Under the aegis of
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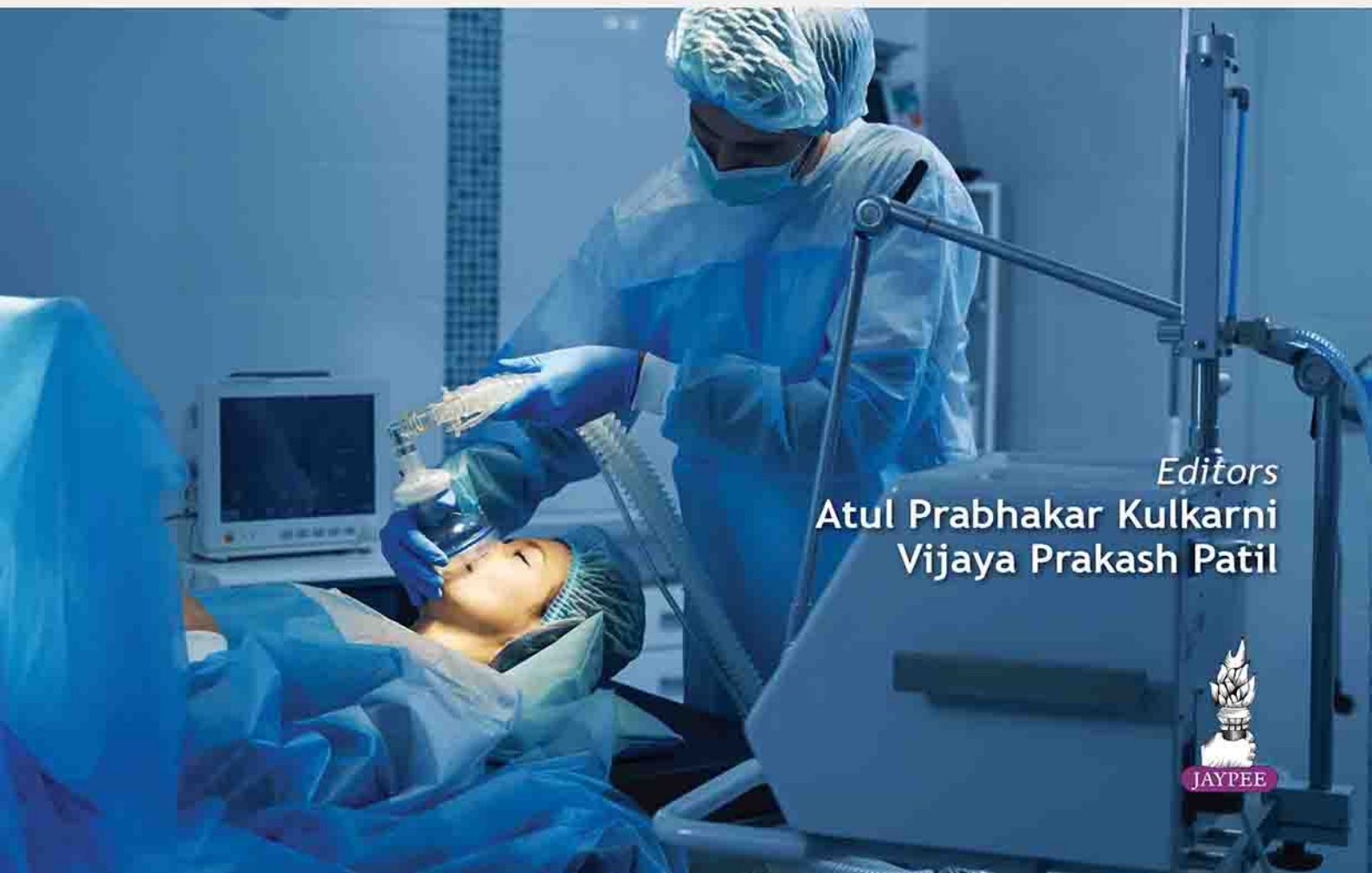
OBJECTIVE ANESTHESIA REVIEW

A Comprehensive Textbook for the Examinees



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Section 1 Case Discussions

CHAPTER

33 Large Thyroid Mass

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A 54-year-old female patient presents with a large thyroid swelling. She has no comorbidities. She is scheduled to undergo thyroidectomy. We discuss the perioperative anesthesia management.

The focus of this case is airway management and management of hypo-/hyperthyroidism.

What specific things are you seeking in the history and examination of a patient with thyroid swelling?

Ans.

Size and duration of swelling:

- *Pressure effects on the trachea:* Dyspnea and stridor (and effects of change in position)
- *Pressure effects on the esophagus:* Dysphagia
- *Pressure effects on the recurrent laryngeal nerve (RLN):* Hoarseness
- *Duration:* Long-standing large thyroid may be associated with tracheomalacia.

Retrosternal extension:

- Dysphagia and dyspnea
- Unable to "get under the swelling"
- Superior vena cava (SVC) obstruction/thrombosis—dilated veins in the neck and upper part of the chest, facial edema.

Intratracheal extension:

- Dyspnea
- Hemoptysis

Thyroid hormone status:

- Signs and symptoms of hypo- or hyperthyroidism

Associated endocrine disorders:

- *Multiple endocrine neoplasia (MEN) type 2A:* Medullary thyroid carcinoma, pheochromocytoma, and parathyroid hyperplasia
- *MEN type 2B:* Medullary thyroid cancer, pheochromocytoma, and growths around nerves (neuromas)
- *MEN I:* Pituitary adenoma, parathyroid hyperplasia, pancreatic tumor-like insulinoma, and rarely thyroid and adrenal involvement.

Thyroid malignancies:

- Usually present as thyroid nodules and are usually metabolically inactive
- *Common types:* Papillary, follicular, medullary, and anaplastic
- Intratracheal extension may present as dyspnea and hemoptysis
- Distant metastasis to the lung, bones, and liver may be present, especially in the follicular type.

How would you present your diagnosis?

Ans. A 54-year-old female with size/side (e.g., large thyroid swelling/4 × 3 cm swelling on right) with/without airway compromise (e.g., with tracheal deviation and compression) with/without complications (e.g., retrosternal extension without SVC syndrome or compression of esophagus/RLN) with normal thyroid function (or controlled/uncontrolled hypothyroidism/hyperthyroidism) with/without comorbidities (e.g., well-controlled diabetes mellitus) is scheduled for subtotal thyroidectomy.

How are thyroid hormones synthesized in the body?

Ans. In the thyroid gland, active uptake of iodide occurs in exchange for Na^+. Iodide uptake is stimulated by thyroid-stimulating hormone (TSH). Iodide is oxidated to active iodine by hydrogen peroxide (H_2O_2). The reaction is catalyzed by thyroid peroxidase (TPO). Iodine is actively transported across the apical surface of the follicular cell. Active iodine is incorporated into the tyrosine residues of thyroglobulin molecules to form mono- and di-iodotyrosines (MIT and DIT).

Thyroglobulin is taken up into the colloid of the follicle where a coupling reaction between pairs of iodinated tyrosine molecules occurs. The coupling of two DIT produces tetraiodothyronine or thyroxine (T_4) while the

combination of DIT with MIT produces triiodothyronine (T_3). This reaction is catalyzed by TPO. Thyroid hormones are stored in this state and are only released when the thyroglobulin molecule is taken back up into the follicular cells. Thyroglobulin droplets are taken up by the follicular cells by the process of pinocytosis which is stimulated by TSH. Fusion of the droplets with lysosomes results in hydrolysis of the thyroglobulin molecules and the release of T_4 and T_3. Approximately, 100 μg of thyroid hormones are secreted from the gland each day, with around 90% in the form of T_4 and the remainder as T_3. T_4 undergoes peripheral conversion to T_3 (T_3 is three to five times more active than T_4) in the liver and kidney or to reverse T_3 (little or no biological activity). Glucocorticoids, propranolol, and amiodarone block conversion of T_4 to T_3.

