A STUDY OF POST OPERATIVE COMPLICATIONS AFTER THYROID SURGERY

Dissertation Submitted

for M.S. Degree in General Surgery

Branch I

The Tamil Nadu

Dr. M.G.R. Medical University

Chennai

September **2006.**

CERTIFICATE

This is to certify that this dissertation entitled **"A STUDY OF POST OPERATIVE COMPLICATIONS AFTER THYROID SURGERY"** submitted by **Dr. S. MARY PUNITHA** to the faculty of General Surgery, The Tamil Nadu Dr. M.G.R. Medical University, Chennai, in partial fulfillment of the requirement for the award of M.S. Degree Branch I (General surgery) is a bonafide research work carried out by her under our direct supervision and guidance.

Prof. Dr. S.K. PREM THAMARAI SELVI, M.S.,
Unit Chief

Dr. K.P. ARUNKUMAR, M.S.,
Professor and Head of the Department

Dr. T.P. KALANITHI, M.D.,
Dean
Coimbatore Medical College Hospital,
Coimbatore.

DECLARATION

I, **Dr. S. MARY PUNITHA** solemnly declare that the dissertation titled **"A STUDY OF POST OPERATIVE COMPLICATIONS AFTER THYROID SURGERY"** has been prepared by me.

This is submitted to The Tamil Nadu Dr. M.G.R. Medical University, Chennai, in partial fulfillment of the requirement for the award of M.S., degree Examination (General Surgery) to be held in SEPTEMBER 2006.

Place : Coimbatore.

Date : 10.04.2006. **Dr. S. MARY PUNITHA**

ACKNOWLEDGEMENT

I am extremely thankful to our **Dean Dr. T.P. KALANITHI, M.D**., for having allowed me to conduct this study in this hospital.

I am extremely grateful to **Prof. Dr. K.P. ARUNKUMAR, M.S.,** Professor and Head of the Department of Surgery for his expert help and friendly advice.

I express my sincere gratitude and extreme thankfulness to **Prof. Dr. S.K. PREM THAMARAI SELVI, M.S.,** my Unit Chief and for her valuable guidance at all stages of this study.

My greateful thanks to other Unit Chiefs **Prof. Dr. R. PERUMAL RAJAN, M.S., Prof. Dr. EASWARAN, M.S., Prof. Dr. A. RAMAMURTHY, M.S.,** and **Dr. G.S. RAMACHANDRAN M.S.,** and all **E.N.T. Unit Chiefs** for their kind Co-operation and for permitting me to study their patients.

I thank all the assistant professors in the surgical units without whom the study would not have succeeded.

I thank my Co-PGs and last but not the least, I thank all the Patients for their kind co-operation extended to me throughout the study.

INDEX

" Surgeons who take unnecessary risks and operate by the clock are exciting from the onlookers standpoint but they are not necessarily those in whose hand you would by preference choose to place yourself ".

- Theodore Kocher

Introduction

INTRODUCTION

Surgical procedures on the thyroid gland are generally safe and well tolerated. Nonetheless, the occasional complications that may follow such surgery may be life threatening or atleast permanently disabling. The complications derive from

1) anatomical variations of many vital structures associated with the gland in the neck

2) proximity of the vital structures like RLN and Parathyroid glands to the gland. Since these complications occur so infrequently despite the high volume of surgery on the thyroid no surgeon is likely to encounter a large experience with a particular complication.

A thorough knowledge of the complications, their prevention, and the ability to recognize early and accordingly manage them has become more important because of the increased frequency of surgery on this gland.

The exemplary work of Theodor Kocher during the last century in developing a safe technique for thyroidectomy resulted in a "noble prize" and this allowed continuing progress in surgery by his successors.

The importance of safe operative technique can hardly be ever emphasized for it is far better to "prevent" a complication than to "treat" it.

This study is a review of the morbidity attributed to surgical procedures on thyroid gland.

"The extirpation of thyroid gland for goitre typifies,
Perhaps better than any operation the supreme
Triumph of surgeons art"

- "WILLIAM - S - HALSTED"

Aim of the

AIM OF THE STUDY

(i) To understand the anatomy, pathophysiology and surgical procedures of thyroid gland.

(ii) To asses varies reasons for complications that arise during the surgery of thyroid gland.

(iii) To analyse the long and short-term complications of surgery done for various goiters and malignancies of thyroid gland.

(iv) To identify basic ideas for safety during thyroid surgeries.

(v) Enlist various methods to rectify the attendant complications following thyroid surgery.

Review of

REVIEW OF LITERATURE

A HISTORICAL PERSPECTIVE:

It was probably Galen who first gave a descriptive account though very brief, about the thyroid gland in the second century A.D. the time when the art of Medicine was slavish following the doctrines laid out by him. It was then well accepted that the diseases were caused by the humors-bile, phlegm and blood. Of course, no surgery was possible on tumours.

An improved description of the gland is seen in ***"De Humani Corporis Fabrica"*** of ***Vasalius*** in 1543. In 1656, ***Wharton*** who was interested in the peculiar shape of the gland, found the resemblance of it to an oblong shield and thus came the thyroid (GK Thyros-Shield). But his concept of the role of the gland in the human body was not of a physician but more of a beautician. To him it was an organ to fill spaces in front of neck, so to have a delicate smooth and elegant neck. This concept virtually explained and probably convinced many that why females did have a larger thyroid gland.

Funnier explanations of its existence in the body came later. Many were of the opinion that it was a lymphatic gland and it received worms! For some it provided the lubricants for larynx. Probably till towards the middle of the nineteenth century thyroid was only a shunting cushion to protect brain from increased blood flow.

The first ever thyroidectomy which was in the year of AD 952, is credited to the bold and venturesome operator of Moorish Origin ***Albucasis*** who did it in the Spanish Arab City of Zahra. He knew very well how to control haemorrhage by ligature and hot iron, it is quite natural for one to wonder about the indication of thyroidectomy then. 'Was it for a female who thought a few crevices, curves and a scar in front of the neck would improve her looks?

In fact, no basic techniques of thyroidectomy were known and nothing progressed for hundreds of years. Surgical literature of nineteenth century is deplete with details of fatalities from thyroid surgery. The hemorrhage which then surgeons encountered was massive and simply uncontrollable. Desperation and despotism that was growing among the surgeons were rather tremendous, statement from ***Leipzig*** in 1848 said: "If we review all we know concerning operations upon hard goitres we can only regard with tremendous aversion, these foolhardy performances". ***Samuel D.Gross*** of Philadelphia went too far to write in 1866. 'Can the thyroid in the state of enlargement be removed? Emphatically, experience answers 'No'. Should a surgeon be so foolhardy to undertake it ....every stroke of the knife will be followed by a torrent of blood and lucky it would be for him if his victim lived long enough for him to finish his blatantly horrid butchery. No honest and sensible surgeon would ever engage in it.

In this technical and professional void, the dedicated genius of ***Emile Theodor Kocher***[1] (1841-1901), Prof. of Surgery in Berne, Switzerland, was the primary force that moved thyroid surgery forward. Switzerland being a severe goitre endemic area, it provided extensive experience in thyroid surgery to Kocher in his clinic. Kocher

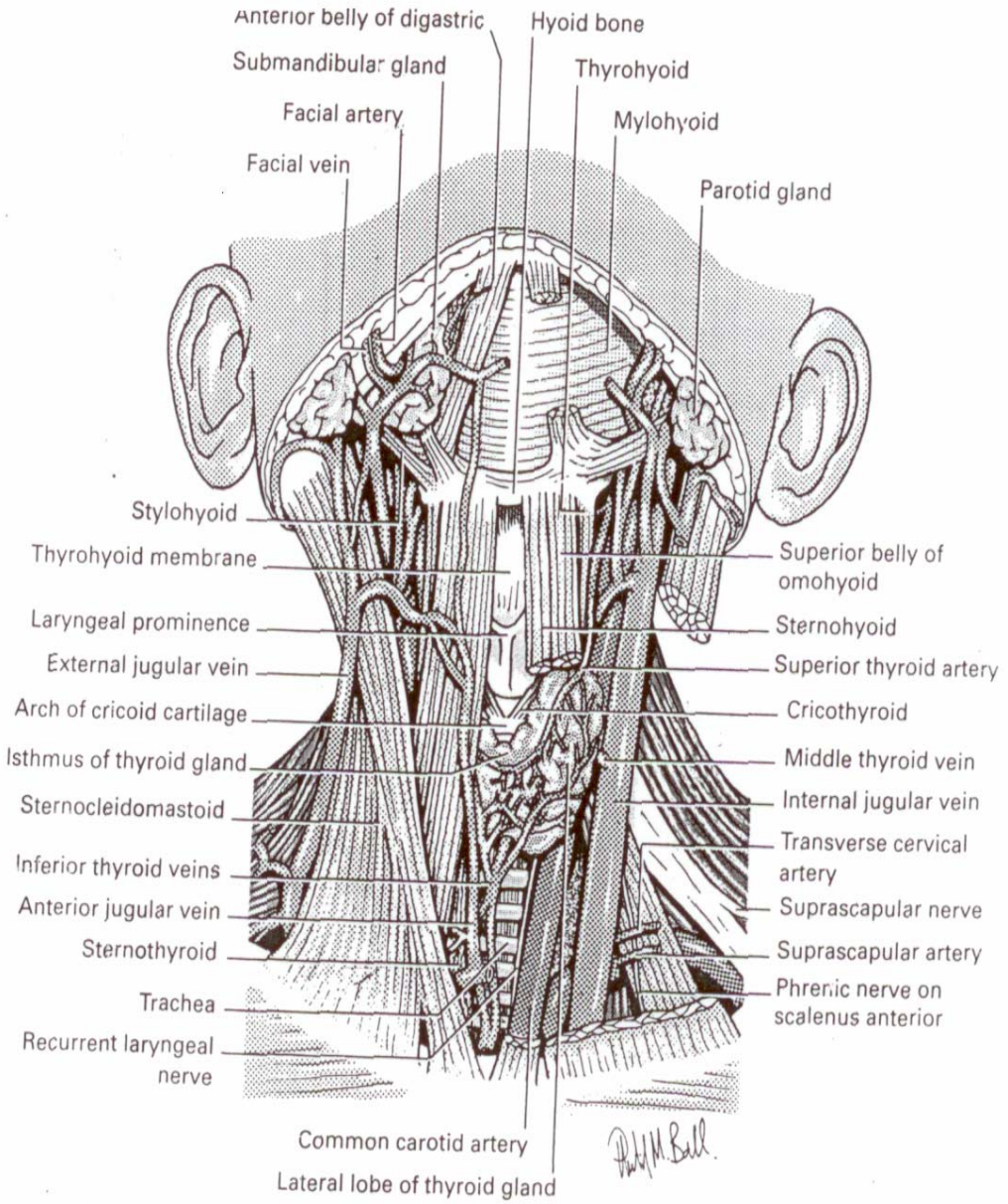
combined antiseptic techniques, thorough knowledge of anatomy and meticulous and gentle handling and dissection of tissues in his operative methods. He advocated a thyroidectomy that observed then yet to be discovered parathyroids and anatomical appreciation of recurrent laryngeal nerve (RLN). Complications of tetany and RLN injury were 'rarely' encountered and he eventually achieved an astonishingly low mortality rate of 0.5% for total thyroidectomy, which was more than 90% prior to Kocher era.

These enviable results were contradistinction to those of Billroth, Kocher's only contemporary with similarly large experience in thyroid surgery, who had complication rates orders of magnitude higher despite being a great surgeon. ***Halsted***[1], who was personally acquainted with both these men, offered the following explanation.

"I have pondered the question for many years and that the explanation probably lies in the operative method of the two illustrious surgeons. Kocher, Neat and precise, operating in a relatively bloodless manner, scrupulously removed the entire thyroid gland doing little damage outside its capsule. Billroth operating more rapidly, as I recall, with less regard for the tissues and less concern for haemorrhage, might easily have removed the parathyroids or atleast have interfered with blood supply and have left fragments of the thyroid".

Thus it is evident that Kocher's technique captivated ***Halsted*** and evoked his interest in thyroid surgery Halsted later evolved his own method of thyroidectomy and described it in a monograph 'the operative surgery of goitre' which to this day remains as a model of surgical scholarship of highest quality.

Surgical Anatomy



Thyroid gland and the front of the neck.

SURGICAL ANATOMY

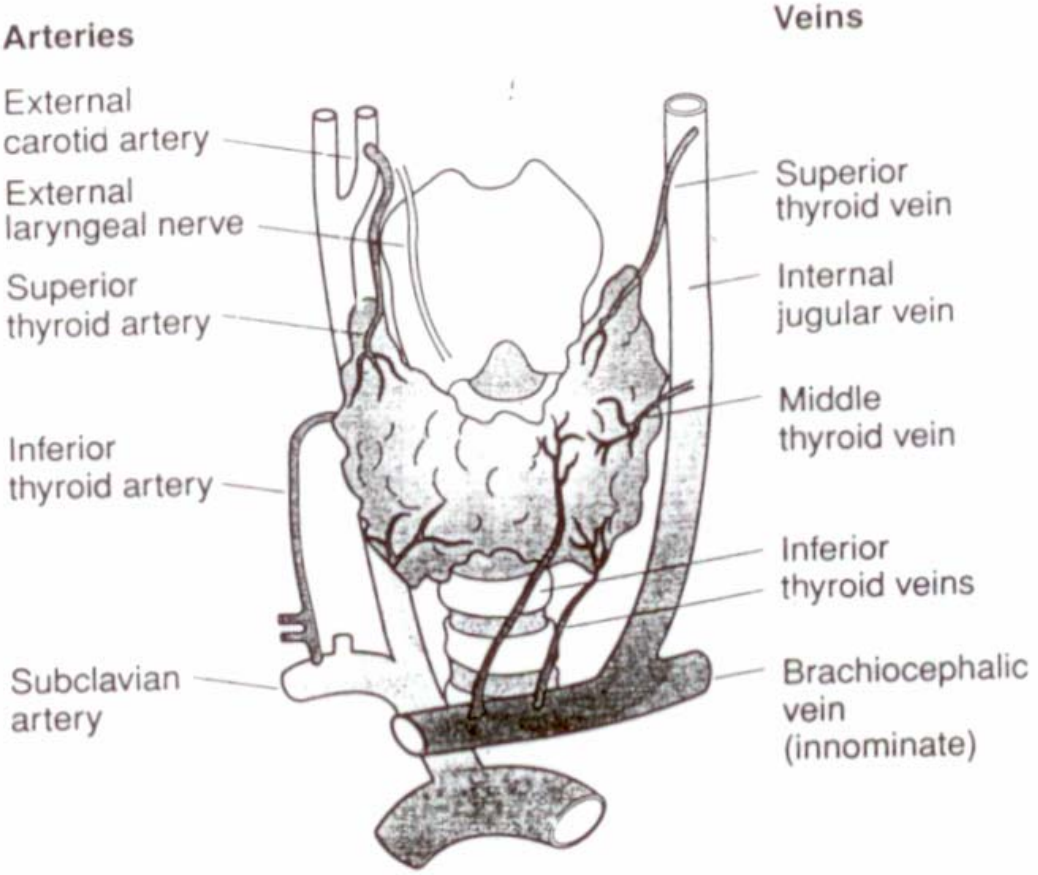
The thyroid gland is placed in front of the neck. It has an average weight of 20 gms It is composed of two lobes joined together by an isthumus. The lobes are 2 to 2.5 cms thick and wide and 4 cm long. Each lobe occupies the thyroid cartilage in its middle and extends below to the 6^{th} tracheal ring. The thyroid is related to the larynx and trachea in its posterior aspect and pharynx and oesophagus in its medial aspect posterolaterally it is related to the carotid sheath[2].

Anteriorly it is covered by the ribbon muscles namely the sternohyoid and sternothyroid, medially it is related to the inferior constrictor muscle and the cricoid cartilage.

Sometimes a pyramidal lobe ascending from the upper border of the isthmus to one side of midline is seen. It may be attached to the thyroid cartilage by a fibro muscular slip called levator glandulae thyroideae[2].

The thyroid is invested by the pre tracheal fascia a fibrous sheath derived from deep cervical fascia. It is thick on the posteromedial aspect and these thickenings are called the lateral ligaments of Berry[2].

The posterior lamina fuses with the pre-vertebral fascia and participates in the formation of a well-defined posterior wall of retro pharyngeal and retro oesophageal space. The gland can be easily separated from the trachea except at the posterior aspect of the isthmus were it is intimately connected. The gland is also provided by a true capsule, which envelops it closely and sends in numerous fibrous septae to separate the lobules and acini from one another[3].



The thyroid gland from the front.

DEVELOPMENT

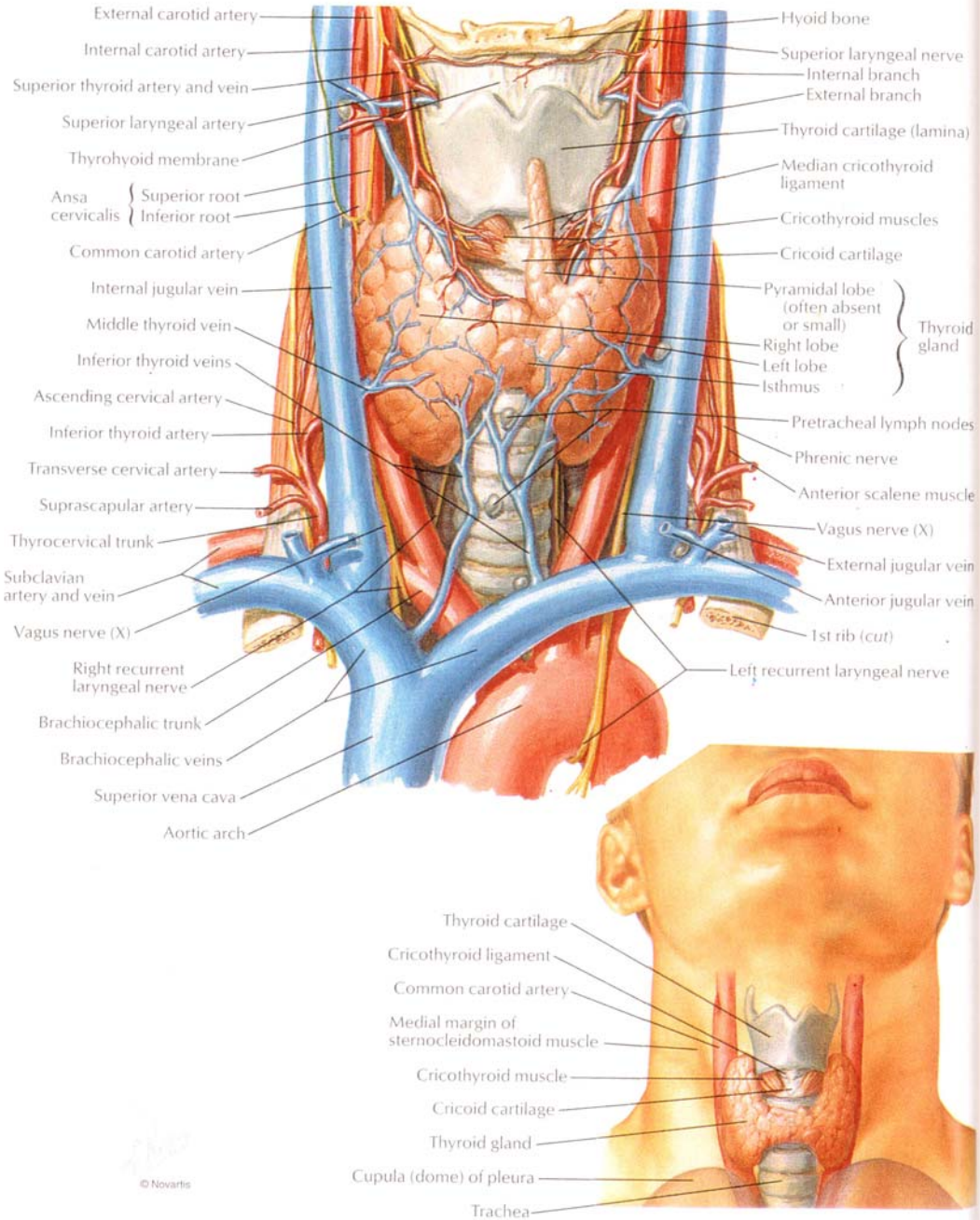
The thyroid gland develops as a median downward growth of column of cells from the pharyngeal floor between the 1st and 2nd pharyngeal pouches, subsequently marked by the foramen caecum of the tongue. The diverticulum named as median thyroid diverticulum[1] grows down in the midline into the neck, its tip bifurcates and proliferation of the cells of this bifid end gives rise to the two lobes of the thyroid gland. A portion of the duct forms the pyramidal lobe of thyroid. The proximal part of the duct usually disappears and persistence of a part of it gives rise to thyroglossal cyst[3].

