in elongation of the vocal folds. Trauma to the nerve results in an inability to lengthen a vocal fold and, thus, an inability to create a high-pitched sound. The external branch of the SLN is probably the nerve most commonly injured in thyroid surgery. The rate of injury to the external branch of the SLN is estimated at (0-25%). This rate is probably underestimated, because the diagnosis is frequently missed. The clinical presentation of a patient with SLN paralysis may be subtle. Most patients do not notice any change. On occasion, a patient presents with mild hoarseness or decreased vocal stamina. However, for the singer or person who professionally relies on his or her voice, paralysis of the SLN may threaten his or her career.

The most damaging consequence is loss of the upper register. Diagnosing an SLN injury with indirect or fiberoptic laryngoscopy is difficult. Posterior glottic rotation toward the paretic side and bowing of the vocal fold on the weak side may be noted. In addition, the affected vocal fold may be lower than the normal vocal fold. Use of videostroboscopy and laryngeal EMG has enhanced the ability of otolaryngologists and speech pathologists to diagnose SLN paralysis. Videostroboscopy demonstrates an asymmetric, mucosal traveling wave. EMG demonstrates cricothyroid muscle denervation. The external SLN branch travels inferiorly along the lateral surface of the inferior constrictor until it terminates at the cricothyroid muscle. This branch is intimately related to the superior thyroid artery, though its exact relation to the artery varies. Data from a recent cadaveric study suggested that the nerve may cross the superior thyroid artery more than (1 cm) above the upper pole of the thyroid gland (42%), less than (1 cm) above the upper pole (30%), or under the upper pole (14%). In some people, the nerve runs dorsal to the artery and crosses only its terminal branches after the artery has ramified (14%). A critical area (1.5-2 cm) from the thyroid capsule is described. In this area, the external branch of the SLN is most intimately involved with the branches of the superior thyroid artery. Most surgeons agree that identifying the SLN, in contrast to the RLN, is unnecessary. Instead, ligate the terminal branches of the superior thyroid artery as close to the thyroid capsule as possible to avoid damaging the nerve. Electrophysiologic monitoring of the SLN is described, but it is not recommended for routine use. Direct trauma to the cricothyroid muscle can cause fibrosis and poor muscle function, which may result in a presentation similar to that of a patient with an injury to the external branch of the SLN, even when the nerve is preserved. Therefore, dissect carefully near this muscle and avoid electrocautery damage when possible. At present, the only treatment available for injury to the external branch of the SLN is speech therapy.

Infection: Infection was the major cause of death from thyroid surgery during the (1800s). Today, infection occurs in less than (1-2%) of all cases. Death is unlikely if the infection is recognized and treated promptly and appropriately. Postthyroidectomy infection may manifest as superficial cellulitis or as an abscess. Patients with cellulitis typically present with erythema, warmth, and tenderness of neck skin around the incision. A superficial abscess may be diagnosed on the basis of fluctuance and tenderness. A deep neck abscess may manifest subtly, but signs such as fever, pain, leukocytosis, and tachycardia should raise clinical suspicion. Send purulence expressed from the wound or drained from an abscess for Gram staining and culturing to direct the choice of

antibiotics. CT imaging is useful when a deep neck abscess is thought possible. In addition, a deep neck abscess should raise concern about possible esophageal perforation. An esophageal swallow study performed with sodium amidotrizoate and meglumine amidotrizoate solution (Gastrografin) may be useful in certain cases. The key to preventing postoperative infection is the use of sterile surgical technique. Routine use of perioperative antibiotics in thyroid surgery has not been proven beneficial. Johnson and Wagner (1987) retrospectively reviewed (438) patients who underwent uncontaminated head and neck surgery at the Eye and Ear Hospital of Pittsburgh. Of (113) patients who received thyroidectomy, only (12) were given antibiotics perioperatively. None of the thyroidectomy-treated patients had a postoperative wound infection. This result suggests that perioperative antibiotics are not useful. Antibiotics should not be used unnecessarily in the current era of multidrug- resistant bacteria. Perioperative antibiotics are not recommended for thyroid surgery. Treat cellulitis with antibiotics that provide good coverage against gram-positive organisms (eg, against staphylococci and streptococci). Drain abscesses, and direct antibiotic coverage according to culture findings. If patients have deep neck abscesses, begin with broad-spectrum antibiotics (eg, cefuroxime, clindamycin, ampicillin-sulbactam) until definitive culture results are available.

Hypothyroidism: Untreated hypothyroidism causes symptoms such as cold intolerance, fatigue, constipation, muscle cramping, and weight gain. Hypothyroidism secondary to thyroid surgery should never be left untreated long enough to elicit signs and symptoms of myxedema (eg, hair loss, large tongue, cardiomegaly). Expect, diagnose, and promptly treat postoperative hypothyroidism. The most useful laboratory test for detecting or monitoring of hypothyroidism in the patient who has undergone thyroidectomy is the measurement of thyrotropin (thyroid-stimulating hormone [TSH]) levels. Total T4 and T3 levels may be useful to fine- tune the dosing of levothyroxine (Synthroid), but may be unhelpful in the typical postoperative patient. Hypothyroidism is an expected sequela of total thyroidectomy. In goiter surgery, the extent of thyroidectomy is controversial. The main goal of surgery is to prevent recurrent hyperthyroidism because recurrent hyperthyroidism after surgery is more difficult than permanent hypothyroidism to manage. Because of this fact, the present authors recommend total thyroidectomy in this setting. For hypothyroid patients, start levothyroxine (about 1.7 mcg/kg/d). Check their thyrotropin level in approximately (4-6 weeks), and adjust the dosage appropriately. Patients who are to receive postoperative radioiodine scanning must stop taking levothyroxine before the procedure. Consider assistance from an endocrinologist during this period to ensure appropriate monitoring (eg, of renal insufficiency due to hypothyroidism).

Conclusion and Recommendation

Sterile surgical arenas, general anesthesia, and improved surgical techniques have made morbidity and mortality from thyroid surgery extremely rare today. In June 2013, the American Academy of Otolaryngology—Head and Neck Surgery Foundation issued new guidelines designed to highlight the importance of a patient's voice and to improve voice outcomes in patients receiving thyroid surgery. The guidelines include:

- Strong recommendation that surgeons performing thyroid surgery identify the recurrent laryngeal nerve or nerves,
- Assess the patient's voice prior to surgery,
- Examine vocal fold mobility if the patient's voice is impaired,
- Examine vocal fold mobility under defined circumstances if the patient's voice is normal,
- Educate the patient about the possible effect of thyroid surgery on the voice,
- Inform the anesthesiologist of any abnormal preoperative laryngeal assessment,
- Preserve the external branch of the superior laryngeal nerve during surgery,
- Document any voice change after surgery,
- Examine vocal fold mobility in patients with voice change,
- Refer patients with abnormal vocal fold mobility to an otolaryngologist,
- Counsel patients on voice rehabilitation options (Pullen L, 2013), (Chandrasekhar SS, Randolph GW, Seidman MD, *et al*, 2013).

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