About 99.98% of T_4 and 99.8% of T_3 is bound. It is the free fraction which is responsible for the actions and the negative feedback to the pituitary gland.

T_3 and T_4 regulation: The production of T_4 and T_3 is regulated by TSH released by the anterior pituitary, which is in turn released as a result of thyrotropin-releasing hormone (TRH) released by the hypothalamus. The thyroid and thyrotropes form a negative feedback loop. TSH production is suppressed when the free T_4 levels are high and vice versa.

What are the actions of thyroid hormone?

Ans. Thyroid hormones act on almost all the cells of the body with effects on growth and development. Many of the actions of thyroid hormones are mediated by their binding to nuclear receptors that have a preferential affinity for T_3. T_3 receptors are steroid hormone receptors which regulate the expression of genes coding for enzymes which regulate cell function.

Central nervous system (CNS): Effect on neuronal function and reflexes. The reaction time of stretch reflex is shortened in hyperthyroidism. The thyroid hormone also affects the reticular activating system.

Cardiovascular system (CVS): Actions of thyroid hormones and catecholamines are interrelated. Thyroid hormones have actions similar to epinephrine in stimulating the cardiovascular system, nervous system, and metabolic rate. Thyroid hormones increase the number and affinity of β-adrenergic receptors, thus increasing their sensitivity to catecholamines causing increased cardiac output and tachycardia.

Metabolism: Thyroid hormones increase the metabolic rate and oxygen consumption of most tissues. They also increase carbohydrate absorption from the gut, increase protein catabolism, mobilize fatty acids, and lower serum cholesterol.

Growth: Thyroid hormones are important for brain development and skeletal maturation.

How will you clinically assess the thyroid function status in the patient? What are the signs and symptoms of hypo- and hyperthyroidism? State eye signs in hyperthyroidism.

Ans. *History of taking any medicines*—duration and dose, previous thyroid function test (TFT).

Symptoms and Signs of Hyperthyroidism

Hyperactivity, weight loss, and tremor (tested in outstretched hands and tongue) are classical symptoms. Other findings are warm moist skin, diarrhea, intolerance to heat, weakness in large muscle groups menstrual abnormalities, and osteopenia.

Common cardiac findings are tachycardia (especially a high sleeping pulse rate), dysrhythmia (atrial fibrillation is common), mitral valve prolapse, heart failure, and ischemic heart disease (IHD) **(Table 1)**.

- These ocular signs are not the same as Graves' orbitopathy (GO), which is the main extrathyroidal manifestation of Grave's disease and is seen in about 30% of Grave's patients. The signs and symptoms of GO are as follow:
- *Symptoms:* Lacrimation, grittiness or sandy sensation, photophobia, ocular pain (spontaneous or on eye movement), diplopia (variable or constant), decreased color sensitivity, and visual loss
- *Signs:* Lid retraction, periorbital soft tissue swelling and redness, conjunctival hyperemia and chemosis (conjunctival edema), exophthalmos (proptosis—forward protrusion of the eyes is due to immune-mediated inflammation in the retro-orbital fat), lagophthalmos (incomplete eye closure, especially at night), restricted ocular motility/strabismus, decreased visual acuity [due to dysthyroid optic neuropathy (DON)].

  These eye signs in hyperthyroidism arise from:
  - Sympathetic overactivity
  - Orbital inflammation/fibrosis
  - Autonomic imbalance

TABLE 1: Eye signs for thyroid.

Eye sign	*Description*
Upper eyelid signs:	
Eyelid retraction "stare" (Dalrymple sign)	The eyelids are retracted upward above the superior corneoscleral limbus and the "white" of the sclera is seen
Lid-lag (von Graefe's sign)	When the patient tracks an object downward with his/her eyes, the eyelid fails to follow the downward moving iris, and the same type of upper globe exposure which is seen with lid retraction is seen temporarily
Joffroy's sign:	Absence of wrinkling of the forehead when looking upward with the face tilted downward
• Stellwag's sign • Kocher's sign • Grove's sign • Boston's sign • Gifford's sign • Jellinek's sign	• Infrequent and incomplete blinking • Spasmodic lid retraction on fixation • Resistance felt when trying to pull upper lid forward • Jerky upper lid movement on downward gaze • Difficulty everting the upper eyelid • Hyperpigmentation of upper eyelid
Lower eyelid signs:	
• Enroth's sign • Griffith's sign	• Edema/swelling of lower(or both) eyelids • Lower lid lag or abnormal movement on upward gaze
Pupillary signs:	
• Cowen's sign • Knies sign	• Jerky pupillary constriction in bright light • Unequal dilation of pupils in low light
Conjunctival/vascular signs:	
• Goldzieher's sign • Riesman's sign	• Deep injection (redness) of temporal conjunctiva • Audible bruit over the closed eyelid (due to increased orbital blood flow)
Ocular movement signs:	
• Mobius sign • Suker's sign • Ballet's sign • Jendrassik's sign • Wilder's sign	• Lack of convergence (failure to maintain medial eye movement) • Inability to maintain fixation in extreme lateral gaze • Restricted movement in one or more extraocular muscles • Paralysis of all extraocular movements • Jerky eye motion from abduction to adduction

And these clinical signs help in diagnosis and monitoring of thyrotoxicosis and thyroid eye disease (TED).

Thyroid function tests:

- Low TSH, high free T_3, and T_4
- Subnormal TSH response to TRH
- *Other blood tests:* Mild anemia, thrombocytopenia, hypercalcemia, and raised alkaline phosphatase.

Symptoms and Signs of Hypothyroidism

Cardiovascular system: Bradycardia, abnormal baroreceptor function, reduced plasma volume, signs of heart failure, and pericardial effusion.

Respiratory system (RS): Impaired respiratory center control mechanisms, respiratory muscle weakness, and pleural effusions

Central nervous system: Slow movement, mental slowing, excessive sleepiness, and depressed mood

Gastrointestinal system: Delayed gastric emptying and constipation. Impaired clearance of free water and impaired hepatic drug metabolism:

- Fatigue, modest weight gain, cold intolerance, menorrhagia, muscle cramps, vague aches, and pain
- Dry coarse hair, dry skin, large tongue, and swelling of the legs

- Anemia, hypoglycemia, hyponatremia, and increased cholesterol levels
- Carpal tunnel syndrome.

How will you test thyroid function?

Ans. Total T_4, T_3, and TSH are the basic tests for diagnosis of hypothyroidism and hyperthyroidism **(Table 2)**.

Free T4 and T3: These are better indicators of the functional thyroid status than total levels as they are the metabolically active fractions and are not affected by thyroxine-binding globulin (TBG) levels. The relationship between total and free levels depends upon binding to TBG which is affected by various conditions. TBG is increased by estrogens, tamoxifen, and pregnancy and in some cases of liver disease, including hepatitis. Here, there would be a high total value but normal function. Decreased TBG is found with some cases of chronic liver disease, nephrosis, acromegaly, severely ill patients, and patients on large doses of glucocorticoids and androgens/anabolic steroids.