Paired lateral anlages originate from the fourth branchial pouch and fuse with the median anlage at approximately the fifth week of gestation. The lateral anlages are neuroectodermal in origin (ultimobranchial bodies) and provide the calcitonin producing parafollicular or C cells, which come to lie in the superoposterior region of the gland. Thyroid follicles are initially apparent by 8 weeks and colloid formation begins by the 11th week of gestation[1].

HISTOLOGY

Microscopically, the thyroid is divided into lobules that contain 20 to 40 follicles. There are roughly $3x10^6$ follicles in the adult male thyroid gland. The follicles are spherical and average 30 μm in diameter. Each follicle is lined by cuboidal epithelial cells and contains a central store of colloid secreted from the epithelial cells under the influence of the pituitary hormone, TSH. The second group of thyroid secretory cells is the "C cells" or "parafollicular cells", which contain and secrete the hormone calcitonin. They are found as individual cells or clumped in

Thyroid Gland: Anterior View



small groups in the interfollicular stroma and located in the upper poles of the thyroid lobes[4].

BLOOD SUPPLY

The thyroid gland is supplied by the superior and inferior thyroid arteries.

Superior thyroid artery arises as the first branch of external carotid artery. At its origin it is closely related to the external laryngeal branch of superior laryngeal nerve. As it nears the superior pole of the thyroid gland it divides into an anterior and a posterior branch. A part of the anterior branch runs close to the medial side of the thyroid lobe to anastamose above the isthmus with its fellow from the opposite side[5].

Inferior thyroid artery arises from the thyrocervical trunk, which is a branch of the first part of the subclavian artery. Typically it ascends along the medial side of the anterior scalene muscle behind the prevertebral fascia and loops down medially on the anterior surface of longus colli. It penetrates the prevertebral fascia and at about the point of its branching, crosses the more vertically directed inferior laryngeal nerve, then it turns medially behind the carotid sheath to reach the middle of the back of the thyroid and divides into upper and lower branches[5].

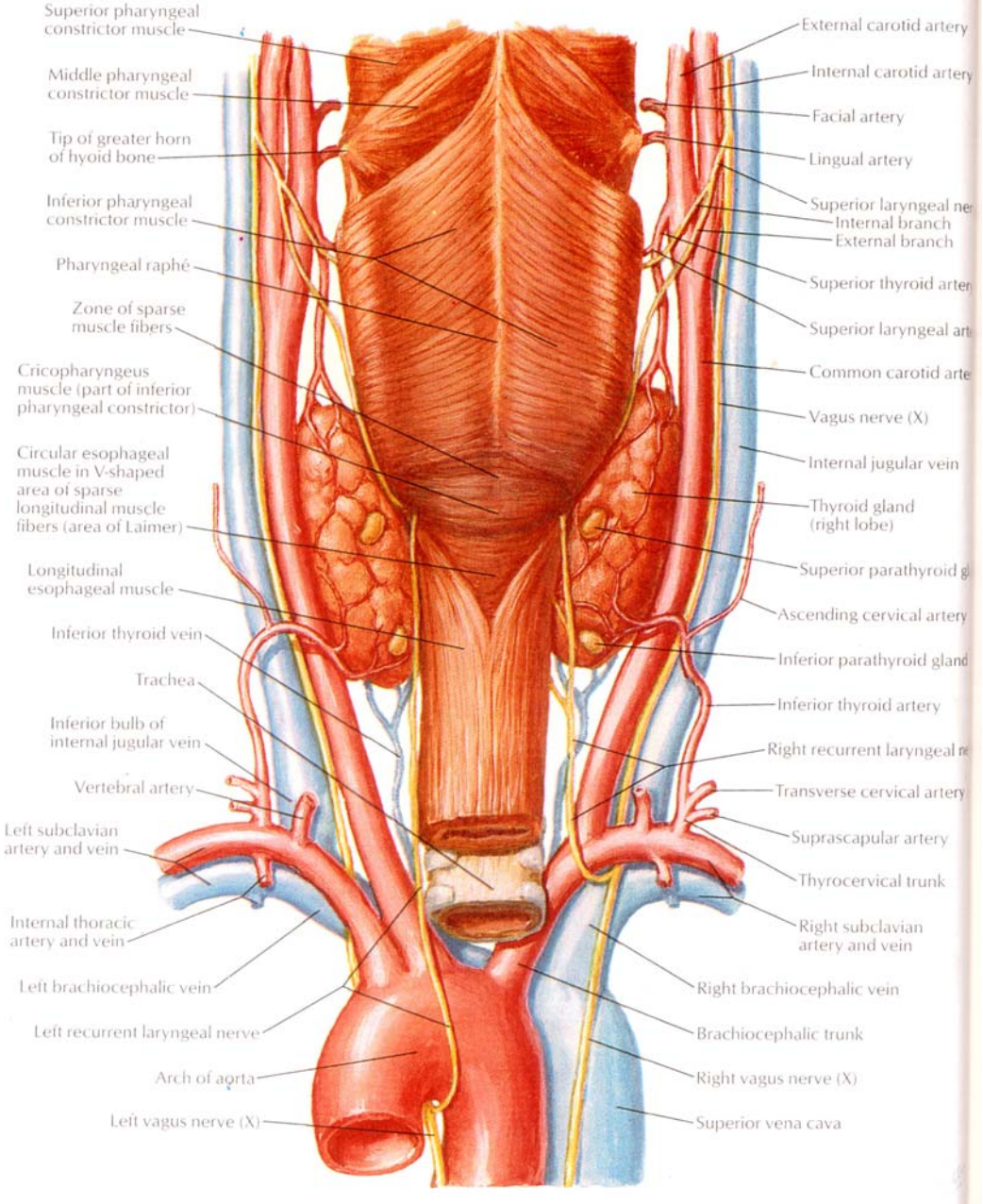
Occasionally the Arteria Thyroidea Ima branch may supply the gland arising from either the innominate artery or the aortic arch[3].

Normally anastamosis of considerable size exists between the arteries of the thyroid lobes of the same and opposite side. These vessels also anastamose with the vessels of the trachea and the oesophagus.

VENOUS DRAINAGE

The superior thyroid vein emerges from the upper pole of the gland, runs backwards across the carotids to join the internal jugular vein[4].

Thyroid Gland and Pharynx: Posterior Vie



The inferior thyroid vein emerges from the isthmus and the medial aspect of the lower part of the gland. It descends in front of the trachea to end in the inominate vein[6].

The middle thyroid vein emerges from the lower part of the lateral border of the gland. It runs across the common carotid artery to join the internal jugular vein[4].

LYMPHATIC DRAINAGE

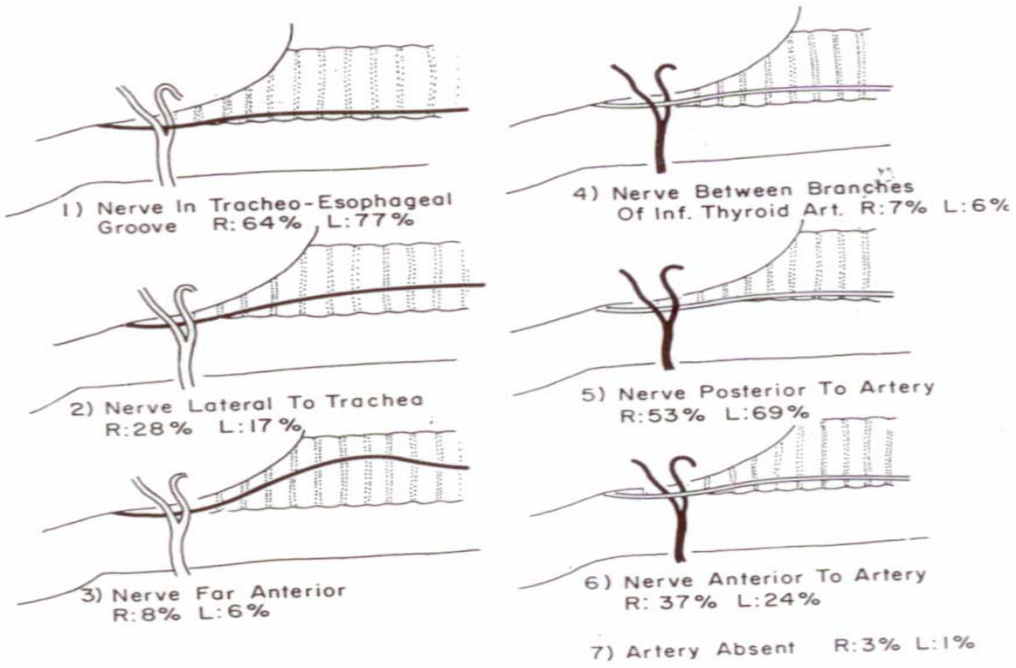
There is an extensive lymphatic network within the thyroid gland the superior lymphatic channel drains the cranial border of the isthmus, much of the medial surface of the lobes and ventral and dorsal surface of the upper part of the lobes. The inferior channel drains the major portion of the isthmus and the lower portion of the lobes.

The upper channel empties into the upper deep cervical nodes. The lower ones drain into the lower deep cervical nodes including the supraclavicular, pre tracheal and pre laryngeal nodes. There is an additional drainage from the middle of each lobe, which passes directly laterally to enter the deep cervical nodes. The deep cervical nodes communicate with the mediastinal group of lymph nodes[6].

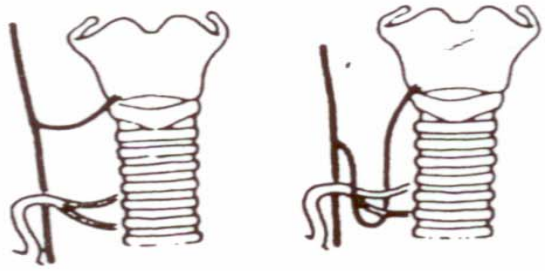
NERVE SUPPLY

The thyroid gland in relation to the recurrent laryngeal nerve and to the external laryngeal branch of the superior laryngeal nerve is of major surgical significance since damage to these structures leads to a disability in phonation.

The recurrent laryngeal nerve innervates the intrinsic muscles of the larynx except for the cricothyroid muscle, damage to this nerve leads to vocal cord paralysis on the same side. ***Reddell*** indicated that among cases in which surgeons avoid rather than expose the recurrent laryngeal nerve there is a 4% incidence of vocal cord



A



B

A. The relation of the inferior thyroid artery to the recurrent laryngeal nerve. *B.* "Nonrecurrent" right recurrent laryngeal nerves, coursing near the superior pole vessels *(left)* or around the inferior artery *(right)*. Because of the abnormal location of "nonrecurrent" nerves, they are much more likely to be damaged during operation.

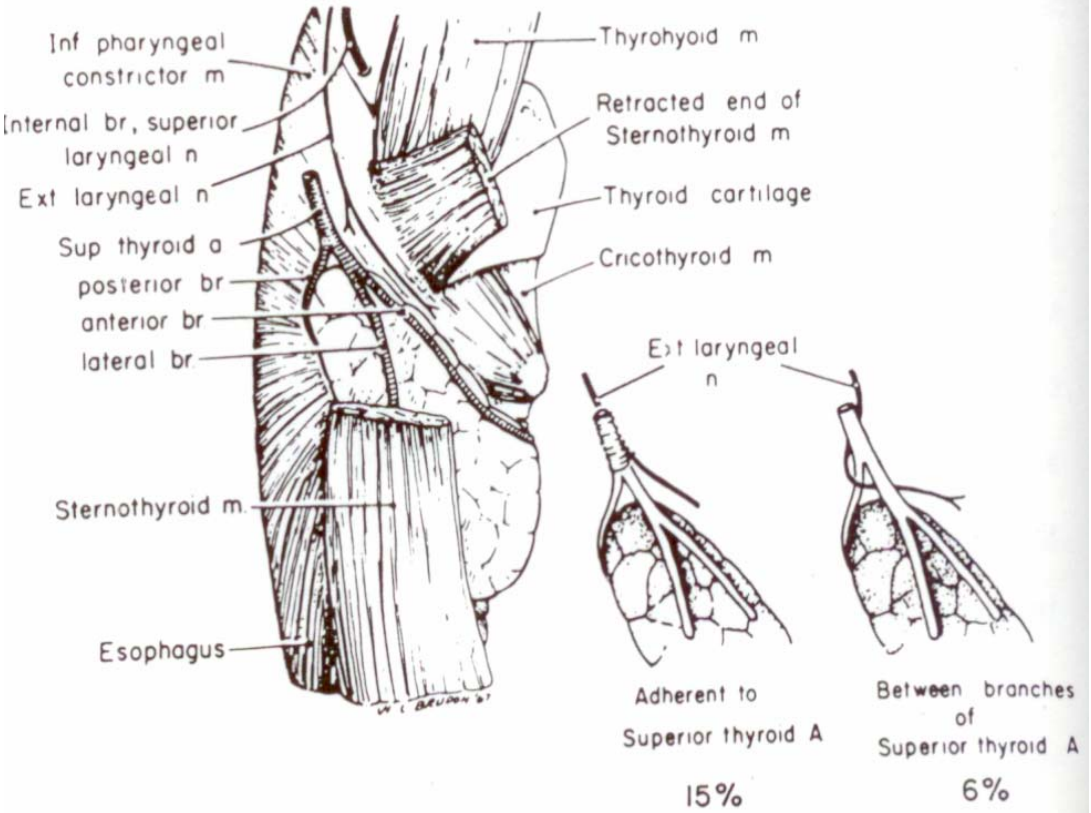
damage. It is very important for the surgeon to carefully identify this nerve at the time of operation.

The right recurrent laryngeal nerve resides in the tracheoesophageal groove in 64% of cases whereas the left recurrent laryngeal nerve was similarly located in 77% of cases[7]. The nerve was lateral to the trachea in 28% of cases on the right side and 17% on the left side. In some cases it was found antero lateral to the trachea and in danger of division during subtotal thyroidectomy if it were not carefully exposed and its course visualised. The inferior thyroid artery is often used as a landmark for demonstration of the recurrent laryngeal nerve.

The recurrent laryngeal nerve is thought by many surgeons to run posteriorly to the inferior thyroid artery. However it passed anterior to the inferior thyroid artery in 37% of cases on the right side and 27% of cases on the left. In almost 10% of cases it runs in between the branches of the artery. In 50% of cases the nerve may be embedded in the ligament of Berry which is of importance because traction of the gland would put the nerve on stretch and make it subject to section[6,10].

A non-recurrent laryngeal nerve can also occur more often on the right side than on the left[6]. On the right side it occurs in the presence of a vascular anomaly of the right subclavian artery, while a left sided non-recurrent laryngeal nerve arises when the aortic arch develops from the right side. A non-recurrent laryngeal nerve is at much greater risk of injury during operations in the neck.

The external laryngeal branch of superior laryngeal nerve innervates the cricothyroid muscle[5]. In most cases the superior laryngeal nerve lies close to the vascular pedicles of the superior poles of the gland requiring the vessels to be ligated with care to avoid injury. In 15% of cases the nerve is intimately related to the



Proximity of the external branch of the superior laryngeal nerve to the superior thyroid vessels is clearly shown.

vessels while in another 6% the nerve passes through the divisions of the superior thyroid vessels, coursing over the antero superior portion of the gland. Thus in 21% of cases this nerve is in great danger of injury when the superior thyroid vessels are ligated. In only 15% of cases does the superior laryngeal nerve enter the thyroid pharyngeal muscle before reaching the region of superior pole of thyroid gland thus protecting it from manipulation by the surgeon[8].

PARATHYROID GLANDS

These are two pairs of reddish brown glands, which are usually related to the posterior aspect of the lobes of the thyroid.

The superior parathyroid is fairly constant in position, lying behind the upper one third of the lobes and related to the lateral surfaces of the trachea[1]. It is to be invariably found in between the capsule of the thyroid and its fascial sheath. It may be placed forward in which case it is liable to be removed in subtotal thyroidectomy.

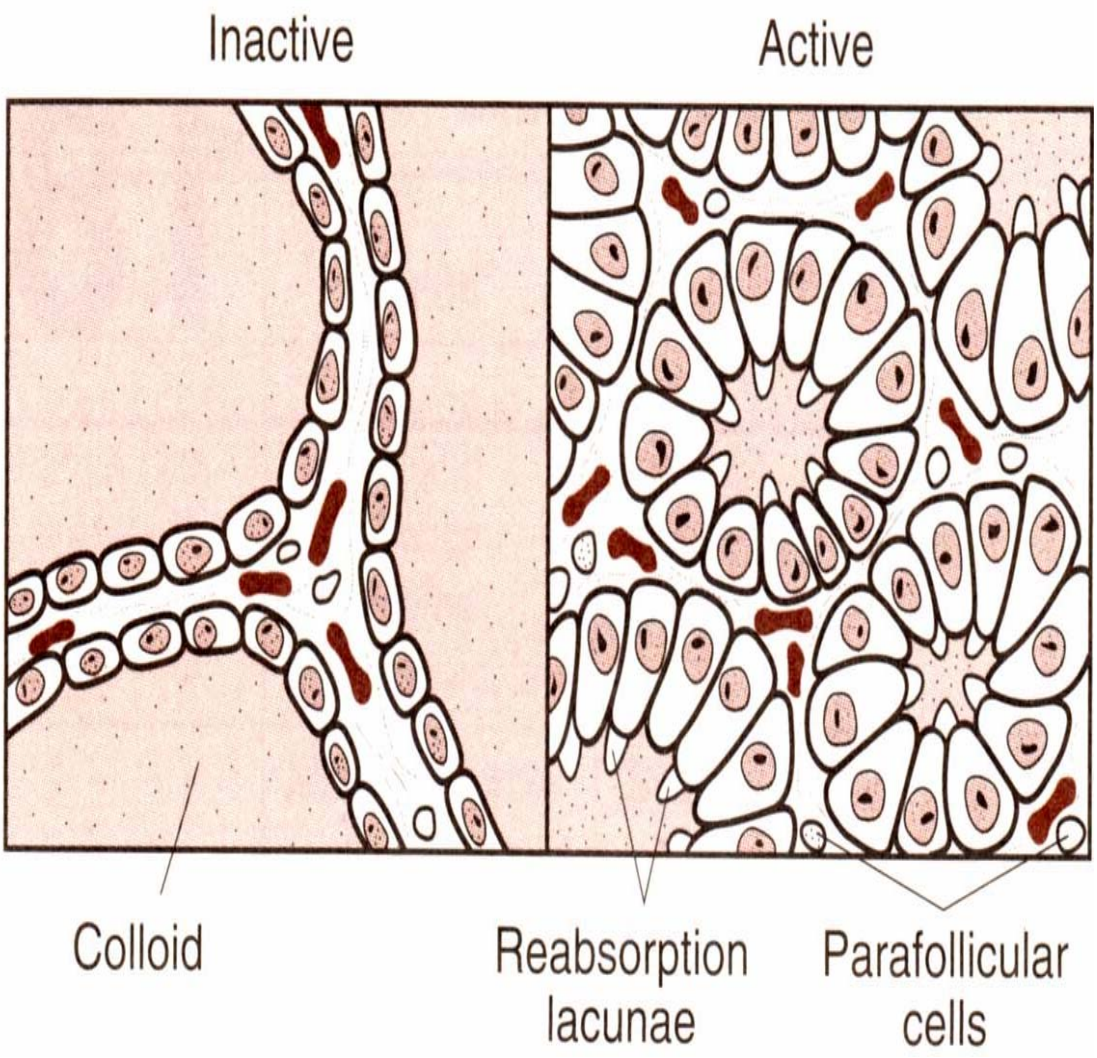
The inferior parathyroid glands are usually situated behind the lower part of the lobes either above or below the inferior thyroid artery as it enters the thyroid substance, rarely they may be found behind the oesophagus either in the neck or in the posterior mediastinum or in the retrosternal space[9].