Thyroid-stimulating hormone: It is an early indicator of hypo- and hyperthyroidism in the presence of a normal pituitary thyroid axis and is a useful screening test. TSH is not useful when the pituitary thyroid axis may be disturbed such as at initiation of thyroxine therapy, pregnancy, and in patients with hyperthyroidism in the early stage of treatment and in rare situations such as central hypothyroidism, end-organ thyroid hormone resistance, and TSH-secreting pituitary adenomas.

Other advanced investigations include TBG level, radioiodine uptake test, and antithyroglobulin antibodies. TFTs are advisable in patients with goiter to detect subclinical hypo- or hyperthyroidism.

TABLE 2: Diagnosis of hyperthyroidism and hypothyroidism.

		Hypothyroidism	
	Hyperthyroidism	*Primary*	*Secondary*
TSH	Low	High	Low
T_4	High	Low	Low

How is subclinical hypothyroidism (SHypo) and subclinical hyperthyroidism (SHyper) managed?

Ans. The subclinical condition is diagnosed when TSH levels are abnormal in the presence of normal free triiodothyronine 3 (FT_3) and FT_4.

There are small differences between the guidelines by different societies on this topic.

SHypo treatment (2013 European Thyroid Association Guidelines):

- *TSH >10 mU/L:* Age <70 years—treated with levothyroxine, age >70 years—symptomatic patients should be considered for treatment
- *TSH 5–10 mU/L:* Age <70 years—symptomatic patients should be given a trial of 3 months to look for a response.

All other patients with TSH >5 mU/L should get a TFT done in 6 months to look for progress.

SHyper treatment (2015 European Thyroid Association Guidelines):

- *TSH 0.1–0.39 mU/L:* Follow up at 3–6 months. Patients with persistent SHyper may require treatment with either antithyroid drugs, β-blockers, or radioiodine therapy based on risk factors.
- *TSH <0.1 mU/L:* All patients need further evaluation and most would need treatment.

Subclinical hypothyroidism or hyperthyroidism is unlikely to impact perioperative care significantly and should not routinely lead to postponement of surgery.

What is euthyroid sick syndrome?

Ans. The euthyroid sick syndrome is described as abnormal TFTs that occur in the setting of acute and severe nonthyroidal illness without preexisting hypothalamic pituitary and thyroid gland dysfunction. The most common findings are low T, T, and TSH. The TFT abnormalities are reversible after recovery from the illness.

These changes may be caused partly by cytokines or other inflammatory mediators acting at the hypothalamus, pituitary, thyroid gland, and hepatic deiodinase system. The degree of thyroid function disturbance generally correlates with disease severity and could be a marker of poor prognosis. It is not certain whether these changes are part of an adaptive response to lower tissue energy requirements during disease or could be a maladaptive response. Administration of thyroid hormones in this situation is controversial and is generally not recommended.

How important is it to treat hyperthyroidism/hypothyroidism?

Ans. The thyroid hormone is highly metabolically active, has vital effects on cardiovascular physiology, and affects the response to anesthetic drugs. The stress of surgery and anesthesia can lead to decompensation of both hypothyroid and hyperthyroid states increasing

perioperative morbidity and mortality. Also, manipulation of the thyroid gland during surgery may cause the release of thyroid hormone and precipitate a hyperthyroid crisis. So, the endocrine status should be medically controlled and patients rendered euthyroid prior to surgery.

What are the causes of hyperthyroidism?

Ans. The causes are Graves' disease, thyroiditis, toxic multinodular goiter, toxic adenomas, iodine-induced, struma ovarii, thyroxine overdose, side effects of certain medications such as amiodarone, irradiation thyroiditis, etc.

How is hyperthyroidism treated?

Ans. Hyperthyroidism can be treated by antithyroid medications, surgery, and radioactive iodine 131 therapy.

The treatment has to be individualized to each patient. Antithyroid drugs, namely carbimazole, methimazole, and propylthiouracil (PTU), act by interfering with the organification of iodine. Carbimazole acts by its conversion to methimazole in the liver. PTU in large doses also blocks the peripheral conversion of T_4 to T_3. Antithyroid drugs are usually the first-line treatment and can be continued or followed by surgery or radioactive iodine in suitable cases.

The initial oral dose of methimazole is 5–40 mg/day and that of carbimazole is 10–60 mg/day based on the free T4 levels. The PTU dose is 100 mg every 8 hours. The doses are titrated to response. When initiating treatment, the patient is investigated 3–4 weekly for free T_3 and T_4 levels till control is achieved. TSH is a poor indicator of response at the initiation of treatment. The maintenance dose is usually 2.5–10 mg of methimazole or 50–100 mg of PTU daily.

β-blockers are used in early treatment for control of palpitations and tremors. Propranolol 20–40 mg 6 hourly is commonly used. Longer acting β-blockers, e.g., atenolol or bisoprolol, may also be used. High doses of propranolol also have an added advantage of inhibiting the peripheral conversion of T_4 to T_3. Cardioselective β-blockers may be preferred in asthmatics and in those at risk for atrial fibrillation. Patients with atrial fibrillation require anticoagulation. It should be noted that patients with hyperthyroidism require higher doses of digoxin.

Patients with Graves' disease in remission need to be investigated for thyroid hormone levels as relapses are known.

What are the common side effects of antithyroid drugs?

Ans. Major side effects of antithyroid drugs (so corresponding investigations should be ordered preoperatively):

- *Agranulocytosis:* May need granulocyte colony-stimulating factor
- *Hepatotoxicity:* Immunoallergic hepatitis seen with PTU
- *Vasculitis:* Acute renal dysfunction, arthritis, skin ulcerations, vasculitic rash, and upper and lower respiratory symptoms, including sinusitis and hemoptysis. May need high-dose glucocorticoid therapy or cyclophosphamide.
- *Teratogenicity:* PTU better than methimazole during pregnancy. Both drugs are safe for lactation.
- *Iodides:* Iodides inhibit hormone release at high concentration, decrease synthesis, and block the conversion of T_4 to T_3. The onset of action is immediate but lasts only for a few weeks. Three drops of a saturated solution of potassium iodide are given 8 hourly or the radiocontrast dye, ipodate, can be given 0.5–3 g every day.

Describe anesthesia management in a hyperthyroid patient.

Ans. Surgery is delayed until the patient is euthyroid. An endocrinologist consultation is required to titrate therapy. Only life-/limb-/organ-threatening emergency surgery should be done in an uncontrolled hyperthyroid state. (Sight-threatening Graves' ophthalmopathy is an endocrine emergency. The patient may require orbital decompression before the hyperthyroid state is completely controlled.)

Elective surgery: Elective surgery should be undertaken after a minimum of 3–4 weeks of therapy with methimazole or PTU. The delay is essential to achieve control as there are large stores of thyroid hormone. Free T_3 and T_4 are recommended for evaluating the control of the hyperthyroid state. Change in TSH levels is slower; therefore, TSH may not be representative of an early response to treatment.

If surgery is semiurgent, it should be scheduled after 7–14 days. Treatment with iodide and propranolol is advisable. Antithyroid drugs should also be started even though they by themselves would not achieve control. Dexamethasone 2 mg 6 hourly intravenous (IV) reduces hormone release as well as inhibits the peripheral

conversion of T_4 to T_3. Lugol's iodine given for 10 days preoperatively in a dose of 0.5 mL three times a day may be useful to reduce the vascularity of the thyroid gland and decrease surgical blood loss as antithyroid drugs make the thyroid more vascular. Although this practice is no longer routinely followed before thyroid surgery, it is finding favor for early control of the hyperthyroid state. It is important that patients are started on antithyroid drugs before iodides are started as the administration of iodides in iodine-deficiency patients may worsen the hyperthyroid state. Vitamin D deficiency should be corrected prior to surgery to reduce the risk of postoperative hypocalcemia.