Occasionally they are situated within the substance of the gland. The thyroid itself may be found in the retrosternal space or as a lingual thyroid.

The importance of the parathyroid lie in their intimate relationship with thyroid and the necessity to protect its integrity during surgical procedures of the thyroid gland. This is further complicated by their variable locations within the neck.

Physi

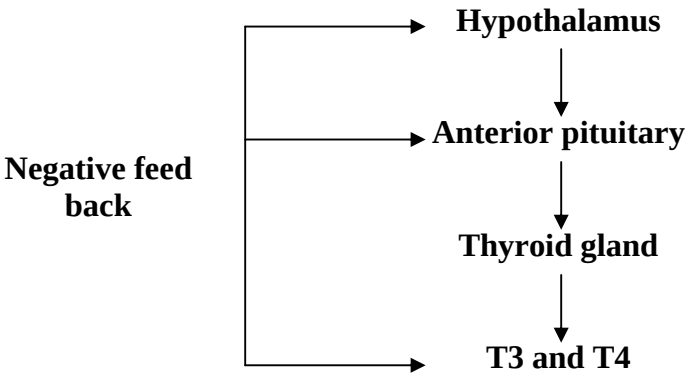
THYROID HISTOLOGY



PHYSIOLOGY

The thyroid gland produces metabolic hormones triiodothyronine - T3 and Thyroxim - T4.

The parafollicular "C-cells produce calcitonin involved in the regulation of calcium metabolism. T3 and T4 production by the thyroid gland, is regulated by the pituitary hormone, thyroid stimulating hormone - TSH - by a negative feed back mechanism, which in turn is regulated by the hypothalamic thyrotrophin releasing hormone - TRH.



T3 and T4 are synthesised from iodine and thyrosine by process of

(i) trapping of inorganic iodide by thyroid cells.

(ii) binding of Iodine to thyrosine.

(iii) coupling to form T3 and T4.

NORMAL THYROID HORMONE VALUES[1,4]

TSH- point 3 to 3.3 mU/l.

Free T3 - 3.5 to 7.5 micromol/l.

Free T4 - 10 to 30 nmol/l.

Total T3 - 1.5 to 3.5 nmol/l.

Total T4 - 55 to 150 nmol/l.

THYROID HORMONE FUNCTION

Free thyroid hormone enters the cell membrane by diffusion or by specific carriers and is carried to the nuclear membrane by binding to specific proteins. T_4 is deiodinated to T_3 and enters the nucleus via active transport, where it binds to the thyroid hormone receptor. The T_3 receptor is similar to the nuclear receptors for glucocorticoids, mineralocorticoids, estrogens, vitamin D, and retinoic acid. In humans, two types of T3 receptor genes (α and β) are located on chromosomes 3 and 17. Thyroid receptor expression depends upon peripheral concentrations of thyroid hormones and is tissue specific- the α form is abundant in the central nervous system, whereas the β form predominates in the liver. Each gene product has a ligand-independent, aminoterminal domain; a ligand binding, carboxyterminal domain, and centrally located DNA-binding regions. Binding of thyroid hormone leads to the transcription and translation of hormone-responsive specific genes.

Thyroid hormones affect almost every system in the body. They are important for fetal brain development and skeletal maturation. T_3 increases oxygen consumption, basal metabolic rate and heat production by stimulation of Na^+/K^+ ATPase in various tissues. It also has a positive inotropic and chronotropic effect on the heart by increasing transcription of the Ca^{2+} ATPase in the sarcoplasmic reticulum and increasing levels of beta-adrenergic receptors and concentration of G proteins. Myocardial α receptors are decreased and actions of catecholamines are amplified. Thyroid hormones are responsible for maintaining the amplified. Thyroid hormones are responsible for maintaining the normal hypoxic and hypercapnic drive in the respiratory center of the brain.

They also increase gastrointestinal motility, leading to diarrhea in hyperthyroidism and constipation in hypothyroidsm. Thyroid hormones also increase bone and protein turnover and the speed of muscle contraction and relaxation. They also increase glycogenolysis, hepatic gluconeogenesis, intestinal glucose absorption, and cholesterol synthesis and degradation.

Pathol

PATHOLOGY

CLASSIFICATION OF THYROID SWELLINGS:

Tumours

Benign	-	Adenoma
Malignant	-	Follicular Carcinoma
	-	Papillary Carcinoma
	-	Medullary Carcinoma
	-	Anaplastic Carcinoma
	-	Lymphoma

Goitres

Toxic	-	Primary toxicosis		
	-	Nodular toxicosis	-	Solitary nodule
			-	Multinodular
Non Toxic	-	Colloid goiter		
	-	Multinodular goitre		

Inflammatory swellings

Autoimmune	-	Chronic lymphocytic thyroiditis
		Hashimoto's disease
Granulomatous	-	deQuervain's thyroiditis
Fibrosing	-	Riedel's thyroidist
Infective	-	Acute - Bacterial,viral thyroiditis, subacute thyroiditis
		Chronic - Tuberculous, syphilitic.
Other	-	Amyloid

Investigations

THYROID SCAN - COLD NODULE

THYROID SCAN - HOT NODULE

DIAGNOSTIC TESTS OF THE THYROID GLAND.

Measurements of thyroid gland function

- Serum T4, Free thyroxine, resin T3 uptake, free thyroxine index, Serum T3, Radioactive iodine uptake, Serum TSH, Thyroxine binding globulin.

Measurers of auto immunity

- Antithyroglobulin antibodies, antimicrosomal antibodies, long acting thyroid stimulator, thyroid stimulating immunoglobulins.

Measurers of thyroid and pituitary responsiveness.

- T3 suppression test, TRH stimulation test and TSH stimulation test.

Assessment of Thyroid anatomy

- Thyroid isotope scan
- Ultra sonic scan

Assessment of thyroid histology

- Aspiration biopsy with cytology
- Core needle biopsy

Assessment of extension of goiter and to detect secondaries

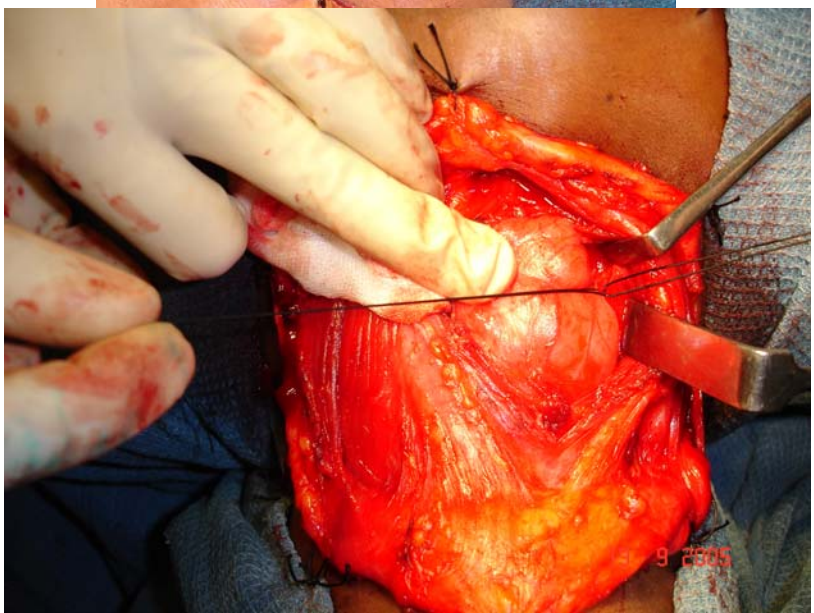
- CT Scan
- MRI

Surgical

Procedures

SUBTOTAL THYROIDECTOMY

SUPERIOR AND INFERIOR FLAPS RAISED.



SURGICAL PROCEDURES

The common types of surgical procedures performed on the thyroid gland are

Hemithyroidectomy	-	Removal of one lobe of thyroid along with the isthumus.
Subtotal thyroidectomy	-	Removal of all but a small remnant of thyroid tissue on both lobes of thyroid.
Total thyroidectomy	-	Removal of all the thyroid tissue.

Surgery of the thyroid gland requires a good knowledge of the anatomical details and a capacity to conduct meticulous and time consuming dissection of vital structures. Extent and type of surgery will depend on the certainty of diagnosis, type and pathological extent of the disease process.

PREMEDICATION AND ANAESTHESIA

In all toxic cases effective premedication is most important, in view of the effect of emotional stress and increased thyroid activity. The patient should have a good night's sleep prior to operation and reach operating theatre in a calm state of mind. General Anesthesia is favored. Endotracheal intubation is advisable in all

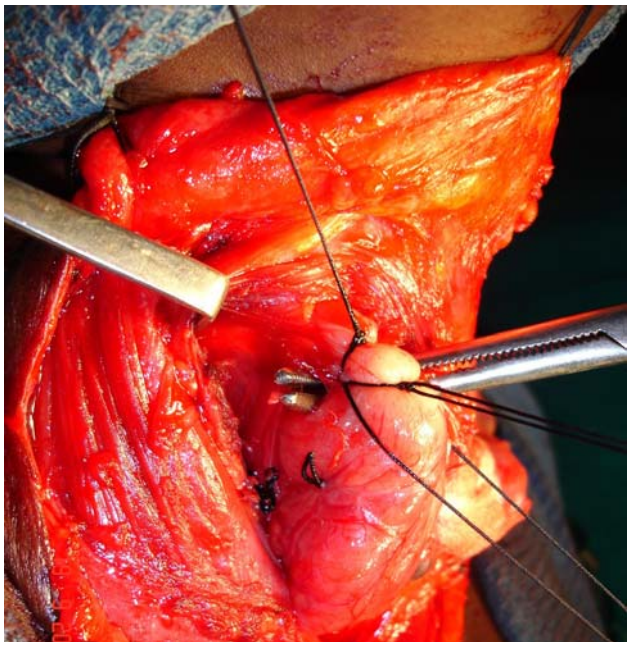
cases, and is essential when deviation or constriction of trachea has been produced since the ensured airway reduces venous congestion. The neck veins can be further emptied by tilting the operating table and patient to about 15^{o} head up position.

TECHNIQUE[6,13]

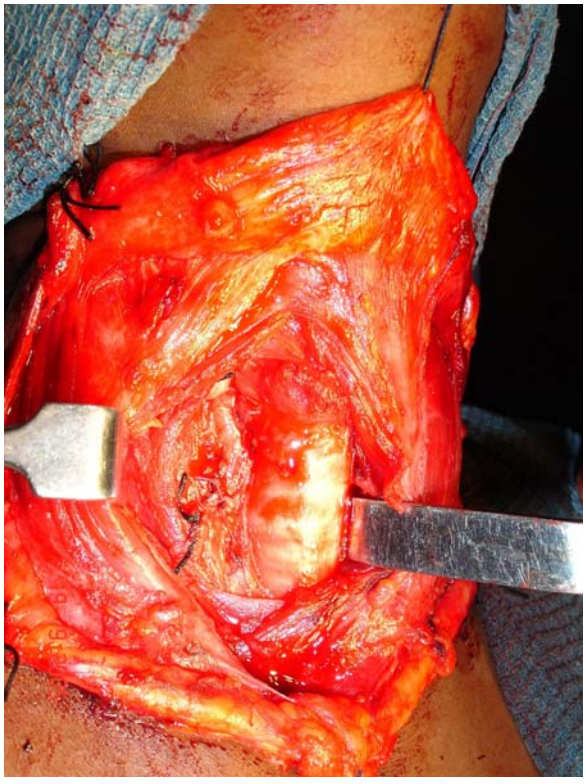
A small pillow or sandbag is placed between the shoulders so that the neck is extended and rotation of the head is avoided by keeping the head of the patient

TOTAL THYROIDECTOMY

SUPERIOR POLE LIGATION



SURGICAL BED AFTER RIGHT HEMI THYROIDECTOMY



supported on a ring. A horizontal incision is made about two fingerbreadths above the clavicles, preferably in a natural skin crease[6].

Incision is carried through the first layer (Superficial) of cervical fascia and platysma muscles. The upper and lower skin flaps are raised upward almost to the notch of the thyroid cartilage and downward to the supraclavicular region respectively.

A midline vertical incision is made through the deep layer of cervical fascia that surrounds the strap muscles. Using blunt dissection, strap muscles are separated from the underlying thyroid gland. Thyroid gland is palpated gently all around. Usually it is not necessary to transect the strap muscles, as they are readily retractable. But if exposure is not adequate, they are sectioned at or above the level of cricoid cartilage to preserve its nerve supply. If malignancy is suspected enlarged lymph nodes in the para glandular and tracheoesophageal regions are carefully looked for. A plane is developed between true and false capsule. Using gentle dissection strap muscles and fascia are dissected from the superior pole. With gentle anteromedial traction of the thyroid lobe, dissection is carried through the areolar connective tissue between thyroid and carotid sheath exposing middle thyroid vein, which is ligated and divided as far from internal jugular vein as possible.

Gentle lateral traction either using fingers or Allis forceps is applied just below, the superior polar vessels which helps to define the plane between superior polar structure, the superior laryngeal nerve and pharyngeal musculature. A right-angled Moynihan's clamp is passed into this plane to free the vessels which then are clamped and doubly ligated with silk or chromic catgut (No.1 to 2 0 are used). This

ligation is kept as low close to thyroid lobe as the pathologic process permits. Then the inferior thyroid vein is dissected free, ligated and divided. After dividing the pedicles, under direct vision and using blunt dissection lateral lobe is liberated forward and medially in a rotatory movement so that its posterior surface is fully brought in view for the visualisation of important structures.

Parathyroid glands are identified as caramel color usually an oval mass of tissue lying in some adipose material[9]. Its blood supply is to be preserved. For RLN to be identified, the dissection is carried to the tracheal rings and the Beahr's triangle[10] is demonstracted. Beahrs triangle is formed by common carotid artery, ITA and RLN. If any troublesome oozing occurs here, clamps and cautery are better avoided.

Now the thyroid isthmus is clamped and divided along with the anterior suspensory ligaments are extension and condensation of pretracheal fascia that runs from gland to cricoid cartilage and trachea. In total thyroidectomy now the complete lobe is removed without injuring the posterior structures. In subtotal resection division is carried through the thyroid gland, so as to leave posterior wedge of tissue, the amount of which varies with the pathology. In tryrotoxicosis 4-5 gms and in nodular or diffuse colloid goitre, 6-8 gms are usually preserved, roughly a piece of 2-3 cm in length and about 1.5cm wide[59]. Resection of the gland is done after applying serial clamps through the tissue over running with 0 chromic catgut is applied to the remnant thyroid tissue for hemostasis.

The same sequences are repeated if it is not hemithyroidectomy on the contralateral side. Wound is examined for bleeding sites after saline irrigation and flexing the neck. Absolute hemostasis is a basic essential.

A small soft tube drain or a corrugated rubber drain is brought out through a small opening in strap muscles and through a small opening in the lower flap. Strap muscles are reapproximated with interrupted 2 0 chromic catgut. Platysma and superficial fascia also are approximated in the same way. Skin is closed with 3 0 non absorbable, interrupted or subcuticular sutures.

Drain usually is kept for 24-48 hours. Sutures are removed on 4^{th} or 5^{th} day, which will usually be the day of discharge if there are no complications.

Complications

COMPLICATIONS AFTER SURGERY

Numerous complications may arise following surgical removal of the thyroid gland. These problems often result from either the surgical technique or from metabolic disturbances. Although the incidence of these complications is low, some problems are seen more frequently than others (Netterville & Ossoff, 1990)[14]. Complications associated with thyroid disease are,

Early Complications

1. Wound problems
   - Collections - Seroma formation
   - Infections - Subcutaneous infection
   - Deeper infection
2. Haemorrhage
   - Tension - haematoma
   - Subcutaneous haematoma
3. Respiratory obstruction
   - Laryngeal oedema
   - Collapse / kinking of trachea
4. Parathyroid insufficiency
   - Due to - removal
   - infarction
5. Thyroid storm
6. Nerve Injuries
   - Injury to superior laryngeal nerve: Recurrent laryngeal nerve paralysis

Late complications

7. Thyroid insufficiency
8. Recurrent Thyrotoxicosis
9. Hypertrophic scar - keloid formation
10. Stich granuloma

Management and Prevention of Complications

MANAGEMENT AND PREVENTION OF COMPLICATIONS

WOUND COMPLICATIONS

Seroma is a collection of serous fluid in the subcutaneous region. Serous oozing from the raw areas collects in the most dependent parts of the neck, usually in the supraclavicular fossa[16].

Treatment is by aspiration with a wide bore needle daily until no more serum collects. After each aspiration apply a mild compression dressing using fluffed up swabs held on with elastic strapping.

Avoiding unnecessary extensive dissections and obliteration of dead spaces and achieving good haemostasis reduces this complication.

INFECTION

Infection was the major cause of death from thyroid surgery during the 1800s. Today, infection occurs in fewer than 1-2% of all cases[16]. Death is unlikely if the infection is recognized and treated promptly and appropriately.

PRESENTATION

Postthyroidectomy infection may manifest as a case of 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 by fluctuance and tenderness. A deep neck abscess may manifest more subtly, but signs such as fever, pain, leukocytosis, and tachycardia should raise clinical suspicion[1].

EVALUATION

Send purulence expressed from the wound or drained from an abscess for Gram stain and cultures to indicate the choice of antibotics. CT imaging is useful when a deep neck abscess is thought possible. In addition, a deep neck abscess should raise concern for a possible esophageal perforation. A gastrographing esophageal swallow study may be useful in certain cases.

PREVENTION

The key to prevention of postoperative infection is sterile surgical technique. The routine use of perioperative antibiotics in thyroid surgery has not been proven beneficial. Johnson and Wagner[16] performed a retrospective review of 438 patients who had uncontaminated head and neck surgery at the Eye and Ear Hospital of Pittsburgh. Of 113 patients who had thyroidectomies, only 12 received perioperative antibiotics. None of the thyroidectomy patients experienced a postoperative wound infection, perioperative antibiotics are not useful. Antibiotics should not be used unnecessarily in this era of multidrug-resistant bacteria. Perioperative antibiotics are not recommended for thyroid surgery.

TREATMENT

Treat cellulitis with antibiotics that have good gram-positive coverage (eg: against staphylococci and streptococci). Drain abscesses, and direct antibiotic coverage according to culture findings. Start patients with deep neck abscesses on broad-spectrum antibiotics (eg: cefuroxime, clindamycin, ampicillin/sulbactam) until definitive culture results are available.

HAEMORRHAGE

It may be primary haemorrhage or reactionary haemorrhage. It may produce either a subcutaneous haemotoma or a deep tension haematoma.

Subcutaneous haematoma

Haemorrhage from smaller vessels may collect between deep fascia and the flap and gives rise to a diffuse swelling of the neck. This can be dealt with by evacuating the clot and insertion of sutures or by probing later after the clot has been allowed to become softer.

Achieving good haemostasis while raising the flaps and dissection of the flaps at the correct plane prevents this complication. Use of diathermy on the flaps carries the risk of producing burn wound of the skin. Bipolar can be used safely on the flaps.

Tension haematoma

Usually occurs due to slipping of vascular pedicle with haemorrhage deep to the strapmuscles. It can produce stridor and cyanosis without obvious swelling of the neck. This is an emergency and is dealt with by promptly removing the sutures at bedside, and evacuation of the clot from beneath the deep fascia thereby relieving the compression on the trachea. Then the patient is taken to the operation theatre and anaesthetised and bleeding points ligated.

Attempts to arrest the oozing haemorrhage from the wound by applying pressure dressing to the neck or attempts to pack the bleeding sites should not be done, precious time is lost and the patient get further exsanguinated and wound

infection, necrosis of skin flaps. Moreover the tension haematoma leads to the development of laryngeal oedema and airway obstruction.

Ensuring perfect haemostasis, leaving the gap in the lower end while suturing the strap muscles in the midline so as to allow the blood to come out of the relatively closed compartment into the sub-platysmal space where it can be easily detected and use of drainage tube wherever necessary prevents this alarming complication[17,19,20].

Respiratory obstruction:

Respiratory tract obstruction following thyroid surgery can be lethal and may be difficult to recognize.

The causes include

1. Tracheal compression due to haemorrhage - Tension haematoma
2. Laryngeal oedema
3. Recurrent laryngeal nerve injury
4. Laryngotracheal displacement from a goitre that is not improved with surgery.
5. Tracheal collapse of kinking.

Respiratory obstruction due to collapse or kinking of trachea is very rare. Most causes are due to laryngeal oedema.

Most important cause of laryngeal oedema is a tension haematoma. If the strap muscles are closed too tightly, haematoma may not be evident under the skin. However, the clots may dissect below the strap muscles in the peritracheal area along the deep neck spaces[17].

Total airway obstruction may progress within notime, once the critical compression in this tight compartment below the strap muscles is reached which leads to impairment of venous and lymphatic drainage leading to laryngo pharyngeal oedema. Total compression may not be the real site of obstruction but the oedema of laryngo-pharynx may be the site of obstruction.

Hence it is suggested not to close the strap muscles very tightly so that any haematoma, which develops, can become apparent in the subcutaneous region[17].

Trauma to the larynx by anaesthetic intubation and surgical manipulation are important contributory factors, particularly if the goitre is very vascular and may cause laryngeal oedema without a tension haematoma.

Unilateral or bilateral recurrent laryngeal nerve paralysis will not cause immediate post-operative respiratory obstruction unless laryngeal oedema is also present but they will aggravate the obstruction.

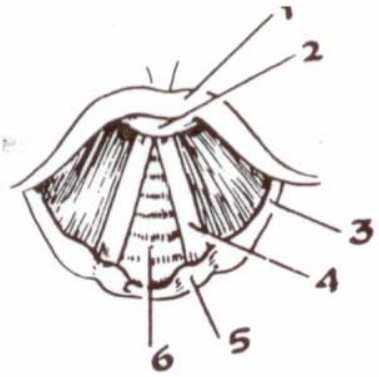
If releasing the tension haematoma does not immediately relieve airway obstruction, the trachea should be intubated atonce. An endotracheal tube can be left in place for several days. Steroids are given to reduce oedema and a tracheostomy is rarely necessary. Intubation in the presence of laryngeal oedema may be very difficult and should be carried out by an experienced anaesthetist. Repeated unsuccessful attempts may aggravate the problem and in a crisis, it is safer for the inexperienced surgeon to perform a needle tracheostomy as a temporary measure, a medicert 12G needle (diameter 2.3 mm) is highly satisfactory[17,46].

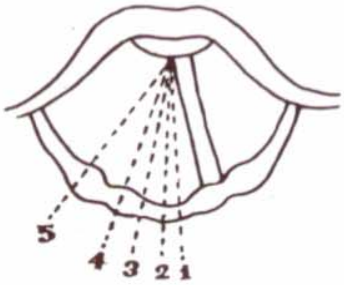
RECURRENT LARYNGEAL NERVE INJURY

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 nerve 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[42].

PRESENTATION

Patients with unilateral vocal fold paralysis present with postoperative hoarseness or breathlessness. Presentation is often subacute. Initially, the vocal fold usually remains in the paramedian position, thus affording a fairly normal voice. The paralysed vocal fold will atrophy, causing the voice to worsen. Other potential sequelae of unilateral vocal fold paralysis are dysphagia and aspiration. Bilateral vocal fold paralysis may occur following a total thyroidectomy, and usually manifests immediately after extubation. Both vocal folds remain in the paramedian position, causing partial airway obstruction. These patients may have biphasic stridor and be in respiratory distress. Occasionally, patient exhibits airway signs in the immediate postoperative period, because the airway is sufficient despite the paralyzed vocal folds. Such patients may present at followup with dyspnoea or stridor with exertion[24].



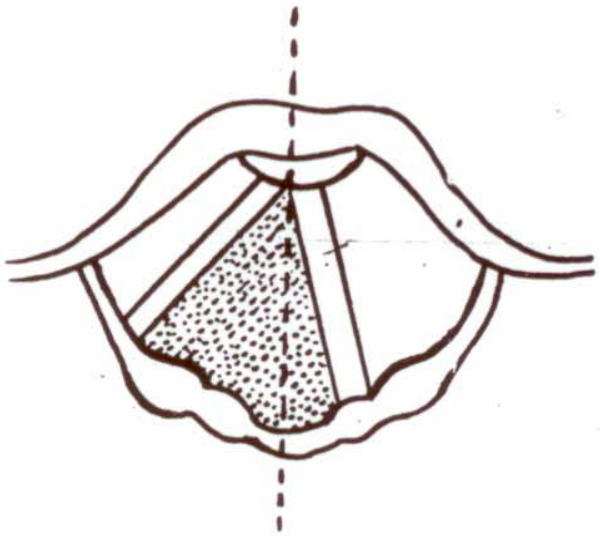


Normal Larynx

1. Epiglottis
2. Tubercle of epiglottis
3. Aryepiglottic fold
4. Vocal Cord
5. Arytenoid
6. Trachea

Vocal Cord Positions

1. Median
2. Paramedian
3. Cadaveric
4. Partial abduction
5. Full abduction



. Vocal cord paralysis
Indirect laryngoscopy showing right vocal cord in cadaveric position

EVALUATION

Techniques for assessing vocal fold mobility include indirect and fiberoptic laryngoscopy.

Laryngeal electromyography[21] (EMG) may be useful to distinguish a vocal fold paralysis from cricoarytenoid joint injury secondary to intubation. Furthermore, EMG may yield information concerning the prognosis of RLN injury. **Parnes et al[22]** performed laryngeal EMG on 24 patients with vocal fold paralysis from numerous etiologies. Since, most of these tests were performed more than 6 months after the onset of paralysis; this study reveals little regarding the usefulness of early EMG testing.

The patient with bilateral true vocal fold paralysis presenting with airway obstruction after extubation likely requires emergent reintubation or tracheotomy.

PREVENTION

Deliberate identification of the RLN minimizes the risk of injury[23,25]. When the nerve is identified and dissected, the reported RLN injury rate during thyroidectomy is 0-2.1 %. Conversely, when the nerve is not clearly identified the reported injury rate is 6.6%[24]. The RLN can be located in many ways. Intraoperative hemostasis and a thorough understanding of the anatomy are essential for nerve identification and preservation.

The course of the RLN differs on the right and left sides of the neck[7]. Classic descriptions of the RLNs hold that they ascend within the tracheoesophageal groove; however, they may in fact be lateral to it. Low in the neck, the course of the right

RLN is more oblique, lateral, and, probably, more prone to injury than the left RLN. The nerve may branch multiple times before entering the larynx. Take care to identify and preserve each branch[6].

In approximately 5 of 1000 patients, a nonrecurrent laryngeal[1] nerve is found on the right[23]. This occurs when a retroesophageal right subclavian artery arises from the dorsal side of the aortic arch. A left-sided nonrecurrent laryngeal nerve can occur rarely.

The inferior thyroid artery has been described as an important landmark for identifying the RLN[24]. However, its relationship to the nerve is subject to much 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. On the right, the nerve runs between branches of the artery in approximately 50% of patients. The nerve is found 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, but the exact relationship cannot be determined with certainty. The inferior thyroid is therefore not a dependable landmark for identifying the nerve.

Perhaps the most efficient way to identify the nerve is to locate it within the carotid triangle[24,25]. The carotid artery and trachea comprise the lateral and medial sides of the triangle, respectively. The tissue within this triangle is spread gently, a layer at a time, until the nerve is identified. Spread in the direction of the nerve, and take care not to disrupt the surrounding vascular network of the nerve. The inferior cornu of the thyroid cartilage has been described as a reliable landmark for

identification of the nerve[26]. The nerve may be identified 0.5cm below the inferior cornu.

The thyroid is attached to the trachea by thick connective tissue, called Berry ligament, at the level of the second or third tracheal ring. This is the most common site of injury to the nerve. 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 more susceptible to transection. The path of the nerve must be clearly identified.

The use of electrophysiologic monitoring of the RLN during thyroid surgery is described in the literature[27,28,29]. The latest EMG devices include a postcricoid laryngeal surface electrode and an endotracheal tube with EMG electrodes running along the tube and exposed at the glottis to contact the vocal folds. EMG has not been recommended for routine thyroid surgery, given the low rate of RLN injury. EMG may be beneficial during revision thyroid surgery, with previously radiated necks, or with very large masses, when the nerve is at increased risk[29].

TREATMENT

Generally corrective procedures for unilateral vocal fold paralysis are not done until at least 6 months after thyroidectomy. A reversible injury improves by that time. If the nerve was definitely transected during surgery, treatment for the paralyzed fold may be performed sooner.

Two surgical treatment options are available for patients with unilateral vocal fold paralysis: medialisation and reinnervation[30,31].

Medialisation of the impaired vocal fold improves contact with the contralateral mobile fold. It may be accomplished through injection laryngoplasty or laryngeal framework surgery. Type I thyroplasty is probably the most commonly performed 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. Medialisation with Gelfoam injection may be performed before 6 months if the patient desires or is aspirating. Gelfoam resorbs over time and is therefore a temporary treatment. A Silastic or Gore-Tex implant is considered permanent, although most authorities agree that no negative consequences occur if the nerve recovers function after a type I thyroplasty. In addition, the implant may be removed, although this requires another surgical procedure.

A number of reinnervation procedures have been described for 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 voice.

Primary neurorrhaphy may be used to immediately repair the transected RLN. This typically results in synkinesis because of nonselective reinnervation of abductor muscles[25]. Reinnervation procedures have been described using the phrenic nerve, ansa cervicalis, and preganglionic sympathetic neurons[32,33]. Although animal models have demonstrated EMG and histologic evidence of reinnervation, as well as restoration of vocal fold movement, experience in humans has not been as impressive[34]. Improvement in phonation quality has been documented in humans after reinnervation with the ansa cervicalis, but no movement is observed.

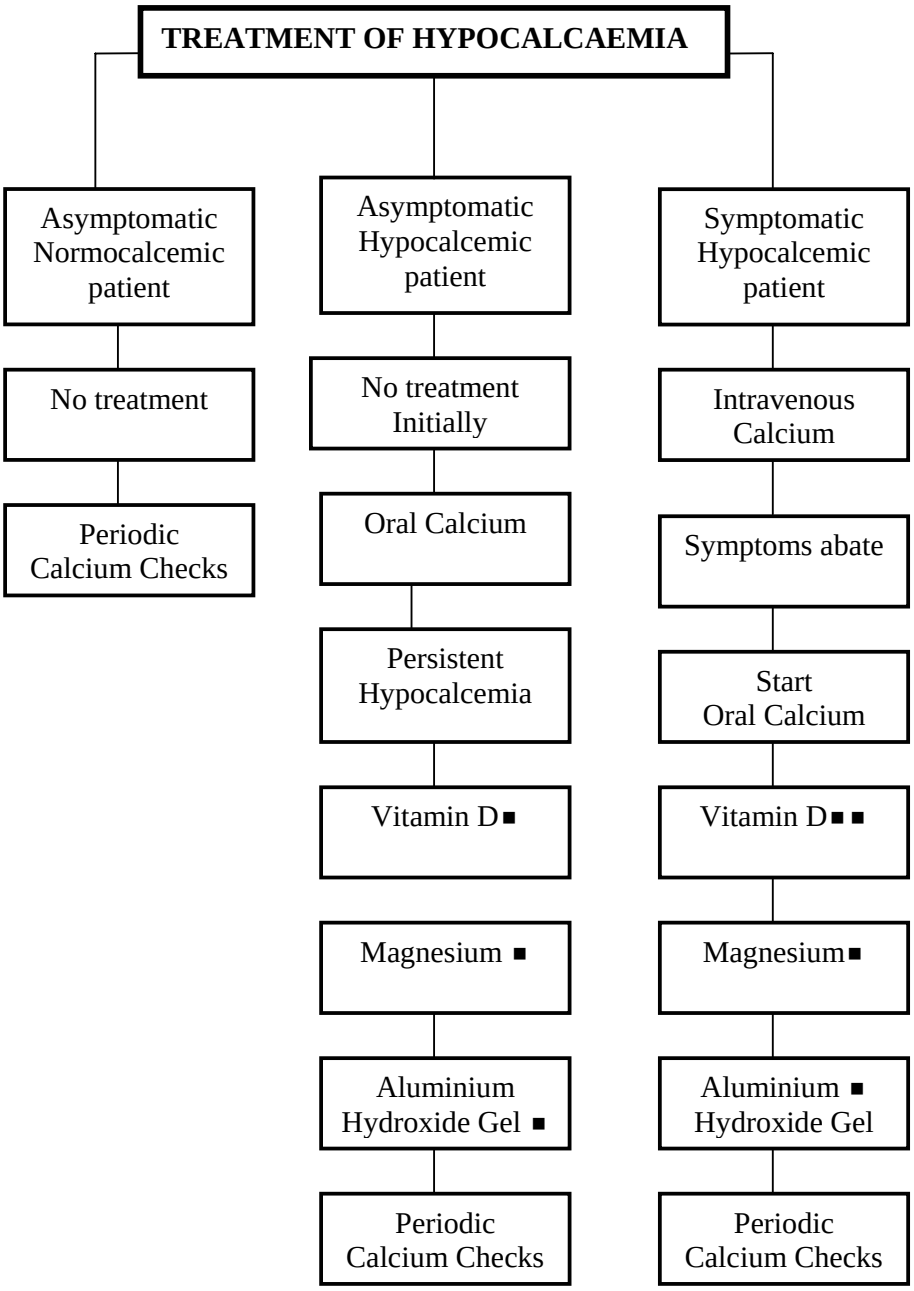
Neuromuscular pedicle transfers have been described and reportedly restore vocal fold movement[35], but these reports are limited and such success is not universal.

In the case of bilateral vocal cord paralysis, initial treatment involves obtaining an adequate airway. Tracheotomy may be emergently required. If possible, first perform endotracheal intubation. Consider exploring the neck to rule out reversible causes of nerve injury (eg: misplaced, ligature) when certain that the RLNs are both well. Intravenous steroids may be beneficial in this situation. Extubate over a Cook catheter and in a controlled setting in case reintubation is necessary. Be ready to perform an emergent tracheotomy. If nerve function has not recovered after the second extubation trial, a tracheotomy is certainly warranted.

The principal goal for surgery in the case of 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 tracheostomy decanulation. However, the patient must be counseled that his or her voice will likely worsen postoperatively. Neuromuscular pedicle transfer has been reported to improve the airway in cases of bilateral true vocal fold paralysis, but again, these reports are limited and this is not a widely accepted treatment.

HYPOPARATHYROIDISM

Hypoparathyroidism is another feared complication of thyroid surgery. The parathyroid glands produce parathyroid hormone (PTH), which is intimately involved in the regulation of serum calcium. PTH acts to increase serum calcium level by causing bone resorption, increasing renal absorption of calcium, and stimulating the synthesis of the biologically active form of vitamin D (1,25-dihydroxy vitamin D).



Treatment Strategy for post-thyroidectomy Hypocalcemia

■ To be implemented if oral calcium alone is unsuccessful

■ ■ Patient should be simultaneously started on a fast-acting Vitamin D Compound, such as (DHT) Hytakerol, and a slow onset Vitamin D Compound such as (D2) Calciferol. The fast acting form should be discontinued in 2 weeks.

1,25-Dihydroxy vitamin D increases serum calcium level by a number of mechanisms, including increasing intestinal absorption of calcium[36].

Inadequate production of PTH leads to hypocalcemia. Risk factors for hypocalcemia after thyroidectomy include resection for Graves disease or malignancy and the type of procedure performed. Total thyroidectomy, thyroidectomy with neck dessection, and repeat thyroidectomy have all been shown to increase risk for postoperative hypocalcemia[37]. The more parathyroid gland inadvertently removed, the greater the risk of hypocalcemia. PTH also increases renal excretion of phosphorous. Thus, low PTH levels result in high serum phosphorous levels.

Hypoparathyroidism, and the resulting hypocalcemia, may be permanent or transient[18]. The rate of permanent hypoparathyroidism is 0.4-13.8%[36]. The condition may be due to direct trauma to the parathyroid glands, devascularization of the glands or actual removal of the glands during surgery[37].

The rate of temporary hypocalcemia is reportedly 2-53%[37]. The cause of transient hypocalcemia postoperatively 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 2. Endothelin 1 is an acute-phase reactant known to suppress PTH production, and levels have been found to be elevated in patients with transient hypoparathyroidsm.

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, leading to decreased serum calcium levels[49].

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.

EVALUATION

Evaluate ionized calcium (or total calcium and albumin) in the perioperative period in patients undergoing total thyroidectomy. If concern for iatrogenic hypoparathyroidism exists, close follow-up care is warranted for at least 72 hours or until the calcium levels demonstrate that parathyroid function is intact.

"Chvostek" and ***"Trousseau signs"*** may both be elicited at bedside to confirm hypocalcemia. The Chvostek sign is elicited by tapping the facial nerve in the preauricular area and observing for facial contractions; the Trousseau sign, by induction of carpal spasm upon inflation of a blood pressure cuff.

PTH levels may be checked to confirm the cause of hypocalcaemia and to identify recovery of parathyroid gland function. Serum phosphorous levels are

elevated in patients with hypoparathyroidism secondary to decreased renal excretion, which may help distinguish low PTH levels from other etiologies of hypocalcaemia (eg: hungry bone syndrome). Consider other causes of hypocalcaemia as well (eg: medications, hypomagnesemia, renal failure, pancreatitis).

PREVENTION

The best way to preserve parathyroid gland function is to identify the glands and to maintain their blood supply[38,39]. 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, intimately associated with the RLN[9]. Twenty-two percent of the superior parathyroid glands were on the posterior surface of the upper lobe of the thyroid. Approximately 1% of the superior glands were located behind the junction of the hypopharynx and upper esophagus.

The study demonstrated that the location of the inferior parathyroid glands is more variable. Forty-two percent were found 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 within 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, which explains the close association of these structures[5]. 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 from direct branches off the thyroid gland.

The keys to parathyroid preservation are to identify the parathyroids and preserve their blood supply by ligating all vessels distal to them[39]. Ligate vessels as close to the thyroid gland as possible. Recognition of the parathyroid glands, which appear in a variety of shapes and have a caramel-like color, is critical. When they lose their blood supply, they turn black. The devascularized gland (confirmed pathologically by frozen-section analysis), should be removed, cut into 1-to-2-mm pieces, and reimplanted in the sternocleidomastoid muscle or the forearm. The location should be marked with a permanent suture or Ligaclip. Sometimes, the inferior glands are so anterior that preserving their blood supply is difficult. In this case, the glands should be reimplanted as well. Once removed, examine the thyroid gland. Any parathyroid glands in excised thyroid tissue are then reimplanted[6].

TREATMENT

Patients who have asymptomatic hypocalcemia in the early postoperative period should not be treated with supplemental calcium. Authorities believe that the hypocalcemic state stimulates the "stunned" parathyroid glands to produce PTH[14].

Patients who have symptomatic hypocalcaemia in the early postoperative period, or whose calcium levels continue to fall require treatment[14]. In the symptomatic patient replace calcium with intravenous calcium gluconate. Ten mL of 10% solution (1g) may be administered over 10 minutes. A calcium drip may be started at 1-2 mg/kg if symptoms do not resolve. Titrate the infusion to the patient's symptoms and calcium levels. Start oral calcium when the patient is able to tolerate

this. One to 2 g of elemental oral calcium should be supplied each day. Calcium carbonate at a dose of 1250 mg provides 500 mg of elemental calcium; therefore, the patient should take 2500-5000 mg of calcium carbonate a day. The patient will need concomitant vitamin D replacement in the form of calcitriol at a dosage of 0.25-1 mcg/d.