Emergency surgery: Esmolol is titrated to control heart rate [caution in patients with congestive heart failure (CHF)]. It is administered in the dose of 0.5 mg/kg IV followed by an infusion of 0.03–0.3 mg/kg/min.

Anesthesia management for thyroidectomy in a patient with hyperthyroidism: General anesthesia is the technique of choice for thyroidectomy. Some centers specialize in thyroid surgery under cervical plexus blocks or cervical epidural anesthesia. These should only be attempted by anesthetists skilled in the procedure for a select group of patients.

All antithyroid medications are continued on the morning of surgery. Sedative premedication is prescribed if the airway is not involved. Benzodiazepines reduce anxiety and catecholamine release. Anticholinergic drugs cause tachycardia and alter heat-regulating mechanisms and are best avoided.

Monitoring would include oxygen saturation (SpO_2), BP, electrocardiography (ECG), end-tidal carbon dioxide ($EtCO_2$), and temperature. Arterial cannula and continuous BP monitoring are indicated in patients with an uncontrolled thyroid condition.

Maintain a deep plane of anesthesia so as to reduce the sympathetic response to surgical stimulation. Hyperthyroidism has not been seen to increase minimum alveolar concentration (MAC) in animal studies. Isoflurane and sevoflurane are inhalational agents of choice.

Thiopentone decreases peripheral conversion of T_4 to T_3 and is preferred. Ketamine is avoided due to sympathomimetic action. Clearance and distribution volume of propofol are increased in hyperthyroid patients. When total IV anesthesia (TIVA) is used, propofol infusion rates should be increased.

Besides pancuronium which has a vagolytic action, muscle relaxants in current practice, e.g., vecuronium or rocuronium, have minimal hemodynamic side effects and are safe. Succinylcholine may be used in indicated cases.

Avoid indirect-acting vasopressors, e.g., ephedrine. Phenylephrine, a directly acting alpha-adrenergic receptor agonist, is the drug of choice for hypotension. Avoid adrenaline-containing solutions. Glycopyrrolate is used with neostigmine for reversal.

Antithyroid drugs are stopped after thyroidectomy but β-blockers are continued for 7–10 days as the half-life of thyroxine is 7–8 days.

What is thyroid steal?

Ans. "Thyroid steal" is a historical practice used by George W Crile in the early 1900s for patients planned for thyroidectomy for Graves' disease. The aim was to avoid preoperative stress associated with the anticipation of surgery which could lead to thyroid storm. The patients were admitted without knowing the day of surgery. Every morning, saline injections were given to the patient which were substituted with anesthetic drugs on the day of surgery. Thus, in a way, as the patient was not actually aware that he/she was to be operated that day, the thyroid was "stolen." *This is not to be confused with the "thyroid steal syndrome" where blood is stolen (diverted) from cerebral circulation to thyroid circulation due to excessive vascularity of thyroid gland or nodule, causing transient cerebral ischemia.*

What is a thyroid storm?

Ans. It is a life-threatening condition where exacerbation of the hyperthyroid state is precipitated by acute stress such as infection, surgery, and trauma. It has been reported up to 12–18 hours after surgery in hyperthyroid patients. Patients on amiodarone can sometimes develop this condition. Tachycardia and hyperthermia are classical signs. Other diagnostic criteria for thyroid storm in Graves' disease include arrhythmias, CHF, agitation, delirium, psychosis, stupor, coma, nausea, vomiting, diarrhea, and hepatic failure.

Thyroid function tests are not diagnostic because the condition is thought to be precipitated by decreased

Reference: Donnelly PE, Winch DE, Pollard J. "Thyroid Steal"–a historical approach to thyroid surgery for Graves' disease. Thyroid. 2016;26:201.

binding of thyroid hormones to TBG increasing the amount of the free fraction.

- *Differential diagnosis:* Malignant hyperthermia, neuroleptic malignant syndrome, and pheochromocytoma
- *Treatment:* Treat the precipitating cause, aggressive fluid management, cooling, PTU, sodium iodide, and methylprednisolone 50 mg IV

α- and β-blockers should be given under intensive hemodynamic monitoring. Magnesium has also been used to control dysrhythmias.

What are the causes of hypothyroidism?

Ans. Hashimoto's thyroiditis, lymphocytic thyroiditis, thyroid destruction (from radioactive iodine or surgery), pituitary or hypothalamic disease, radiation therapy to the neck for nonthyroid malignancies, medications, e.g., tyrosine kinase inhibitors, and severe iodine deficiency.

Some genetic disorders present as multiple autoimmune endocrinopathies (MAEs). They are subdivided as type 1 MAE and type 2 MAE on the basis of the following characteristics:

- *Type 1 MAE:* Hypoparathyroidism, Addison's disease, and mucocutaneous candidiasis. Autoimmune thyroiditis is present in about 10–15% of patients.
- *Type 2 MAE:* Addison's disease, autoimmune thyroiditis, and type 1 diabetes.

What is myxedema coma?

Ans. Myxedema coma is a loss of brain function as a result of severe, long-standing hypothyroidism. This condition is mostly seen in elderly patients and tends to occur more often in women. The condition carries a very high mortality. Factors which may trigger myxedema coma in a person with poorly controlled hypothyroidism include drugs (particularly sedatives, narcotics, anesthesia, lithium, and amiodarone), infections, stroke, trauma, heart failure, gastrointestinal bleeding, hypothermia, and failing to take thyroid medications as prescribed.

Treatment: The combination of IV T_3 and T_4 is recommended. Fluid resuscitation is done with a normal saline solution. Glucose supplementation is given as required. Normothermia should be achieved with active warming. Mechanical ventilation and vasopressors may be required.

How will you treat hypothyroidism?

Ans. *L-thyroxine*: Treatment is started with 50–100 μg (25 μg in the elderly or in patients with IHD). The dose is titrated to clinical improvement and by monitoring TSH levels. Estimation of TSH and free T_4 is recommended for evaluating control.

T_4 has a half-life of 7 days. The onset of action is at 12 hours and it takes almost 2 weeks for peak action. The half-life of T_3 is 1.5 days and is available in an injectable form.

Thyroxine supplementation may precipitate or aggravate IHD. In patients with significant IHD, early revascularization and immediate postoperative initiation of thyroxine supplementation are advisable.

Discuss the implications of hypothyroidism on fitness for anesthesia and surgery.

Ans. Patients with overt hypothyroidism should be treated prior to elective surgery. Normalization of thyroid status will typically take weeks. If surgery is urgent but can be delayed for 24–48 hours, IV T_3 can be given for quicker action. The peak action of T_3 occurs at 36–72 hours. Because of an autoimmune etiology, both adrenocortical insufficiency and myasthenia gravis are more common in hypothyroid patients. Due to reduced adrenocorticotropic hormone response to stress, hypothyroid patients should receive hydrocortisone cover during periods of increased surgical stress. Hypothyroid patients should receive their usual morning dose of thyroxine. This is especially important for T_3, which has a shorter half-life.