The authors recommend assistance from an endocrinologist to ensure close monitoring of calcium levels and for 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 probably indicates permanent hypoparathyroidism[2,14].

THYROTOXIC STORM

Thyrotoxic storm is an unusual complication of thyroid surgery[18]. The condition may occur from manipulation of the thyroid gland during surgery in the hyperthyroid patient and can occur intraoperatively or postoperatively. Thyrotoxic storm is potentially lethal and must be dealt with astutely.

PRESENTATION AND EVALUATION

Signs of thyrotoxic storm in the anesthetized patient include evidence of increased sympathetic output, such as tachycardia and hypertherimia. 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.

PREVENTION

Preoperative awareness of the hyperthyroid patient and appropriate medical management are the keys to prevention of thyrotoxic storm, medications used in the hyperthyroid patient include propylthiouracil, methimazole, and/or Lugol solution. Beta-blockers are useful in controlling peripheral manifestations of hyperthyroidism. Steroids may be useful preoperatively.

TREATMENT

The first step when faced with a thyrotoxic crisis during thyroidectomy is to stop the procedure. Intravenous beta-blockers, propylthiouracil, sodium iodine and steroids are administered to control sympathetic activity, thyroid hormone release, and hyperthermia. Use cooling blankets and cooled intravenous fluids to reduce body temperature. Carefully monitor oxygenation, because oxygen demands increase dramatically during thyroid storm[4].

SUPERIOR LARYNGEAL NERVE INJURY

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[5]. The external branch provides motor function to the cricothyroid muscle and is at risk during thyroidectomy[8]. This muscle is involved with elongation of the vocal folds. Trauma to the nerve results in an inability to lengthen a vocal fold and thus to create a higher-pitched sound. The rate of injury to the external branch of the SLN has been estimated at 0-25%[8]. This rate is probably underestimated, because the diagnosis is frequently missed.

PRESENTATION

The clinical presentation of a patient with SLN paralysis may be quite subtle. Most patients do not notice any change. Occasionally, one presents with mild hoarseness or decreased vocal stamina. For the singer and or professional voice user, however, paralysis of the SLN may be career threatening. The most damaging consequence is loss of the upper register[3].

EVALUATION

Diagnosing an SLN injury with indirect or fiberoptic laryngoscopy is very difficult[40]. Posterior glottic rotating 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.

The use of videostroboscopy and laryngeal EMG has increased otolaryngologists' and speech pathologists' ability to diagnose SLN paralysis. Videostroboscopy demonstrates an asymmetric mucosal travelling wave. EMG demonstrates cricothyroid muscle denervation.

PREVENTION

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.

Most surgeons agree that, in contrast to the RLN, identifying the SLN 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[40]. electrophysiologic monitoring of the SLN has been described but is not recommended for routine use[58].

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 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.

TREATMENT

The only treatment currently available for injury to the external branch of the SLN is speech therapy.

HYPOTHYROIDISM

PRESENTATION

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[4].

EVALUATION

The most useful laboratory test for detection or monitoring of hypothyroidism is in the postthyroidectomy patient is the thyrotropin (thyroid-stimulating hormone [TSH]) level. Total thyroxine (T_4) and triiodothyronine (T_3) levels may be useful to fine-tune levothyroxine (Synthroid) dosing but are less likely to be helpful in the typical postoperative patient.

PREVENTION

Hypothyroidism is an expected sequela of total thyroidectomy. In goiter surgery, the surgeon must balance the risks of leaving too much thyroid tissue thus requiring repeat surgery, with excising too much thyroid tissue, which results in hypothyroidism. This balance comes with experience and adequate follow-up care[18].

TREATMENT

Start hypothyroid patients on levothyroxine (~1.7 mcg/kg/d). Check the thyrotropin level in approximately 4-6 weeks, and adjust the dosage appropriately. Patients who are to receive postoperative radioiodine scaning must be off levothyroxine before the procedure.

HYPERTROPHIC SCAR OR KELOID SCAR

This is more likely to form if the incision overlies the sternum. Especially the drain site or when the wound is complicated by wound infection and gaping.

Intralesional injections of corticosteroid should be given at once and repeated monthly if necessary.

STITCH GRANULOMA

This may occur with or without sinus formation and is seen after the use of non-absorbable suture material.

Absorbable ligatures and sutures must be used throughout thyroid surgery.

INFREQUENT POSTOPERATIVE COMPLICATIONS

Some postoperative complications, such as sympathetic nerve injury, chylous fistula, infection, and thyroid storm, have decreased in incidence over the past several

years. The decrease is associated with a better understanding of thyroid anatomy, as well as advances in the medical treatment of thyroid disease (Netterville & Ossoff[14], 1990).

Injury to the sympathetic chain from stretching or compressing during thyroid surgery may result in the development of Horner's syndrome. This condition is characterized by such symptoms as contraction of the pupil, partial ptosis of the eyelid, and anhydrosis of the ipsilateral side.

Damage to the thoracic duct during thyroid surgery may result in a chylous fistula. This complication manifests itself by the presence of profuse and continuous drainage of chyle (a milky or opaque fluid) from the operative would.

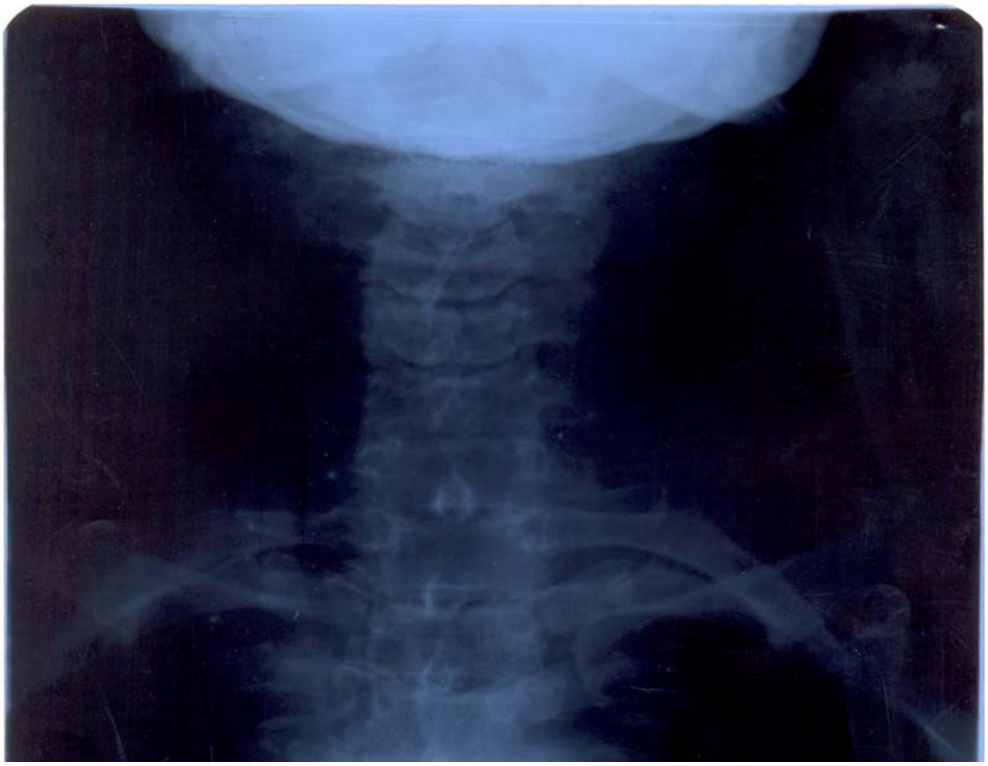
MORTALITY

The mortality rate from thyroid surgery during the 1800s was approximately 40%. Most deaths were caused by infection and hemorrhage. Sterile operative arenas, general anesthesia, and improved surgical techniques have made death due to thyroid surgery extremely rare today.

Materials and

FOLLICULAR CARCINOMA THYROID

ROENGENOGRAM OF NECK - AP VIEW FOR THE SAME PATIENT SHOWING LATERAL DISPLACEMENT OF TRACHEA TO RIGHT SID



MATERIALS AND METHODS

MATERIALS

The present study included 110 patients who underwent thyroid surgery in various surgical units from July 2004 to May 2005 in the Department of General Surgery, Coimbatore Medical College Hospital, Coimbatore.

CRITERIA OF ELIGIBILITY

Age **:** All age groups from 14 to 75 years were included in this study.

Sex **:** Both males and females were included.

Pathology **:** All Benign, Malignant, Toxic and non toxic cases who underwent surgery were included.

Surgery **:** Surgeries on thyroid included are Hemithyroidectomy, Subtotal Thyroidectomy and Total Thyroidectomy.

CRITERIA FOR EXCLUSION

Patients who had recurrent laryngeal nerve paralysis and hypocalcaemia preoperatively were exempted from this study.

Hypothyroidism following TT was not considered as complication in this study.

METHODS

All cases of Thyroidectomy underwent following basic investigations preoperatively.

-Chest X-ray.

-X-ray neck - both lateral and AP view.

-ECG.

-Basic haematological and biochemical investigations.

-Vocal Cord examination by ENT surgeon with indirect laryngoscopy.

-FNAC of Thyroid nodule.

-Thyroid function tests.

All patients underwent Laryngoscopy examination as follows,

1. Pre operatively by IDL.

2. After surgery once the patient had recovered from anaesthesia by DL

3. At the time of discharge and earlier by ENT surgeon with IDL if necessary.

All the toxic cases were well controlled preoperatively with the use of Antithyroid drug-carbimazole AND beta-blockers. Lugols iodine was given 10 days prior to surgery to reduce the vascularity of the gland and to make the gland firm.

TECHNIQUE

Surgical procedures that were performed include

Hemithyroidectomy	-	25
Subtotal thyroidectomy	-	67
Total thyroidectomy	-	15
Total thyroidectomy with Lymphnode dissection	-	3

All the cases underwent surgery under general anaesthesia through a low collar crease transverse incision. The surgeries were performed by surgeons of good experience in the surgical field. Identification of the recurrent laryngeal nerve was done routinely. Drain was used in all the cases and was kept in situ for about 48 hours. Both lateral and suprasternal areas were used to position the drain. Corrugated rubber drain or suction drain both were used. Vocal cords examination was done at the time of extubation by the anesthesiologist with direct laryngoscopy.

Blood transfusion was given in the intraoperative period for 19 patients and for 3 patients in the postoperative period.

Serum calcium level was measured preoperatively and postoperatively on the 3rd postoperative day for all cases of subtotal and total thyroidectomy.

All the cases were discharged on the 3rd or 4th POD except those who had complications.

FOLLOW UP

Follow up of cases were done postoperatively for a variable period of time, atleast for 6 months at 2 weeks interval for the first two months and then monthly interval for one year after that patients were reviewed once in 6 months .

Clinical examination was done for all cases.

Serum calcium was checked for all the cases who had postop. hypocalcaemia.

TSH was done for all the patients who had malignance thyroid disease once in 3 months and for other patients wherever necessary.

Many of our cases were lost for follow up.

There was no mortality in this study.

No case of Ectopic thyroid was diagnosed in this study.

Observ

OBSERVATION

This study was performed in a series of 110 patients of which 95 were females, 15 were males. Most of the cases were in the middle age group 20 - 40 years of age.

67 patients underwent subtotal thyroidectomy, 25 underwent hemithyroidectomy, 15 underwent total thyroidectomy and 3 underwent lymphnode dissection in addition to total thyroidectomy. (Table - 4)

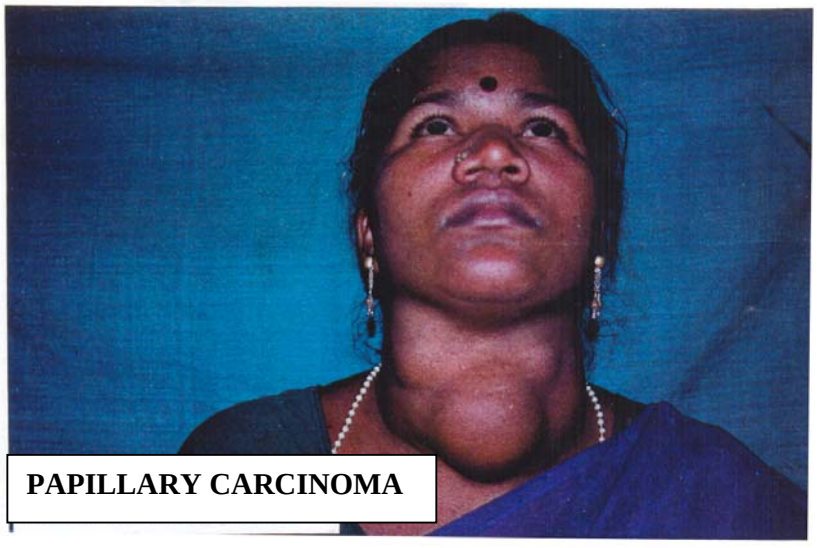
The pathological break up of thyroidectomies given in Table - 3.

Majority of the patient were with non-toxic MNG and they all underwent subtotal thyroidectomy. (Table-3)

The split-up of cases along with the type of surgery performed and the complications encountered in each of the category according to the indication for surgery is given below.

PAPILLARY CARCINOMA THYROID AFTER TOTAL THYROIDECTOMY

WOUND INFECTION WITH MARGINAL NECROSIS



PAPILLARY CARCINOMA

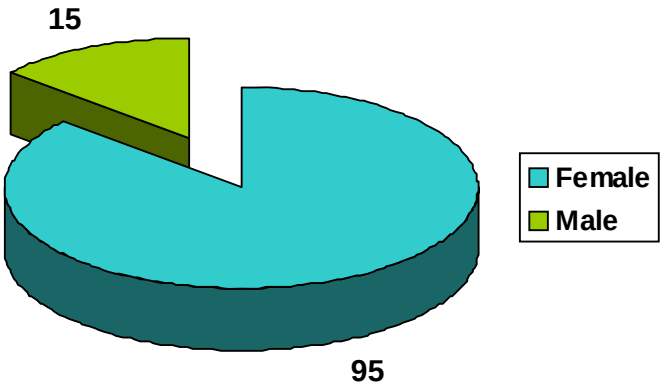
TABLE 1

SEX DISTRIBUTION

SEX	NUMBER OF CASES	%
Male	**15**	**13.63**
Female	**95**	**86.36**

FIGURE - 1

SEX RATIO



PAPILLARY CARCINOMA THYROID

TABLE - 2

AGE DISTRIBUTION

AGE IN YEARS	NUMBER OF CASES	%
Upto 20	**6**	**5.45**
21-30	**32**	**29.09**
31-40	**31**	**28.18**
41-50	**25**	**22.72**
51-60	**8**	**7.27**
61 and above	**7**	**6.36**

TABLE - 3

PATHOLOGICAL BREAK UP OF THYROIDECTOMIES.

DURING THE SAME PERIOD MAY 2004 TO JULY 2005

DISEASE	Number	%
Non-toxic multinodular goitre	**48**	**43.63**
Solitary nodule thyroid benign	**24**	**21.81**
Toxic multinodular goitre	**13**	**11.81**
Papillary carcinoma thyroid	**11**	**10.00**
Follicular carcinoma thyroid	**7**	**6.36**
Primary toxic goitre	**4**	**3.63**
Colloid goitre	**3**	**2.72**


19 9 2005

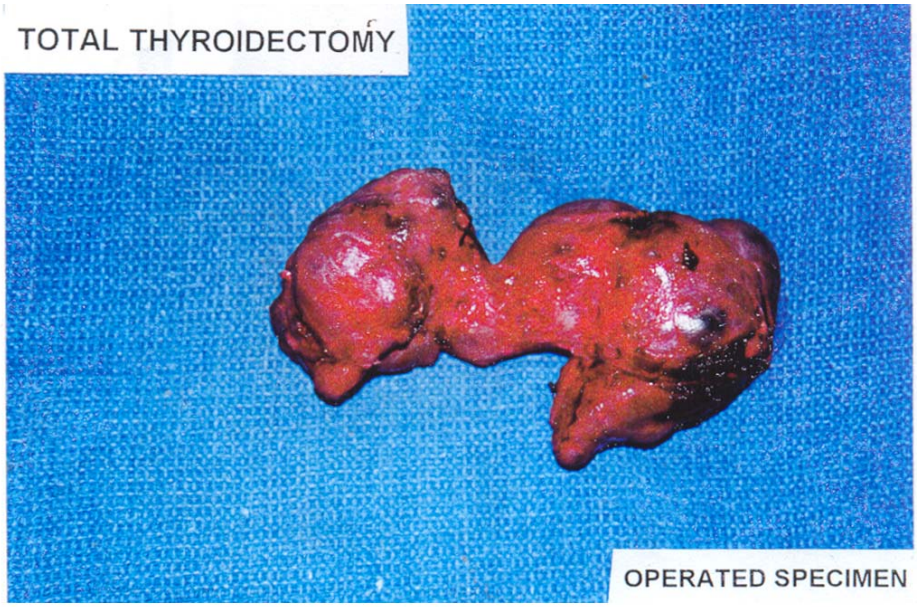

TOTAL THYROIDECTOMY
OPERATED SPECIMEN

FIGURE - 2

PATHOLOGICAL DIAGNOSIS

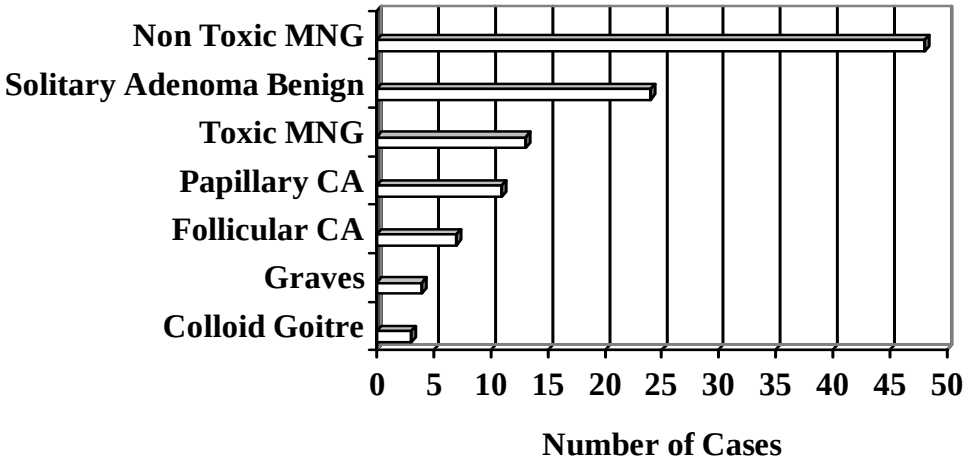


TABLE - 4

BREAK UP OF TYPE OF THYROIDECTOMIES STUDIED IN THE PRESENT SERIES

SURGERY	Number	%
Subtotal thyroidectomy (STT)	**67**	**60.90**
Hemithyoidectomy (HT)	**25**	**22.63**
Total thyroidectomy	**14**	**12.72**
Total thyroidectomy with lymphnode dissection (functional & unilateral)	**3**	**2.72**
Completion thyroidectomy	**1**	**0.90**