Mild or subclinical hypothyroidism is unlikely to affect the perioperative course; therefore, postponement of surgery is not generally advocated.

Describe anesthesia in a hypothyroid patient (emergency surgery).

Ans. The risk of complications is higher when patients have an FT_4 <0.5 μg/dL or T_4 <1 μg/dL.

Use regional anesthesia wherever possible.

Specific investigations:

- Hemoglobin count as anemia is common
- Platelet count and clotting tests as dysfunction is known
- Serum electrolytes as hyponatremia can be present
- Blood sugar monitoring to detect hypoglycemia
- ECG—bradycardia, conduction blocks.

Avoid sedative premedication in overtly hypothyroid patients as they show increased sensitivity to sedative and anesthetic drugs, probably related to low cardiac output, reduced blood volume, impaired hepatic metabolism, and abnormal baroreceptor function. Chemoreceptor

responses are also blunted, increasing the risk of respiratory depression. Close postoperative monitoring is advisable.

Induction: Anesthetic drugs have to be used judiciously. Ketamine has the advantage of sympathetic stimulation. Fentanyl may be used in small titrated doses. Nitrous oxide is useful in reducing the requirement of volatile anesthetics as these patients are sensitive to the myocardial depressant action of the latter. Intermediate-duration nondepolarizing agents, e.g., vecuronium, are used. Pancuronium is not preferred in spite of its cardiovascular effects because of the risk of prolongation of action and residual neuromuscular blockade (NMB).

Exaggerated hypotension is common and should be treated with judicious fluid and ephedrine. Pure alpha-adrenoreceptor agonists are avoided. Dopamine and epinephrine can be used for severe hypotension. Such patients should also receive an additional dose of steroids.

Patients may have airway edema, large tongue, and enlarged thyroid making laryngoscopy and intubation difficult.

Intraoperatively monitor temperature, fluid and electrolyte status, and blood sugars. Invasive BP monitoring (± central venous cannulation and cardiac output monitoring) may be required in hypothyroid patients undergoing major surgery (fluid shifts, blood loss, etc.) and in those with cardiovascular involvement (arterial cannula under local anesthesia prior to induction is advisable). Neuromuscular monitoring with a peripheral nerve stimulator is advised as these patients have increased the incidence of myasthenia gravis. Higher current levels may be needed for nerve stimulation in hypothyroid patients due to thick skin and hypothermia. Warming measures are instituted to prevent hypothermia. Delayed recovery is common and postoperative ventilation may be required.

What problems do you anticipate in patients with large thyroid mass? What are the complications associated with retrosternal thyroid?

Ans.

- Tracheal compression, deviation, and invasion. Tracheomalacia may be present in long-standing cases which can predispose to intraoperative and postoperative airway collapse.
- Worsening of airway compromise in the supine position and with loss of muscle tone. Loss of spontaneous breathing and negative intrathoracic pressure after anesthesia and paralysis can also precipitate the collapse of the intrathoracic airway.
- *Retrosternal extension:* This can lead to SVC syndrome, airway compromise, cardiac compression, cerebral hypoperfusion as a result of arterial compression and thyrocervical steal, phrenic and RLN palsies, Horner's syndrome, pleural effusions, chylothorax, and pericardial effusions.
- Increased intraoperative bleeding
- Injury to laryngeal nerves—vocal cord palsy causing stridor and impaired airway reflexes, thereby increasing the aspiration risk.

How will you assess for tracheal deviation and compression?

Ans. Patients with tracheal narrowing by >30% are usually symptomatic and the risk of airway events increases when >50% narrowing is present. Patients with malignant tumors may have lymph node masses which may exert pressure effects on the surrounding structures.

History: Difficulty in breathing, stridor, or stridor on lying down.

Examination:

- *For deviation:* Palpation of and auscultation on the anterior neck to diagnose deviation
- *For compression:* Positive Kocher's test: Slight push on the lateral lobes causes stridor.

Investigations:

Computed tomography (CT) scan of the neck and upper thorax: Patients with significant mass effects or malignant tumors are likely to undergo a CT scan as part of the surgical workup. If not, it should be advised by an anesthetist in case of suspected airway compromise.

This helps to accurately delineate the site and degree of airway compromise. This also helps to predict the tracheal tube diameter and guide placement. A note of caution when assessing tracheal dimensions on CT scan: The tracheal cuts are slightly oblique on a standard CT image and may overestimate the tracheal diameter.

CT scans performed in different positions may be useful to record positional changes in the airway compression.

X-ray neck: Anteroposterior (AP) and lateral. With the widespread availability of CT scans, X-ray imaging of the trachea has a very limited role in current practice. It may be useful to detect tracheal compression and deviation in a resource-poor environment.

Flow–volume loops: These may help in diagnosing subclinical airway obstruction, particularly dynamic obstruction. Flow–volume loops will not change management in the presence of overt obstruction. Their practical role in airway assessment in an asymptomatic thyroid patient is debatable. The pattern seen on the flow volume loop depends on the site of tracheal compression (if it is a variable obstruction). Suprasternal compression will show an extrathoracic pattern, i.e., the inspiratory flow is predominantly affected whereas infrasternal compression will have an intrathoracic pattern, i.e., expiratory flow is affected. Large goiters and intratracheal extensions usually cause fixed obstruction where both inspiratory and expiratory flows are compromised.

What are the problems associated with intratracheal extension of the tumor?

Ans. These patients have a high risk of bleeding and tumor dislodgement leading to critical airway obstruction. Intubation should be attempted after a thorough examination of the airway including CT scan and flexible bronchoscopy. Rigid bronchoscopy with appropriate instruments and efficient suction should be immediately available for removal of clots or tumor fragments. These patients also have an increased risk of perioperative lung infections due to aspiration of blood or tumor. Surgery may involve tracheal resection and airway management should be planned accordingly.

How will you assess for retrosternal extension of thyroid mass?

Ans.

- *Symptoms:* Dyspnea, choking, hoarseness, and dysphagia
- Unable to "get below the swelling"
- Percussion over the manubrium sternum (seldom followed in current practice)
- Lateral neck X-ray
- CT scan

Superior vena cava obstruction: The patient will have edema and dilated veins on the head, neck, and upper part of the chest. Severe cases may have respiratory distress due to airway edema. Increased intraoperative bleeding is expected.

Large-bore venous access should be secured in lower limbs or femoral vein.

Pemberton's sign is the development of facial flushing, distended neck and head superficial veins, inspiratory stridor and elevation of the jugular venous pressure (JVP) upon raising of the patient's both arms above his/her head simultaneously, as high as possible. A positive sign is indicative of SVC syndrome. In current practice, with the widespread use of CT scan, this test is rarely performed as it can be dangerous in the OPD setting.

Which investigations will you prescribe and why? What is the role of flow–volume loops?

Ans.

Hemoglobin: Baseline. To estimate the maximum allowable blood loss and to detect anemia as antithyroid medication may cause bone marrow suppression.

Thyroid function tests: To confirm the euthyroid state (to detect subclinical abnormality). Patients with hypothyroidism on stable thyroxine dose with documented normal TFTs in the preceding 6 months do not require repeat TFT testing prior to surgery, if clinically euthyroid. Patients with Graves' disease in remission should have TFTs as relapses are known.

- *ECG:* Screening for IHD/dysrhythmias
- Serum creatinine if patient >60 years
- X-ray chest

CT neck: The anesthetist should always review the anatomy on CT scan images to assess airway status.

X-ray neck AP/lateral if CT neck not available—for tracheal deviation/compression.