Trousseau's sign

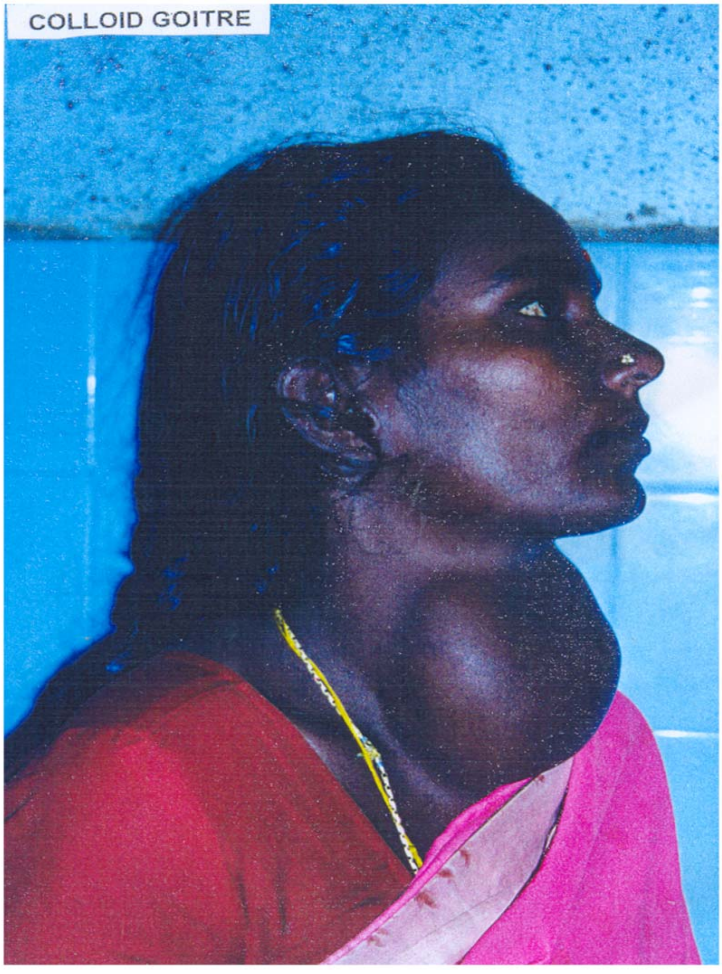

COLLOID GOITRE

FIGURE - 3

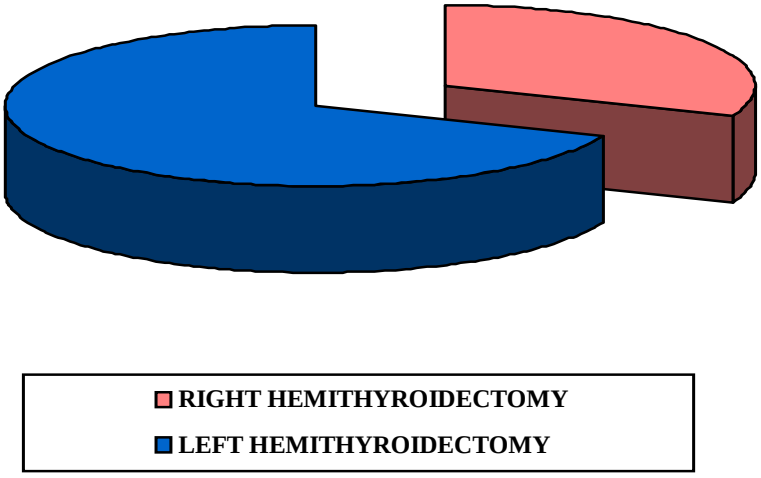
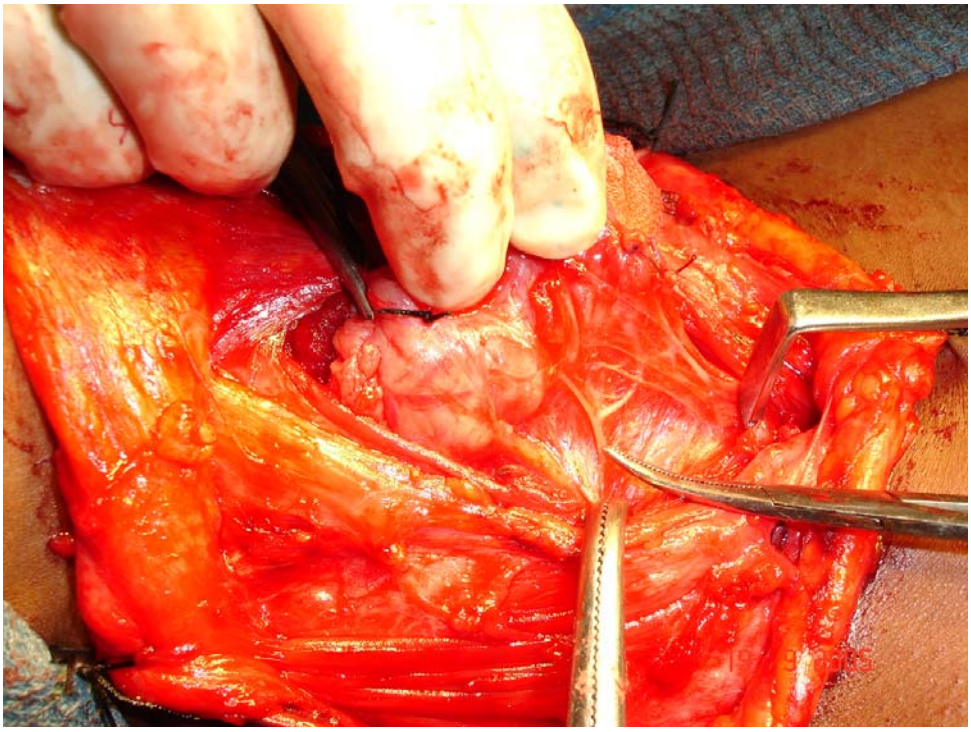


TABLE - 5

COMPLICATION RATE

COMPLICATIONS	No. of Cases	%
Unilateral cord palsy	**5**	**4.54**
Post operative tetany	**6**	**5.45**
Post operative haemorrhage	**1**	**0.90**
Tracheomalacia	**1**	**0.90**
Wound infection	**1**	**0.90**
Hypothyroidism	**1**	**0.90**
Nil complications	**98**	**89**

HEMI THYROIDECTOMY RLN IDENTIFIED



TOTAL THYROIDECTOMY MIDDLE THYROID VEIN IDENTIFIED

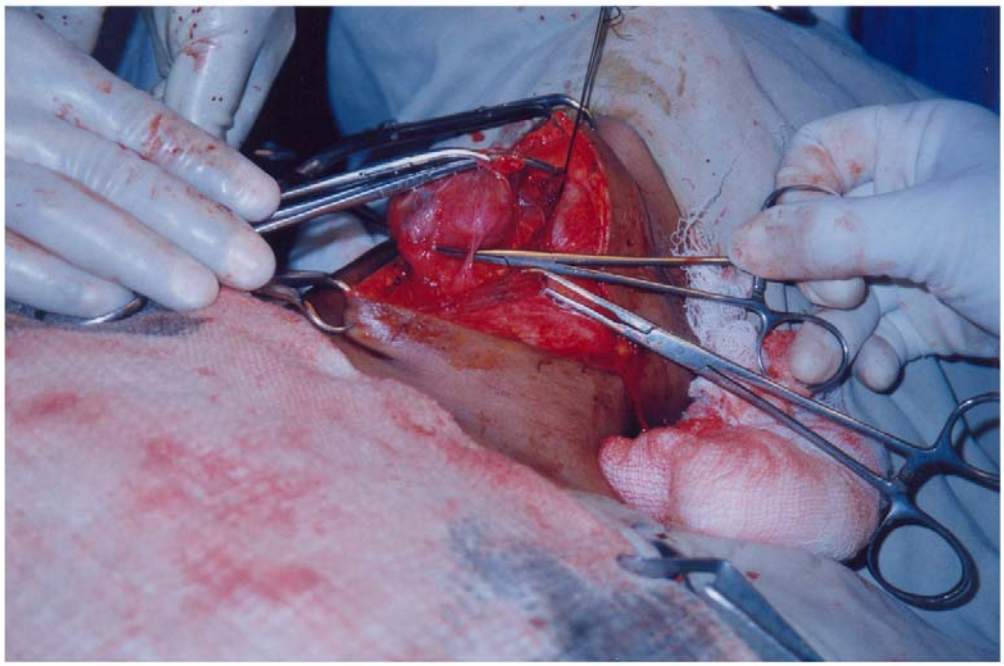


FIGURE - 4

COMPLICATIONS

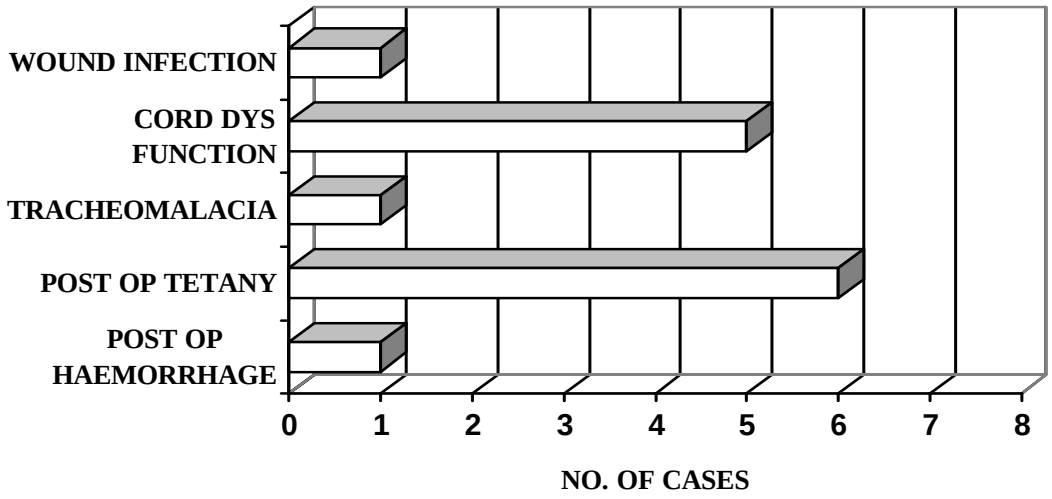


TABLE - 6

THE TYPES OF SURGERY AND ITS COMPLICATIONS

	Hemi Thyroidectomy	**Subtotal Thyroidectomy**	**Total Thyroidectomy**
Right cord palsy	**0**	**1**	**1**
Left cord Palsy	**1**	**1**	**1**
Tetany	**0**	**3**	**3**
Postoperative haemorrhage	**0**	**1**	**Nil**
Tracheomalacia	**0**	**Nil**	**1**
Wound infection	**0**	**Nil**	**1**
Others	**Nil**	**1** (Hypothyroidism)	**Nil**

FIGURE - 5

SUBTOTAL THYROIDECTOMY - PATHOLOGICAL DIAGNOSIS

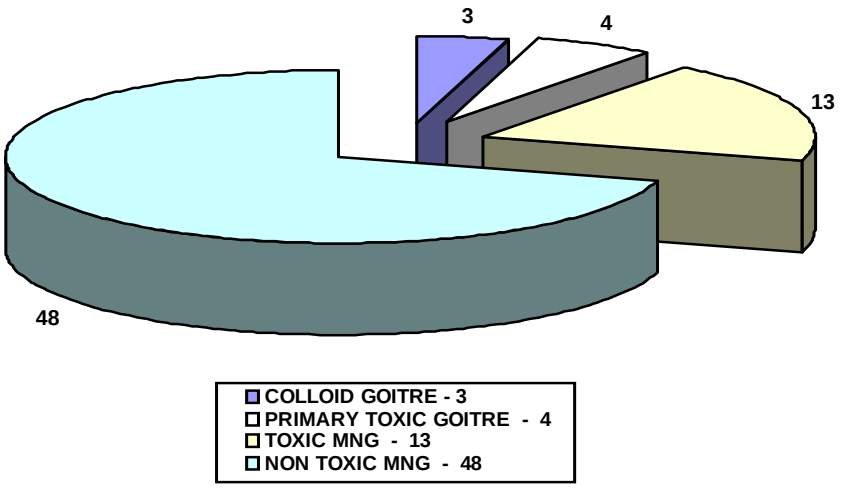
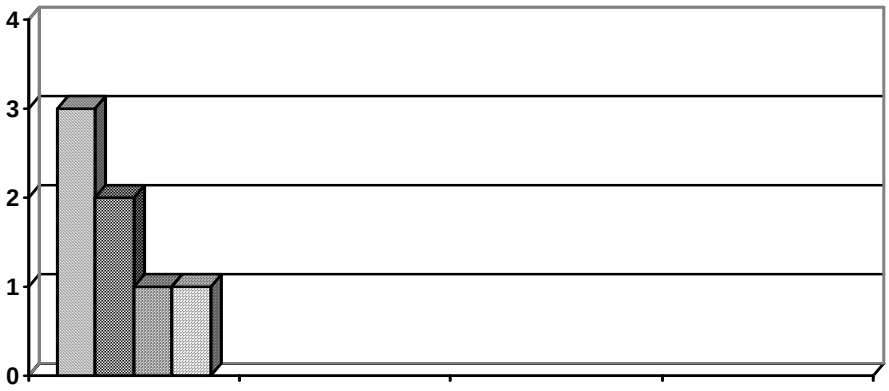


FIGURE - 6

POST OP. COMPLICATIONS AFTER SUBTOTAL THYROIDECTOMIES



Post op hypocalcaemia

Vocal cord palsy

PostOp. haemorrhage

PostOp. hypothyroidism

TABLE - 7

COMPLICATION AFTER HEMI THYROIDECTOMY

COMPLICATION	NUMBER	%
Left vocal cord palsy	1	4
Right vocal cord paralysis	0	-
Tetany	0	-
Other complications	Nil	-

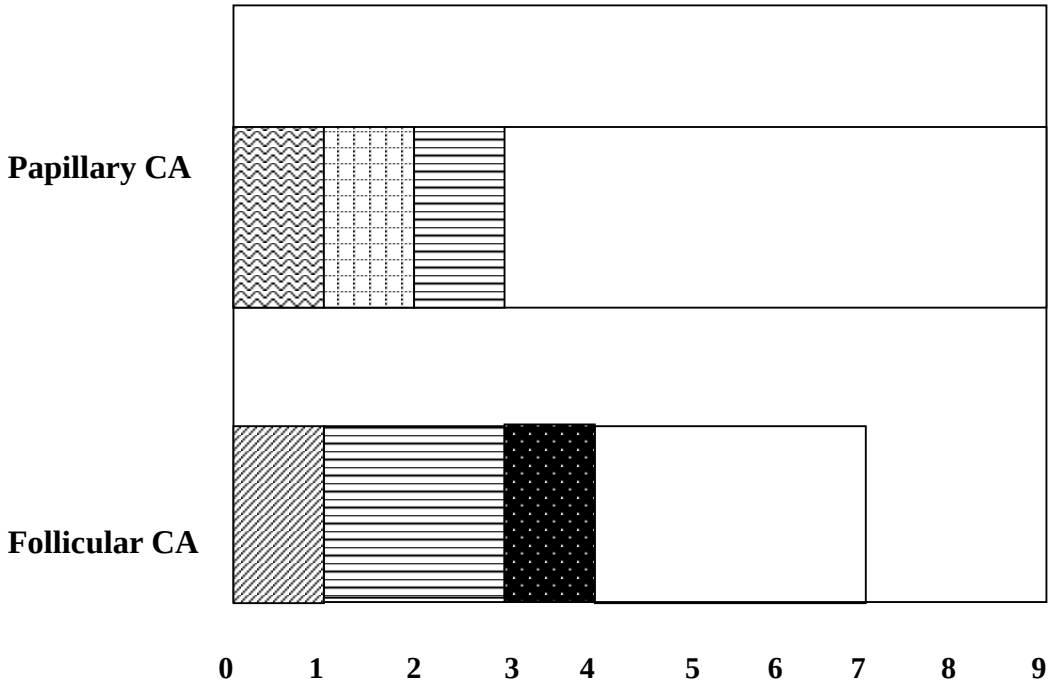
TABLE - 8

COMPLICATIONS AFTER SUBTOTAL THYROIDECTOMIES

COMPLICATION	NUMBER	%
Post op. bleeding	1	1.49
Right vocal cord paralysis	1	1.49
Left vocal cord paralysis	1	1.49
Post op. Hypocalcaemia	3	5.97
Hypothyroidism	1	1.49

FIGURE - 8

COMPLICATION AFTER TOTAL THYROIDECTOMY - PATHOLOGICAL BREAKUP



Tracheomalacia

Wound Infection

Right Vocal cord palsy

Post op. Tetany

Left vocal cord palsy

Normal

TABLE - 9

COMPLICATIONS AFTER TOTAL THYROIDECTOMY

COMPLICATIONS	NUMBER	%
Left vocal cord palsy	**1**	**5.5**
Right vocal cord palsy	**1**	**5.5**
Tracheomalacia	**1**	**5.5**
Postoperative hypocalcaemia	**3**	**16.66**
Wound Infection	**1**	**5.5**

FIGURE - 7

COMPLICATIONS AFTER TOTAL THYROIDECTOMY

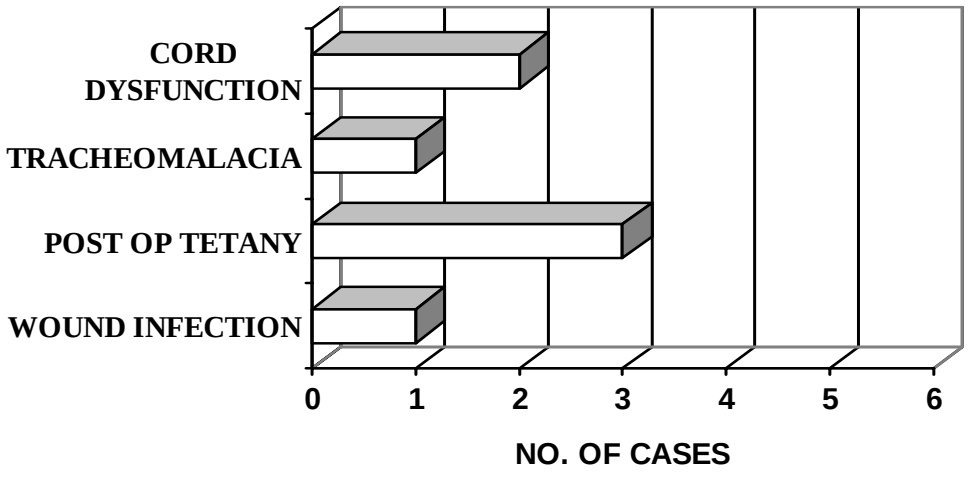


TABLE - 10

UNILATERAL CORD DYSFUNCTION

TYPE OF INJURY	NO. OF CASES
Transient (R) VC Palsy	**2**
Transient (L) VC Palsy	**3**
Total	**5**

FIGURE - 9

UNILATERAL CORD DYSFUNCTION

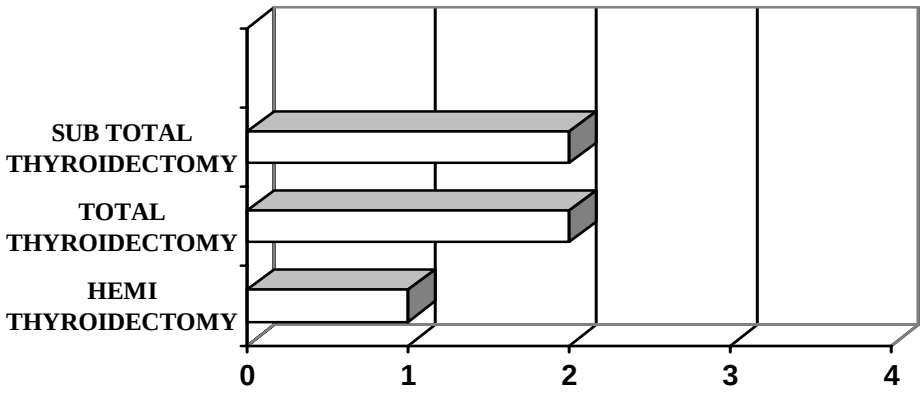


TABLE - 11

POST OP HYPOCALCAEMIA

HYPOCALCAEMIA	NO OF CASES
Permanent	**2**
Temporary	**4**
Total	**6**

FIGURE - 10

HYPOCALCAEMIA

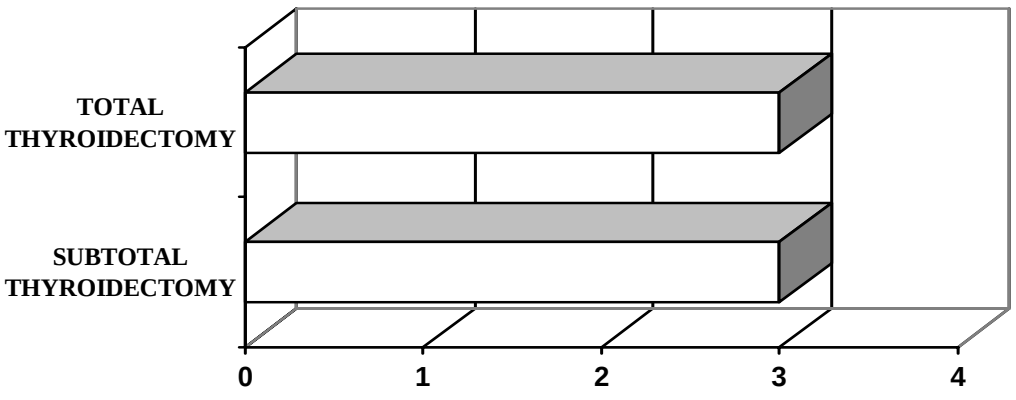


TABLE - 12

POST OPERATIVE HYPO CALCAEMIA

Pathological diagnosis	Type of Surgery	No. of cases	Clinical features	Serum calcium level mg/dl	Symptoms started on
Non - toxic MNG	STT	1	Perioral numbness and tingling	8.7	4th POD
Toxic MNG	STT	2	Carpopedal spasm	9.2, 9.5	1st POD
PCT	TT	2	Carpopedal spasm	8.3, 7.9	3rd POD
FCT	CT	1	Carpopedal spasm	8.1	2nd POD