Flow volume loop may detect subclinical airway obstruction and also suggest the type and site of obstruction, i.e., fixed/variable, intrathoracic/extrathoracic. In clinical practice, with the availability of CT scan images, flow–volume loops do not contribute much to the management in suspected airway compromise.

Indirect laryngoscopy is used to diagnose the preexisting vocal cord palsy.

What are the principles of anesthetic management for this patient?

Ans.

- Airway management at the induction of anesthesia, maintenance, and during the recovery period
- Management of complications of hypo-/hyperthyroidism
- Management of problems due to retrosternal extension

What monitoring will you institute?

Ans.

- ECG, SpO_2, $EtCO_2$, core temperature, and BP
- Invasive arterial pressure monitoring if the patient is hyperthyroid or major blood loss is expected.

How will you manage the airway in this patient?

Ans.

Preparation: Preparation and planning are of the utmost importance when dealing with a difficult airway. Alternate plans of action, if the primary plan fails, should be discussed by the anesthesia and surgical team to prevent panic and delays in management.

The patient might have airway obstruction due to external pressure, invasion into the trachea, severe deviation, or vocal cord palsy.

Problems anticipated:

- *Deviation:* Distorted anatomy—visualization of the larynx may be difficult.
- *Compression of the trachea:* The patient may be unable to lie supine. A small-sized endotracheal tube (ETT) may be required.

In a large thyroid, emergency airway by cricothyroid puncture or tracheostomy may be difficult or impossible. However, in very severe obstruction, tracheostomy under local anesthesia should be considered by the surgeon after careful review of the distorted anatomy. Ultrasound may be useful in identifying the trachea and the vascular structures near the trachea.

Review CT scan—to assess the site and extent of tracheal/bronchial obstruction and plan airway management.

A preoperative awake fiberoptic bronchoscopy may help to assess the airway involvement and help decide the induction and intubation technique. If a flexible/fiberoptic bronchoscope (FOB) is not available, videolaryngoscopy under topical anesthesia can help to assess the anatomy of the larynx and decide the plan for securing the airway:

- Avoid sedative premedication
- Difficult airway kit should be ready
- Keep small size flexometallic tubes handy
- A rigid bronchoscope (and a skilled operator) should be immediately available if required to hold the airway open and allow ventilation if airway collapse occurs on the induction of anesthesia. Turning the patient lateral or using forceps to lift thyroid away may also help to relieve the compression.
- Equipment for cricothyroidotomy/tracheostomy. Its role in emergency airway control in large anterior neck mass is limited.
- An experienced surgeon should be immediately available.

In case of a compromised airway, maintenance of spontaneous breathing by inhalational induction is the traditional approach. This is advocated for its potential reversibility if attempts at intubation fail. Sevoflurane in oxygen is the preferred agent. IV agents were previously not popular due to the increased incidence of apnea. However, the approach is slowly changing though it is still controversial.

It is important to note that in the presence of an obstructed airway, inhalational induction is likely to be slow with a longer time spent in the light plane of anesthesia. This can lead to higher incidence of adverse airway events such as coughing and laryngospasm. Attempting laryngoscopy during light plane of anesthesia can causing gagging, vomiting, laryngospasm, poor intubating conditions, and worsening of the airway. Complete airway obstruction during inhalational induction has the added disadvantage of delayed elimination of inhalational agent and slow return to the preanesthesia state.

Judicious intravenous anesthesia with/without muscle relaxation can give optimum conditions for airway control and should be given due consideration. Thus, the choice between asleep versus awake intubation and inhalational versus IV anesthesia should be done by an experienced anesthesiologist with readiness for emergent rigid bronchoscopy and other rescue procedures.

Awake videolaryngoscopic intubation under topical anesthesia is steadily finding popularity in management of a compressed or deviated trachea in patients with a thyroid mass. This technique would also require expertise and familiarity, as with other approaches. A hybrid approach, combining awake videolaryngoscopy and fiberoptic bronchoscopy, is a valuable option in selected patients with distorted or partially visualized airway anatomy. The advantages of this technique being a panoramic view of the oropharynx and laryngeal inlet with VL and ease to negotiate a narrowed, deviated, or compressed trachea beyond the glottis with FOB. This hybrid technique helps to improve success particularly in patients with glottic or subglottic narrowing and displaced trachea.

In patients who cannot lie supine due to airway obstruction, intubation may have to be done in a semisitting position and induction of anesthesia in this position can lead to severe hypotension.

In severe displacement of the trachea, a fiberoptic-guided intubation under topical anesthesia is the recommended technique. However, this technique should be used with caution in severe obstruction as the small

caliber of the airways may sometimes make the passage of adult fiberoptic scope difficult and precipitate complete obstruction which is not tolerated by the patient.

Controlled tracheostomy following a femorofemoral cardiopulmonary (CP) bypass that had been initiated under local anesthetic has been described in a case of airway obstruction with supraglottic edema due to a large thyroid mass. This is described as the ultimate solution to the problem of a difficult airway. However, it should be understood that a "stand by" CP bypass may not be able to rescue a "cannot intubate", "cannot oxygenate" situation. When indicated, the femoral vessels should be cannulated and CP bypass circuit primed and ready for use. More recently, venovenous ECMO has emerged as an option in such cases due to its advantages over conventional CP bypass.

A standard polyvinylchloride (PVC)-cuffed ETT is adequate in most cases. A flexometallic tube is preferred when there is tracheal compression and, if possible, the ETT should be placed beyond the length of (lower limit of) compression. It may also be useful in a tortuous airway. Extra care should be taken to secure the flexometallic tube as it is more prone to dislodgement.

Opioid with a shorter duration of action, e.g., fentanyl, is preferred in order to have minimal sedative effects at recovery. Isoflurane and sevoflurane are volatile agents of choice. Muscle relaxants (e.g., vecuronium) are guided by peripheral nerve stimulators.

If intraoperative RLN monitoring is practiced, muscle relaxants are avoided.

Other perioperative considerations:

- The eyes should be protected, especially if exophthalmos is present.
- The patient is positioned head-up to help venous drainage.
- The neck is hyperextended and should be well supported to avoid strain on cervical spine ligaments.
- Extension tubing for IV lines and long respiratory hoses may be required.
- Valsalva maneuver in Trendelenburg position is carried out to check hemostasis.
- Steroids may be given if extensive tracheal handling and edema are suspected.

Extubation should be smooth and coughing should be avoided to prevent bleeding. The surgeon may wish to observe the movement of the vocal cords at the end of the surgery. Though widely practiced in the past, it is not much favored in current practice. This could be due to the difficulty in performing an adequate laryngoscopy in a patient with recovering neuromuscular function and light plane of anesthesia and the attendant sympathetic stimulation. Also, vocal cord status at the end of surgery has been shown to be a poor predictor of postoperative cord function. If the assessment of vocal cord function is deemed necessary, flexible laryngoscopy may be more useful. Ultrasound imaging has also been used to observe the vocal cord movement.

At extubation, the possibility of tracheomalacia (due to the weakness of the tracheal rings) and vocal cord palsy should be kept in mind and the patient should be observed closely. Tracheomalacia should be suspected in long-standing goiters and the surgeon may palpate the trachea intraoperatively to assess the quality of tracheal cartilages. Tracheomalacia is defined as dynamic airway collapse with a decrease in the tracheal lumen by >50%. It causes collapse of the extrathoracic trachea during inspiration and collapse of the intrathoracic trachea in expiration. Extubation should be performed after adequate preoxygenation and with preparation for reintubation if required. An ETT may be slowly withdrawn and signs of obstruction watched for. In difficult intubation, it is advisable to extubate over an airway exchange catheter. Flexible laryngoscopy during spontaneous respiration in an extubated trachea or through a laryngeal mask airway (LMA) is useful for the diagnosis of respiratory obstruction due to tracheomalacia or vocal cord palsy.

How is the leak test performed?

Ans. With the ETT in place, the cuff is deflated. A leak around the ETT can be detected on intermittent positive pressure ventilation (IPPV) by palpating over the trachea or observing for the difference between the set and expired tidal volume. During spontaneous respiration, the patient should be able to breathe around the tube when the tubal lumen is plugged, suggesting free space around the tube.

The leak test has been described for airway edema and its usefulness in tracheomalacia is controversial.

What is the management of tracheomalacia?

Ans. Post-thyroidectomy, tracheomalacia is rare in developed countries but has a higher incidence in countries such as India where patients do present with large goiters.

Tracheomalacia causing airway obstruction requires intubation. Noninvasive continuous positive airway pressure (CPAP) may help to relieve symptoms in mild

cases. Fibrosis around the trachea stabilizes the tracheal wall but may take several days. Tracheostomy may be required in some cases. Tracheal stents or surgical repair may be required in patients with a persistent problem.

What is the postoperative analgesia plan?

Ans. Severe pain is uncommon and simple analgesics such as paracetamol and nonsteroidal anti-inflammatory drugs (NSAIDs) are usually sufficient. Wound infiltration with bupivacaine may also be used.

What is the anesthesia management for intraoperative nerve monitoring (IONM) in thyroid surgery?

Ans. In IONM in thyroid surgery, the RLN is stimulated and electromyographic (EMG) signals are picked up from the vocal cords which help in the identification of RLN, thereby avoiding injury. IONM is increasingly being used during thyroid surgery, particularly in patients with malignant tumors. However, a recent Cochrane database review has shown no conclusive evidence for the superiority or inferiority of IONM over visual nerve identification for RLN injury. The two main components of IONM are nerve stimulation and assessment of vocal fold response to nerve stimulation. The stimulation of RLN can be done with:

- Intermittent stimulation—a low-voltage electric current delivered by a handheld probe
- Continuous stimulation—an electrode attached to the ipsilateral vagus nerve.

The vocal fold response to nerve stimulation is measured by a laryngeal surface electrode system either built into or attached to the ETT **(Fig. 1)**.

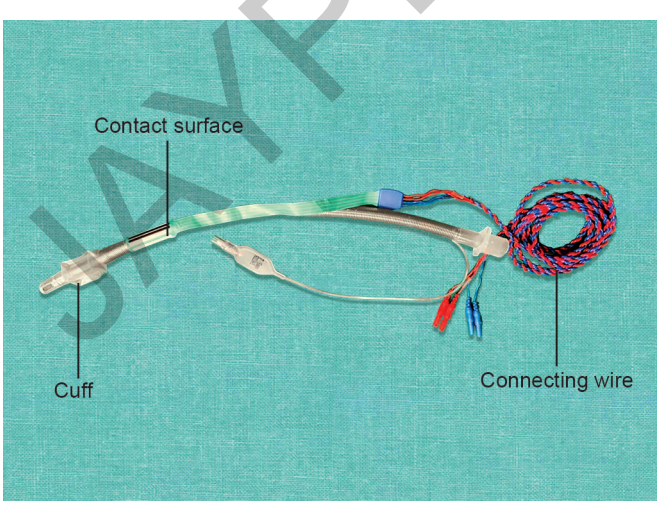

Fig. 1: Electromyography endotracheal tube.

During surgery, the vocal fold response to nerve stimulation is displayed on a nerve monitor as auditory or visual EMG signals. Change in the EMG signal pattern alerts the surgeon to possible RLN irritation. Before starting the IONM, primary vagal stimulation is done to confirm correct ETT placement and to rule out any preexisting defects in vocal cord activity and other confounding factors such as residual neuromuscular junction blockade (NMB) and device malfunction.

Anesthesia concerns during thyroid surgery under IONM are:

- NMB interferes with monitoring as paralyzing agents will interfere with obtaining of an EMG signal. Therefore, *neuromuscular conduction should be normal at the time of nerve monitoring.* The strategies for NMB management for IONM are as follows:
  - Avoiding NMB completely. Intubation is performed without a muscle relaxant. This technique needs expert care to avoid violent coughing and pressor response.
  - Using succinylcholine to facilitate endotracheal intubation, thus allowing rapid termination of NMB. However, one has to consider the side effects and contraindications of succinylcholine.
  - Using a single induction dose of short-/intermediate-acting nondepolarizing neuromuscular blocking agents (NMBAs) for intubation and allowing spontaneous recovery. This is ideal if the time interval between intubation and IONM is around 45–60 minutes. A peripheral nerve stimulator is used to confirm complete recovery of the neuromuscular junction.
  - Using rocuronium for intubation. The reversal of NMB is achieved with sugammadex at any point in time after rocuronium administration. This strategy has the dual advantage of good intubating conditions and complete neuromuscular junction recovery immediately, without having to wait for the elimination of the muscle relaxant. In India, the availability and high cost of sugammadex are prohibiting factors.
  - Anesthetic agents
  - Inhalational anesthetic agents do not interfere with triggered EMG signals and can be used during thyroid surgeries (unlike neurosurgery IONM cases)
  - TIVA with propofol and remifentanil is advised for neurosurgery IONM and can be used in thyroid

surgeries also. If remifentanil is not available, TIVA using propofol, fentanyl, and small doses of dexmedetomidine provides good conditions for IONM.

- It is advisable (but not mandatory) to use a videolaryngoscope for intubation to confirm the accurate positioning of electrodes assembly. The ETT should be positioned such that the electrodes embedded in the ETT should come in contact with vocal cords. Hyperextension of the neck for surgical exposure may cause outward movement of the tube, and therefore a check laryngoscopy is advisable to confirm the correct ETT position.

What are the problems of positioning for thyroid surgery?

Ans.

Thyroid surgery requires hyperextension of head. Care should be taken to support the head adequately to prevent strain on cervical spine ligaments and spine injury. In patients with restricted or painful neck extension or severe osteoporosis, the limit of comfortable neck extension should be assessed prior to anesthesia and extreme position is avoided during intubation and surgery.

Arms should be adducted by the patient's sides whenever possible. When arms are positioned at right angles, care should be taken to prevent hyperextension at the shoulder joint which can stretch the brachial plexus.

What are the possible intraoperative problems? Discuss their management.

Ans.

- *Increased airway resistance (causes):*
  - Kinking of the tube, secretions obstructing the tube, endobronchial intubation, and bronchospasm
  - Surgeons manipulating the trachea during dissection
  - External compression of trachea/bronchi distal to the ETT in cases of large retrosternal thyroid
  - Rarely, a part of the intratracheal tumor gets dislodged during intubation or manipulation by the surgeon and migrates distally into the tracheal or bronchial lumen causing an obstruction.
  - *Accidental extubation/disconnection:*
    - Hyperextension of the head causes ETT to migrate outward.
    - Surgeons operating near the airway, so more chances of airway accidents.
- *Thyroid crisis*

Can thyroid surgery be done as day surgery?

Ans. It is not routinely recommended as postoperative airway problems are possible. It may be considered in a select group of patients and where a very good community medical support system is available.