TABLE - 13

MANAGEMENT OF POSTOP. HYPOCALCAEMIA

Sl. No.	Pathological diagnosis	Types of surgery	Treatment given - duration	
			10 % Cal. Gl. - 10 ml IV	Oral cal.
1.	Non toxic MNG	STT	BD into 3 days	Never turnedup for followup
2.	Toxic MNG	STT	BD into 2 days	5 weeks
3.	Toxic MNG	STT	BD into 2 days	5 weeks
4.	PCT	TT	BD into 5 days	Till date with Vitamin D
5.	PCT	TT	BD into 5 days	Till date with Vitamin D
6.	FCT	CT	BD into 2 days	3 weeks

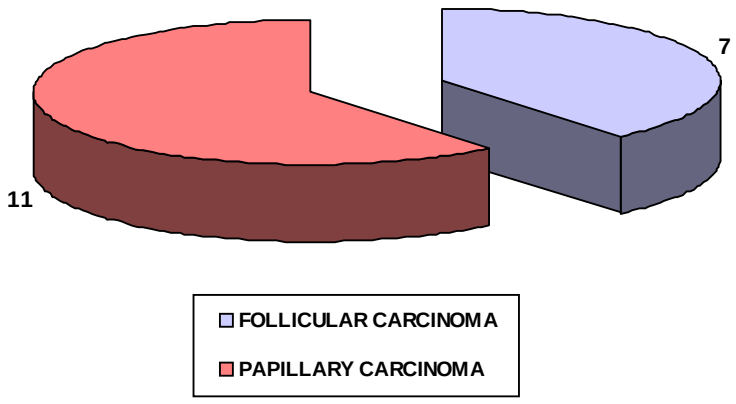
TABLE - 14

RLN INJURY

Pathology	Type of surgery	No. of Cases	Symptoms	Postop. IDL	Treatment given	Duration for complete recovery
Non toxic MNG	STT	1	Hoarseness Of voice, chocking	Right VC sluggish movement	Steroids	Not come for followup
Toxic MNG	STT	1	Hoarseness Of voice, chocking	Left VC sluggish movement	Steroids	4 weeks
SNT	HT	1	Hoarseness Of voice	Left VC sluggish movement	Steroids & Speech therapy	12 weeks
PCT	TT	1	Hoarseness Of voice in effective cough	Right vocal cord paramedian position	Voice rest, steroid & speech therapy	15 weeks
FCT	CT	1	Hoarseness Of voice	Left VC sluggish movement	Steroids & Speech therapy	6 weeks

FIGURE - 11

TOTAL THYROIDECTOMY - PATHOLOGICAL DIAGNOSIS



Out of the 110 cases operated only 5 cases had unilateral cord palsy, 6 cases had tetany, 1 patient had severe bleeding (reopened the wound) one patient developed tracheomalacia and one patient had wound infection with marginal skin necrosis.

COMPLICATION FOLLOWING HEMI THYROIDECTOMY

25 patients underwent hemithyroidectomy. Among them only one patient developed left vocal cord paralysis. (Table 14) The direct Laryngoscopy examination after extubation showed sluggish left vocal cord movements. Patient had no respiratory difficulty, but she had only hoarseness of voice. She was treated with steroids. Speech therapy given for 3 months and her voice improved. IDL examination after 3 months showed bilateral normal vocal cords movements.

COMPLICATION FOLLOWING SUB TOTAL THYROIDECTOMY

67 patients underwent subtotal thyroidectomy for various pathology in the thyroid gland. Among them 1 patient developed post-operative haemorrhage. In the postoperative ward after 3 hours of surgery patient developed some difficulty in breathing but the patient was not restless and there was continuous ooze from the sutured wound. Patient was taken to the operation theatre and wound explored under GA. There was ooze from the superior pedicle which was ligated, and all the haematoma causing respiratory difficulty were evacuated.

Hypocalcemia was diagnosed in 3 of the 67 patients who underwent subtotal thyroidectomy of the 3, 2 cases had carpopedal spasm on the next day of surgery. Both of them were operated for toxic MNG. They were treated with IV 10% calcium gluconate 10ml bd for 2 days and then shifted to oral calcium. There was no evidence of decreased serum calcium level and they did not have any signs and symptoms of tetany from the 4^{th} POD. They were kept under observation for nearly 2

weeks. Oral calcium was continued for 5 weeks and then stopped and till date they are normal.

The other one who had parathyroid insufficiency was a case of non-toxic MNG. She developed only tingling and numbness around the lips on the 4th POD. She was given bd dose of IV 10% Calcium Gluconate for 3 days and then switched over to oral calcium tablet. She got discharged on the 12th day of surgery. Her serum calcium level was 8.7 mg/dl on the 4th POD. 9.2 mg/dl on the 5th POD with inj. Calcium Gluconate and returned to normal on the 6th POD which was 10.6. mg/dl. She never turned up for follow up.

Of the 67 patient who underwent subtotal thyroidectomy, only 2 developed unilateral vocal cord palsy. One was a case of non-toxic MNG who also had postoperative tetany. Both of them had hoarseness of voice and choking. Direct Laryngoscopy examination showed sluggish movement of cord on the right side for one patient and the left side for the other patient after extubation. They were treated with injection decadron 8mg IV bd for 3 days and then the dosage was tappered over the subsequent 6 days. Both of them recovered well and got discharged 2 weeks after surgery.

The one with non-toxic MNG never turned up for follow up. The other patients vocal cords returned to normal position after 5 weeks of surgery.

One patient with non-toxic MNG who underwent STT developed postoperative hypothyroidism after 9 months of surgery. Her thyroid hormones status checked. T3,T4 were suppressed and TSH was elevated. She was started on T. Eltroxin 0.3mg OD and she is on T.Eltroxin till date.

COMPLICATION FOLLOWING TOTAL THYROIDECTOMY

(Table 9) Totally 18 patients underwent total thyroidectomy. Among the 18, 3 underwent unilateral functional lymphnode dissection with TT. One patient underwent completion thyroidectomy following HT.

Of the 18, 2 patients developed unilateral vocal cord palsy. One was a case of PCT (Table - 14) the right vocal cord of that patient was in the paramedian position after extubation. Patient was treated with voice rest, steroids and speech therapy. Patient recovered well with vocal cord return to the normal position on the 15^{th} week of surgery.

The other patient was a case of FCT. He presented with MNG and FNAC which came as follicular neoplasm. STT done. Patient was taken up for completion thyroidectomy on the fourth POD since his HPE report came as FCT. The patient developed neuropraxia of left RLN (Table-14) which was treated with injection methylprednisolone for 2 days, speech therapy was also given patient recovered well with normal vocal cord position on the 6 weeks of surgery.

One of our patients with follicular carcinoma underwent tracheostomy who had a huge tumor which was removed. Tracheostomy done at the end of surgery expecting tracheomalacia since it was a very huge tumor and the patient went home after closure of the tracheostomy and with stabilized trachea after one and a half months.

Two of our patients who underwent total thyroidectomy had post-operative true hypocalcaemia - Table 9,12,13. They were treated with inj. Cal. Gluconate IV

BD for 5 days then shifted to oral calcium and vitamin D. They are still on T.calcium and vitamin D.

One more patient following TT who developed hypocalcaemia with temporary Tetany on the second POD. Serum calcium was 8.1mg/dl. Patient treated with injection calcium gluconate for 2 days. Serum calcium level returned to normal on the fourth POD. Then he was shifted to oral calcium and Vitamin D which was stopped after 3 weeks of surgery. Patient recovered well.

One patient had wound infection with marginal skin necrosis. Necrosed skin excised, higher antibiotics given. The wound healed well.

Discuss

DISCUSSION

This series consisted of 110 patients with various thyroid pathology underwent surgery on the thyroid gland consecutively over a period of 11 months from July 2004 to May 2005.

Most of our cases who underwent thyroidectomy were female patients (95 females, 15 males) in the age group of 20 - 40 years.

Unilateral vocal cord dysfunction and postoperative hypocalcaemia were the major complications encountered in this study.

VOCAL CORD DYSFUNCTION

Unilateral vocal cord dysfunction occurred in 5 of our patients. 2 after subtotal thyroidectomy, 2 after total thyroidectomy and 1 after hemithyroidectomy. All were due to transient recurrent laryngeal nerve palsy and one of which was associated with hypocalcaemia. All were treated with voice rest, steroids and few with speech therapy (Table 14). All were recovered well. One patient did not come for follow up.

According to literature[4,60] transient paralysis occurs in about 3% of nerves at risk and recovers in 3 weeks to 3 months[4,60]. Permanent recurrent laryngeal nerve injury is relatively uncommon event with a rate of approximately 1%[54]. Injury to external branch of superior laryngeal nerve is uncommon (2%)[4,60].

In this study, incidence of recurrent laryngeal nerve paralysis is about 4.54% and it was only transient.

Incidence of recurrent laryngeal nerve palsy (transient) in this study, as follows

4% after Hemi thyroidectomy.

2.98 % after subtotal thyroidectomy.

11.11 % after total thryoidectomy.

HYPOCALCAEMIA

Of the 110 patients, 6 developed postoperative hypocalcaemia 2 cases after subtotal thyroidectomy for toxic MNG, one after STT for non-toxic MNG and 3 for malignancy after total thyroidectomy. They were all treated initially with 10% IV Calcium Gluconate and then shifted to oral Calcium. Of the 6, only 4 had reduced Sr. Calcium level, which returned to normal with treatment (Table - 12,13).

The 2 patients who underwent STT for Toxic MNG and one patient who underwent TT for malignancy were weanedoff oral calcium on the 5^{th} week after surgery. But the 2 patients who underwent TT for malignancy had to continue the drugs till date (>6months) to remain asymptomatic (true hypocalcaemia). The other patient who underwent STT for non-toxic MNG never came for followup.

In this study - incidence of hypocalcaemia

1.81% - Permanent Hypocalcaemia

3.63% - Temporary Hypocalcaemia

According to literature[50,51,25,26] incidence of hypocalcaemia

1-2 % - Permanent Hypocalcaemia

0-50% - Temporary Hypocalcaemia

Incidence of post-operative hypocalcaemia in this study

4.47 % after subtotal thyroidectomy

16.66 % after total thyroidectomy

Review of literature[54,55,56,57,59] showed the incidence is 0.9 to 11% after subtotal thyroidecotmy and 3.2 to 25% after total thyroidectomy.

Hypocalcaemia following STT.

Foster et al	-	0.9 %
Karlon et al	-	2.2 %
Sawyers et al	-	11 %
Present study	-	4.47 %

Hypocalcaemia following TT

Attie et al	-	3.2 %
Thomson et al	-	4.1 %
Block et al	-	4.2 %
Mckenzie et al	-	27 %
Present study	-	16.66 %

Stephan A Falk et al in 1988[61] has shown that post thyroidectomy hypocalcaemia occur, even after careful preservation of parathyroids and their blood supply, since the glands and their vessels are sensitive to ischemia caused by vasospasm due to surgical manipulation. The resultant hypocalcaemia usually is transient and recover later. Considering the technique used in this institution were no determined efforts are done to identify all the 4 parathyroids routinely, a considerable higher incidence of post thyroidectomy hypocalcaemia might be expected, nevertheless the parathyroids are never disturbed by avoiding extensive posterior dissection in most of the cases.

A relatively higher incidence of hypocalcaemia was noted in malignancy and in hyperthyroidism.

Higher incidence in malignancy is directly related to the extensiveness of the surgery in that group, and in hyperthyroidism, previous thyroid over activity is a risk factor[49]. Reasons suggested are

- Hungry bone syndrome
- release of calcitonin during surgical manipulation
- Surgical technical variations.

One of our patients who underwent total thyroidectomy for malignancy developed wound infection & marginal skin necrosis. She needed higher antibiotics and wound debridement. She recovered well. Most of the cases of routine thyroidectomy were given a short course of ampicillin and garamycin parenterally till drain removal and then converted to oral Antibiotics till suture removal.

Of the 110 patients, only one had postoperative haemorrhage and needed wound exploration at operation theatre under anaesthesia.

Only one patient with non-toxic MNG who underwent STT developed hypothyroidism and was started on tablet Eltroxin.

One patient needed tracheostomy for anticipating tracheomalacia .

Out of the study sample, 14 patients developed complications. Among the 14, 3 patients were males and others were females.

Permanent morbidity in the form of hypocalcaemia was noted in two patients, both of whom were operated for malignancy thyroid with extensive dissection.

There was no case of recurrent thyrotoxicosis or thyroid storm encountered in this study.

There was no mortality attributed to thyroid surgery in our study.

Do's and Dont's followed in

DO'S AND DONT'S FOLLOWED IN THIS INSTITUTION

DO'S :

1. Wound complications can be prevented by clean dissection, securing perfect haemostasis.

2. Judicious use of diathermy during surgery for ensuring haemostasis.

3. Identify the recurrent laryngeal nerve routinely for all cases especially for a suspected malignant disease and while doing total thyroidectomy.

4. Identify parathyroid glands and preserve the blood supply to the parathyroid glands by ligating the branches of the inferior thyroid artery, individually after the branch supplying the parathyroid has taken off from the inferior thyroid artery.

5. Care should be taken to avoid injuring the recurrent laryngeal nerve when dividing the ligament of Berry.

6. Use of drain may decrease the incidence of haematoma formation and respiratory compromise in the immediate postoperative period.

7. Good preoperative preparation of thyrotoxic patients rendering them euthyroid greatly reduces the morbidity.

DONT'S

1. Avoid using diathermy while dissecting the flaps and while dissection near the recurrent laryngeal nerve.
2. Mass ligation of superior pole should not be done.
3. Mass ligation of inferior pole veins should not be done.
4. Don't take up the patient for surgery when the thyrotoxic state is not well controlled.

Conclusion

CONCLUSION

In this study, majority of the complication were due to transient unilateral RLN palsy (4.54%) and hypoparathyroidism (5.45%).

The other complications were postop. haemorrhage wound infection, tracheomalacia and hypothyroidism each (0.90%).

For majority of the patients the postoperative periods went uneventful (87.27).

This study has confirmed the low morbidity rates after surgery, on the thyroid gland, in the present context largely due to extensive work on the thyroid gland with greater understanding of anatomical and physiological details of the gland.

Surgery for malignant disease of the thyroid requires more meticulous dissection than that required for benign conditions. Surgery for toxic goiter both primary thyrotoxicosis and secondary thyrotoxicosis has become a safe procedure with good preoperative control of the toxic, hyperthyroid state.

Identification of RLN and parathyroid glands are must to reduce the morbidity associated with their injury.

Mass ligation of superior pole and mass ligation of lower pole veins increases the chances of morbidity.

Surgery on the thyroid gland has come a long way from being a hazardous surgical procedure with high morbidity and mortality to a safe surgical procedure.

Profor

PROFORMA

Serial No:

Name of the patient

Age: Sex: Unit: IP No:

Address:

Date of admission:

Date of surgery:

Date of discharge:

No. of days in Hospital: Pre-operative: Pose-operative:

Clinical Presentation:

Swelling/Pain/Hoarseness/Others Duration:

Size of the swelling

Type of swelling - Nodule / Generalized thyroid enlargement

Clinical features of hyper/hypo function

Any other systemic illness

Biochemical hormonal status - T3, T4, TSH

Preoperative haemogram

FNAC

Histopathology:

Anesthesia - Local / General

Difficulties in intubation:

Type of surgery:

Duration of surgery:

Total duration of anesthesia + surgery

Any excessive bleeding during surgery

Transfusion needed

Recurrent laryngeal nerve identified / Not

Parathyroids identified - One/Two/Three/All

Methylene blue study

Inferior thyroid artery ligated/Not

Thyroid extraction: Marker/Clamp method

Any evidence of parathyroids in the thyroidectomy specimen

Reimplatation of parathyroids done/Not

Drain

After extubation - Direct laryngoscope-position of vocal cords.

Post-operative aspiration from drain

Any immediate post - operative complication

- Prolonged recovery/Hematoma
- Dyspnea/Pneumothorax
- Thyroid crisis/Hoarseness
- Tracheal or Esophageal perforation

Post-operative infection

Antibiotics used

Clinical features of hypocalcemia in post-operative period:

Time of onset:

Initial presentation:

Bibliogra

BIBLIOGRAPHY

1. Schwartz - Principles of surgery 8th edition 1395-1429.
2. Sir Alfred Cushierri - Essential surgical practice - 4th edition 95 - 116.
3. Lee Mc Gregor's synopsis of Surgical Anatomy 12th edition 198-205.
4. Bailey & Love's short practice of surgery 24th edition 776-804.
5. Grays Anatomy 37th edition 1459-1463.
6. Robert J. Baker& Josef E. fisher: Mastery of surgery 4th edition 477-511.
7. Sturniolo G,D'Alia C, Tonante A: The recurrent laryngeal nerve related to thyroid surgery. Am J Surg 1999 Jun;177(6): 485-8[Medline].
8. Kierner AC, Aigner M, Burian M: The external branch of the superior laryngeal nerve: its topographical anatomy as related to surgery of the neck. Arch Otolaryngol Head Neck Surg 1998 Mar;124(3):301-3[Medline].
9. Wang C: The anatomic basis of parathyroid surgery. Ann Surg 1976 Mar; 183(3): 271-5[Medline].
10. Wheeler MH: Thyroid surgery and the recurrent laryngeal nerve. Br J Surg 1999 Mar; 86(3):291-2[Medline].
11. Kopp P. Pendred's syndrome and genetic defects in thyroid hormone synthesis Rev. Endoct Metabolic disorder 1: 109,2000
12. Yoshider. A, Taniquehi. S, Hisatome I, et al; Pendrin is an iodide - specific apical porter responsible for iodide efflux from thyroid cells, Iclin Endernal Hesab 87:3356, 2002
13. Farquharson's textbook of Operative Surgery 9th edition.

14. Netterville JL, Aly A, Ossoff RH: Evaluation and treatment of complications of thyroid and parathyroid surgery. Otolaryngol Clin Am 1990 Jun;23(3); 529-52[Medline].

15. Lore Jn. An atlas of Head and Neck Surgery, 3rd, WE Sauders Company, 19 EY; Chap - B.

16. Johnson JT, Wagner RL: Infection following uncontaminated head and neck surgery. Arch Otolaryngol Head Neck Surg 1987 Apr;113(4):368-9 [Medline].

17. Dr Anjali P. Sarargaonkar: Post Thyroidectomy Haematoma causing total airway obstruction. Indian J Anaesth 2004; 48(6): 483-485.

18. Farrar WB: Complications of thyroidectomy. Surg Clin North Am 1983 Dec; 63(6): 1353-61[Medline].

19. Schoretsanitis G. Melissas J, Sanidas E: Does draining the neck affect morbidity following thyroid surgery? Am Surg 1998 Aug; 64(8): 778-80 [Medline].

20. Debry C, Renou G, Fingerhut A: Drainage after thyroid surgery: a prospective randomised study. J Laryngol Otol 1999 Jan; 113(1): 49-51 [Medline].

21. Flint PW, Downs DH, Coltrera MD: Laryngeal synkinesis following reinnervation in the rat. Neuroanatomic and physiologic study using retrograde fluorescent tracers and electromyography. Ann Otal Rhinol Laryngol 1991 Oct; 100(10):797-806[Medline].

22. Parnes SM, Satya-Murti S: Predictive value of laryngeal electromyography in patients with vocal cord paralysis of neurogenic origin. Laringoscope 1985 Nov; 95(11): 1323-6 [Medline].

23. Cannon CR: The anomaly of nonrecurrent laryngeal nerve: identification and management. Otolaryngol Head Neck Surg 1999 May;120(5): 769-71[Medline].

24. Jatzko GR, Lisborg PH, Muller MG, Wette VM: Recurrent nerve palsy after thyroid operations-principal nerve identification and a literature review. Surgery 1994 Feb; 115(2):139-44[Medline].

25. Gavilan J, Gavilan C: Recurrent laryngeal nerve. Identification during thyroid and parathyroid surgery. Arch otolaryngol Head Neck Surg 1986 Dec;112(12):1286-8[Medline].

26. Wong C: The use of the inferior cornu of the thyroid cartilage in identifying the recurrentlaryngeal nerve. Surg Gynecol Obstet 1975 Jan;140(1):91-4[Medline].

27. Eisele DW: Intraoperative electrophysiologic monitoring of the recurrent laryngeal nerve. Laryngoscope 1996 Apr;106(4): 443-9[Medline].

28. Lipton RJ, McCaffrey TV, Litchy WJ: Intraoperative electrophysiologic monitoring of laryngeal muscle during thyroid surgery. Laryngoscope 1988 Dec;98(12):1292-6[Medline].

29. Rice DH, Cone-Wesson B: Intraoperative recurrent laryngeal nerve monitoring. Otolaryngol Head Neck Surg 1991 Sep;105(3):372-5[Medline].

30. Goding GS Jr: Nerve-muscle pedicle reinnervation of the paralyzed vocal cord. Otolaryngol clin North Am 1991 Oct;24(5):1239-52[Medline].

31. Jacobs IN, Sanders I, Wu BL, Biller HF: Rennervation of the canine posterior cricoarytenoid muscle with sympathetic preganglionic neurons. Ann Otol Rhino Laryngol 1990 Mar;99(3 Pt 1): 167-74[Medline].

32. Baldissera F, Cantarella G, Marini G, et al: recovery of inspiratory abduction of the paralyzed vocal cords after bilateral reinnervation of the cricoarytenoid muscles by the single branch of the phrenic nerve. Laryngoscope 1989 Dec; 99(12):1286-92[Medline]

33. Baldissera F, Tredici G, Marini G, et al: Innervation of the paralyzed laryngeal muscles by phrenic motoneurons. A quantitative study by light and electron microscopy. Laryngoscope 1992 Aug; 102(8): 907-16[Medline].

34. Crumly RL: Experiments in laryngeal reinnervation. Laryngoscope 1982 Sep;92(9 Pt2 Suppl 30): 1-27[Medline].

35. Tucker HM: Long-term results of nerve-muscle pedicle reinnervation for laryngeal paralysis. Ann Otol Rhino Laryngol 1989 Sep;98(9): 674-6 [Medline].

36. Pattou F, Combemale F, Fabre S: Hypocalcaemia following thyroid surgery: incidence and prediction of outcome. World J Surg 1998 Jul; 22(7): 718-24 [Medline].

37. Wingert DJ, Friesen SR, Iliopoulos JI: Post-thyroidectomy hypocalcemia. Incidence and risk factors. Am J Surg 1986 Dec; 152(6) 606-10[Medline].

38. Schwartz AE, Friedman EW: Preservation of the parathyroid glands in total thyroidectomy. Surgery Gynecol Obstet 1987 Oct;165(4): 327-32[Medline].

39. Shaha AR, Jaffe BM: Parathyroid preservation during thyroid surgery. Am J Otolaryngol 1998 Mar-Apr; 19(2):113-7[Medline].

40. Lore JM Jr, Kokocharov SI, Kaufman S: Thirty-eight-year evaluation of a surgical technique to protect the external branch of the superior laryngeal nerve during thyroidectomy. Ann Otol Rhinol Laryngol 1998 Dec; 107(12): 1015-22[Medline].
41. Teitelbaum BJ, Wenig BL: Superior laryngeal nerve injury from thyroid surgery. Head Neck 1995 Jan-Feb;17(1): 36-40[Medline].
42. Kasemsuwan L, Nubthuenetr S: Recurrent laryngeal nerve paralysis: a complication of thyroidectomy. J Otolaryngol 1997 Dec;26(6):365-7 [Medline].
43. John L Cameron, Current Surgical Therapy 8th edition 580-602.
44. Lando MJ, Hoover LA, Zukerbraun L: Surgical strategy in thyroid disease. Arch Otolaryngol Head Neck Surg 1990 Dec;116(12): 1378-83[Medline].
45. Shindo ML: Considerations in surgery of the thyroid gland. Otolaryngol Clin North Am 1996 Aug;29(4):629-35[Medline].
46. Respiratory complications after thyroidectomy and the need for tracheostomy in patients with a large goitre. Volume 86, Issue 1 (1 January 1996). Annals of Surgery. 240(1): 18-25, July 2004.
47. Yeager RM, kremeatz ET. Toluderi blue in identification of parathyroids at operation. Ann. Surg. 1969; 829-837.
48. Filho JG, Kowalski LP: Postoperative complications of thyroidectomy for differentiated thyroid carcinoma. Am J Otolaryngol. 2004 Jul-Aug; 25(4): 225-30 [Medline].
49. Michie K. Duncan T et al: Mechanism of Hypocalcaemia after 508-513.

50. Schroder DM, Chambors A, France CJ: Operative strategy for thyroid cancer. Is total thyroidectomy worth the price? Cancer 1986 Nov 15;58(10):2320-8 [Medline].

51. Zheng H, Zhou S, Chen S, et al: An experimental comparison of different kinds of layngeal muscle reinnervation. Otolaryngol Head Neck Surg 1998 Nov; 119(5): 540-7 [Midline]

52. Van der Sluis RF, Wobbes T: Total thyroidectomy: the treatment of choice in differentiated thyroid carcinoma? Eur J Surg Oncol 1985 Dec; 11(4):343-6[Medline].

53. Chooison NW, Hamers JX, Complication of total thyroidectomy for carcinoma surg. Gyn. Obst. 1970;

54. Foster R Jr et al: Morbidity and mortality after thyroidectomy. Surg. Lyn.Obst. 1978; 146:423-429.

55. Attie JN. Khafil Pm. Preservation of Parathyroids during total thyroidectomy. Am. Jn. Surgery 197; 130; 399-404.

56. Block M. Muller JM. Horn nc. Minimizing hypoparathyroidism after extended thyroid operations. Surg. Gyn. Obs.1966; 123; 501-509.

57. Mc Kenzie Ad: The natural history of thryoid cancer Arch. Surgery. 1971; Lo2r-277

58. Sabiston Text book of surgery 16th edition 603-628.

59. Thomsan Nli: Olsen Ken, Hoffman GL. The continuing development of the technique of thyroidectomy. Surgery 1972;913.

60. Barraclough BH, Reeve Ts: Postoperative complications of thyroidectomy: a comparison of two series at an interval of ten years. Related Articles, Links Aust N Z J Surg:1975 Feb;45(1):21-9[Medline].

61. Faik S. Birken En Baran Dt: Temporary post thyroidectomy hypocalcaemia Arch, Otolaryngol. Head neck surgery, 1986; 114; 168-174.

Master

LIST OF ABBREVIATION

B	-	Benign
BB	-	Beta Blockers
Cal. Gl.	-	Calcium Gluconate
CAR	-	Carbimazole
CT	-	Completion Thyroidectomy
CF	-	Clinical Features
DL	-	Direct Laryngoscope
EMG	-	Electromyography
FNAC	-	Fine Needle Aspiration Cytology
GA	-	General Anaesthesia
HT	-	Hemi Thyroidectomy
IDL	-	Indirect Laryngoscope
Inj	-	Injection
IV	-	Intra Venous
LI	-	Lugols Iodine
MNG	-	Multi Nodular Goiter
N	-	Normal
OT	-	Operation Theatre
PCT	-	Papillary Carcinoma Thyroid
POD	-	Post Operative Day
Post Op.	-	Post Operative
PTH	-	Para Thyroid Hormone
RLN	-	Recurrent Laryngeal Nerve
SLN	-	Superior Laryngeal Nerve
SNT	-	Solitary Nodular Thyriod
Sr.	-	Serum
STT	-	Subtotal Thyroidectomy
TCT	-	Follicular Carcinoma Thyroid
TT	-	Total Thyroidectomy
VC	-	Vocal Cord

MASTER CHART

Sl. No.	NAME	Age	Sex	IP.No.	CLINICAL DIAGNOSIS	FNAC	TFT ON DIAGNOSIS	PRE OP. TREAT MENT	SURGERY	BLOOD TRANS FUSION	HPE
1.	Pappathy	35	F	19208	Non toxic MNG	B	N	-	STT	-	B
2.	Jothy	38	F	21324	Non toxic MNG	B	N	-	STT	-	B
3.	Jeyalakshmi	42	F	21453	Non toxic MNG	B	N	-	STT	-	B
4.	Sumathy	28	F	22711	MNG	B	N	-	STT	-	B
5.	Nataraj	74	M	21667	Solitary nodule (R)	B	N	-	(R) Hemi thyroidectomy	-	B
6.	Vasantha	21	F	25482	Non toxic MNG	B	N	-	STT	+	B
7.	Thaileswari	34	F	25370	Non toxic MNG	B	N	-	STT	-	B
8.	Mohana	40	F	26175	Non toxic MNG	B	N	-	STT	-	B
9.	Sarasemmal	40	F	26534	SNT (L)	B	N	-	(L) HT	-	B
10.	Vasantha	32	F	27471	Toxic MNG	B	T3 , T4 ↑ TSH ↓	CAR, BB, LI	STT	+	B
11.	Beetantra	43	F	26505	Non toxic MNG	B	N	-	STT	-	B
12.	Kavitha	22	F	30235	SNT (R)	B	N	-	(R) Hemi thyroidectomy	-	B
13.	Rahima	42	F	29553	Toxic goitre	B	T3 , T4 ↑ TSH ↓	CAR, BB, LI	STT	-	B
14.	Kandasamy	55	M	29797	MNG	B	N	-	STT	-	B
15.	Chellammal	54	F	31120	SNT (L)	B	N	-	(L) HT	-	B
16.	Amudha	30	F	29358	MNG	B	N	-	STT	-	B
17.	Valliammal	61	F	29924	MNG	Follicular neoplasm	N	-	TT	+	FCT
18.	Rajammal	25	F	29951	Toxic MNG	B	T3 , T4 ↑ TSH ↓	CAR, BB, LI	STT	-	B
19.	Lucy	30	F	32143	MNG	B	N	-	STT	-	B
20.	Shanthy	33	F	34448	MNG	B	N	-	STT	-	B
21.	Rajeswari	22	F	34599	SNT	B	N	-	HT	-	B

22.	Ruckmani	30	F	35290	MNG	B	N	-	STT	-	B	
23.	Ruckmani	40	F	34843	SNT	B	N	-	HT	-	B	
24.	Maduri	45	F	34796	SNT	B	N	-	HT	-	B	
25.	Sarani	50	F	36072	SNT	B	N	-	HT	-	B	
26.	Nirmala	25	F	35009	Toxic MNG	B	T3 , T4 ↑ TSH ↓	CAR, BB, LI	STT	+	B	
27.	Sivagama sundari	41	F	34655	Toxic MNG	B	T3 , T4 ↑ TSH ↓	CAR, BB, LI	STT	+	B	
28.	Meenakshi	53	F	35917	MNG	B	N	-	STT	-	B	
29.	Lakshmi	52	F	13277	MNG	Follicular Neoplasm	N	-	TT	+	FCT	
30.	Malika begam	46	F	41929	MNG	B	N	-	STT	-	B	
31.	Arukani	27	F	43619	MNG	B	N	-	STT	-	B	
32.	Balaji	22	M	42284	Toxic MNG	B	T3 , T4 ↑ TSH ↓	CAR, BB, LI	STT	+	B	
33.	Amirtham	35	F	45064	MNG	B	N	-	STT	-	B	
34.	Gurumurthy	70	M	59648	SNT	PCT	N	-	TT	-	PCT	
35.	Sowmya	16	F	9120	MNG with IIº neck - right	PCT	N	-	TT with FND on (R) side	-	PCT	
36.	Rejena mary	35	F	46647	MNG	B	N	-	STT	-	B	
37.	Eswari	42	F	49484	SNT	B	N	-	HT	-	B	
38.	Reeta	30	F	49686	SNT	B	N	-	HT	-	B	
39.	Nagarathinam	45	F	50709	SNT	B	N	-	HT	-	B	
40.	Kaliyammal	55	F	50438	MNG	B	N	-	STT	-	B	
41.	Syed Mohammed	48	M	50554	MNG	B	N	-	STT	-	B	
42.	Omana	26	F	57528	MNG	B	N	-	STT	-	B	
43.	Saroja	35	F	47051	MNG	B	N	-	STT	-	B	
44.	Panneer selvam	31	M	21165	Toxic MNG	B	T3 , T4 ↑ TSH ↓	CAR, BB, LI	STT	+	B	
45.	Rizwan	22	M	20749	Graves	B	T3 , T4 ↑ TSH ↓	CAR, BB, LI	STT	+	B	

46.	Ranganathan	16	M	48280	MNG	B	N	-	STT	-	B	
47.	Thangamani	28	F	50856	Graves discure	B	T3 , T4 ↑ TSH ↓	CAR, BB, LI	STT	+	B	
48.	Indirani	45	F	54218	MNG	B	N	-	STT	-	B	
49.	Muthammal	54	F	57764	SNT	B	N	-	HT	-	B	
50.	Sivagami	27	F	58771	SNT	B	N	-	HT	-	B	
51.	Thulasiammal	45	F	57045	MNG	B	N	-	STT	-	B	
52.	Chella kannan	53	M	52117	MNG	PCT	N	-	TT	-	PCT	
53.	Susheela	30	F	59162	Non Toxic MNG	B	N	-	STT	-	B	
54.	Subbammal	35	F	61401	MNG	B	N	-	STT	-	B	
55.	Nagajothy	25	F	60327	SNT	B	N	-	HT	-	B	
56.	Abirami	27	F	63199	SNT	B	N	-	HT	-	B	
57.	Muniyammal	45	F	59377	MNG	B	N	-	STT	-	B	
58.	Jegan	48	M	14636	MNG	Follicular neoplasm	N	-	CT	-	FCT	
59.	Lakshmi	35	F	63245	SNT	B	N	-	HT	-	B	
60.	Karpagam	30	F	64586	SNT	B	N	-	HT	-	B	
61.	Sivagami	20	F	65369	MNG	B	N	-	STT	-	B	
62.	Uma	30	F	64723	SNT with II^{o} neck Left	PCT	N	-	TT with FND Left side	-	PCT	
63.	Kaliammal	40	F	65211	MNG	B	N	-	STT	-	B	
64.	Kannappan	45	M	247	MNG	PCT	N	-	TT with FND (R) side	-	PCT	
65.	Shanmugavel	14	M	1656	MNG	B	N	-	STT	-	B	
66.	Kavitha	23	F	1189	MNG	B	N	-	STT	-	B	
67.	Muthusamy	35	M	1665	MNG	B	N	-	STT	-	B	
68.	Revathy	42	F	38840	SNT	B	N	-	HT	+	B	
69.	Selvi	21	F	3577	Toxic MNG	B	T3 , T4 ↑ TSH ↓	CAR, BB, LI	STT	-	B	

70.	Palaniammal	50	F	1175	Toxic MNG	B	T3 , T4 ↑ TSH ↓	CAR, BB, LI	STT	+	B	
71.	Vijayalakshmi	21	F	5536	SNT	B	N	-	HT	-	B	
72.	Mani	42	F	4557	MNG	B	N	-	STT	-	B	
73.	Jaya	36	F	5546	MNG	B	N	-	STT	-	B	
74.	Revathy	42	F	10687	MNG	B	N	-	STT	-	B	
75.	Kaliammal	40	F	14110	Colloid Goitre	B	N	-	STT	-	B	
76.	Pushpa	45	F	14955	MNG	B	N	-	STT	-	B	
77.	Lakshmi	40	F	15345	Toxic MNG	B	T3 , T4 ↑ TSH ↓	CAR, BB, LI	STT	+	B	
78.	Karuppathal	65	F	16938	MNG	B	N	-	STT	-	B	
79.	Subhulakshmi	68	F	17734	MNG	Follicular neoplasm	N	-	TT	+	FCT	
80.	Vellammal	40	F	16721	MNG	B	N	-	STT	-	B	
81.	Melathal	50	F	17716	SNT	B	N	-	STT	-	B	
82.	Subbal	65	F	18618	FCT	Follicular neoplasm	N	-	TT	+	FCT	
83.	Gandimathy	50	F	15400	MNG	B	N	-	STT	-	B	
84.	Ruckmani	30	F	15353	MNG	B	N	-	STT	-	B	
85.	Parvathy	32	F	17762	SNT	B	N	-	HT	-	B	
86.	Krishnasamy	75	M	20731	CA Thyroid	Follicular neoplasm	N	-	TT	+	FCT	
87.	Parvathy	37	F	21822	Graves disease	B	T3 , T4 ↑ TSH ↓	CAR, BB, LI	STT	+	B	
88.	Krishnaveni	37	F	21794	Colloid Goitre	B	N	-	STT	-	B	
89.	Varadhamani	30	F	23189	SNT	B	N	-	HT	-	B	
90.	Fathima	18	F	28922	MNG	B	N	-	STT	-	B	
91.	Fathima	37	F	24876	MNG	B	N	-	STT	-	B	
92.	Prema	28	F	48100	CA thyroid	PCT	N	-	TT	+	PCT	
93.	Basker	22	M	23350	Pap ca thyroid	B	N	-	TT with berry picking of lymp nodes	-	B	

94.	Balamani	40	F	26080	MNG	B	N	-	STT	-	B	
95.	Valupooran	37	M	26242	Toxic MNG	B	T3 , T4 ↑ TSH ↓	CAR, BB, LI	STT	-	B	
96.	Thangamani	35	F	26273	MNG	B	N	-	STT	-	B	
97.	Kalamani	26	F	27405	SNT	B	N	-	HT	-	B	
98.	Rajammal	38	F	20545	Toxic MNG	B	T3 , T4 ↑ TSH ↓	CAR, BB, LI	STT	+	B	
99.	Kaliammal	45	F	27434	SNT	B	N	-	HT	-	B	
100.	Kamatchi	15	F	31592	SNT	B	N	-	HT	-	B	
101.	Devi	26	F	32904	MNG	B	N	-	STT	-	B	
102.	Aye11sha	36	F	31778	MNG	B	N	-	STT	-	B	
103.	Jakkummal	47	F	21363	Colloid Goitre	B	N	-	HT	-	B	
104.	Ammasai	46	F	41342	MNG	Follicular neoplasm	N	-	TT	-	FCT	
105.	Rajammal	38	F	61596	Toxic MNG	B	T3 , T4 ↑ TSH ↓	CAR, BB, LI	STT	+	B	
106.	Pushpavathy	36	F	3010	Toxic MNG	B	T3 , T4 ↑ TSH ↓	CAR, BB, LI	STT	+	B	
107.	Mehraj	28	F	25789	SNT	PCT	N	-	TT	-	PCT	
108.	Ranganayaki	53	F	23803	CA Thyroid	PCT	N	-	TT	-	PCT	
109.	Sundari	23	F	25946	MNG	PCT	N	-	TT	-	PCT	
110	Vasantha	48	F	31042	MNG	PCT	N	-	TT	+	PCT	