What are the possible postoperative problems and their management?

Ans.

Respiratory Distress

Causes:

- Specific to thyroid surgery:
  - *Airway obstruction:*
    - *Wound hematoma:* Traditionally, suture cutters were kept at the bedside to enable rapid relief of a hematoma. It is essential to open all layers to relieve the obstruction. Early intubation is advisable when significant hematoma is present as airway compression and edema (hematoma obstructs venous drainage causing edema) may worsen rapidly.
    - RLN damage
    - *Tracheomalacia causing dynamic airway obstruction:* Urgent reintubation, possibly tracheostomy, and some form of tracheal support.
    - *Laryngeal edema (uncommon):* Mostly related to large hematoma obstructing venous drainage. Other causes are trauma during intubation and myxedema.
    - *Hypocalcemia causing laryngospasm:* Hypocalcemia usually occurs at about 36 hours postoperatively, usually temporary, unless there is severe damage to all the parathyroid glands. Treatment includes 10 mL of 10% calcium gluconate. Long-term calcium supplementation is required.
- All causes of postanesthesia respiratory distress.

Metabolic Disturbances

- *Hypothyroidism:* May need thyroxine supplements
- *Hypoparathyroidism:* This may be due to direct trauma to the parathyroid glands, devascularization of the glands, or removal of the glands during surgery.
- *Hypocalcemia:* The cause of transient hypocalcemia after surgery is not clearly understood. It may be attributable to temporary hypoparathyroidism caused by reversible ischemia to the parathyroid glands. Other

hypotheses to account for transient hypocalcemia not caused by hypoparathyroidism include calcitonin release and hungry-bone syndrome.

Q Describe the nerve supply of larynx.

Ans.

- Most of the muscles of the larynx receive their innervation via the RLN branch of the vagus nerve except the cricothyroid muscle which receives its innervation via the external laryngeal nerve. Cricothyroid muscle is important for tensing of the vocal cord.
- Sensory innervation of the larynx is via branches of the vagus nerve. Above the vocal folds, the sensory innervation of the larynx is via the internal laryngeal nerve. Below the vocal folds, it is by the branches of the RLN.
- Parasympathetic innervation is mainly by the branches of the vagus nerve.

Q Enumerate causes and describe common nerve injuries after thyroid surgery.

Ans. *Causes:* Contusion, traction, entrapment, actual transection, and ischemia

External Branch of the Superior Laryngeal Nerve
Injury causes a change of voice quality and loss of vocal stamina. Treatment involves speech therapy.

Recurrent Laryngeal Nerve
Unilateral injury: Unilateral cord palsy causes hoarseness and a weak cough but usually without significant airway obstruction at rest. The patient has a risk of aspiration. Compensatory hyperadduction by the opposite cord may allow adequate vocal and sphincter function.

Bilateral injury: Bilateral injury of the abductors can cause unopposed adduction of cords causing complete glottic closure. The patient will need immediate tracheostomy. In bilateral complete denervation, both vocal cords are in cadaveric position. Phonation is severely affected. Stridor (predominantly inspiratory) may be present and the patient carries a very high risk of aspiration:

- Initial treatment involves ensuring an adequate airway. Endotracheal intubation is the first line of management. IV steroids may be helpful in some cases of reversible damage.
- A trial of extubation may be performed after several days in a controlled setting as reintubation may be necessary. Extubation over an airway exchange catheter is advisable if the patient has a difficult upper airway. If nerve function has not recovered after the second trial of extubation, tracheostomy is warranted.
- Corrective procedures are generally not performed for at least 6 months after surgery as delayed recovery is possible.
- Treatments of vocal cord paralysis include cordotomy (relieves stridor but increases aspiration risk), intracordal injection, laryngeal framework surgery, thyroplasty, and laryngeal reinnervation.

Q What is the role of cervical plexus blocks and cervical epidural anesthesia in thyroid surgery?

Ans. Cervical plexus blocks and cervical epidural anesthesia in thyroid surgery are controversial. Regional techniques may be possibly used in cooperative patients with small nonmalignant thyroid tumors.

Proponents cite avoiding airway manipulations and monitoring vocal cord function as a major advantage of the technique. Unilateral combined superficial and deep cervical plexus blocks have been performed for unilateral surgery. A bilateral combined block is contraindicated because of the risk of bilateral phrenic nerve palsy. Superficial cervical plexus blocks have been tried unilaterally and bilaterally as part of multimodal analgesic protocols. A meta-analysis by Mayhew and colleagues on the analgesic efficacy of bilateral superficial cervical plexus block (BSCPB) for thyroid surgeries did not find any difference in intraoperative opioid requirement but found a reduction in need for rescue analgesia in the first 24 hours following surgery. More evidence is needed before introducing them into standard postoperative analgesia protocols for thyroid surgery.

Superficial cervical plexus blocks have been used for video-assisted thyroidectomy.

Cervical epidural catheters have been used in some reports. However, the risk of bradycardia, hypotension, and acute ventilatory failure related to respiratory muscle paralysis is high and the patient needs close monitoring.

Q Describe the superficial and deep cervical plexus blocks.

Ans.

Superficial Cervical Plexus Block
The superficial cervical plexus block targets the four sensory branches of the cervical plexus (the lesser occipital, the great auricular, the transverse cervical, and the supraclavicular—which emerge from the anterior primary rami of C2-C4). Bilateral administration of this

block can provide analgesia for surgeries of the anterior neck including thyroid surgery.

Indications: Anterior neck surgery, thyroidectomy, and carotid endarterectomy

Technique: Following sterile preparation of the neck, the cricoid cartilage and the posterior border of the sternocleidomastoid muscle (SCM) are palpated and identified. A 27-gauge needle, bent at approximately 60°, is inserted superficially along the posterior margin of the clavicular head of the SCM at the level of cricoid cartilage. After aspiration, an initial 3-mL local anesthetic is deposited at this point, followed by 7 mL injected subcutaneously in a caudad and cephalad direction along the posterior border of the muscle (3–5 mL per each redirection). More recently, ultrasound guidance has been employed to aid in locating the superficial cervical plexus. Under ultrasound guidance, the branches are seen posterior to the sternocleidomastoid and superficial to prevertebral fascia (which overlies the middle and anterior scalene muscles). 5–10 mL of local anesthetic may be instilled in the plane superficial to the prevertebral fascia and deep to SCM.

Complications: Inadvertent deep cervical plexus block and consequent Horner syndrome, hematoma, and local anesthetic systemic toxicity (LAST).

Deep Cervical Plexus Block

This has been described as essentially a paravertebral block at the level of C2-C4 spinal nerves. A line is drawn joining the tip of mastoid process to the Chassaignac tubercle (at C6 transverse process). C2 transverse process is 1 finger breadth (or 1.5 cm) below the mastoid process on this line, and C3 and C4 are marked at similar distances inferiorly along the same line. (These distances are estimates at best.) At each of these three levels, after raising a skin wheal with a local anesthetic, a 22-G needle is advanced medially and caudally, with an aim to hit the corresponding transverse process. The needle is walked off laterally till it slips off the most lateral tip of the bone and then relocated to this lateral most tip (to avoid contact with the vertebral artery). 4–5 mL of local anesthetic is injected at each level after careful aspiration.

Complications: Accidental intra-arterial injection (into vertebral artery) and LAST, phrenic nerve block, Horner syndrome, RLN palsy, brachial plexus blockade **(Fig. 2)**.

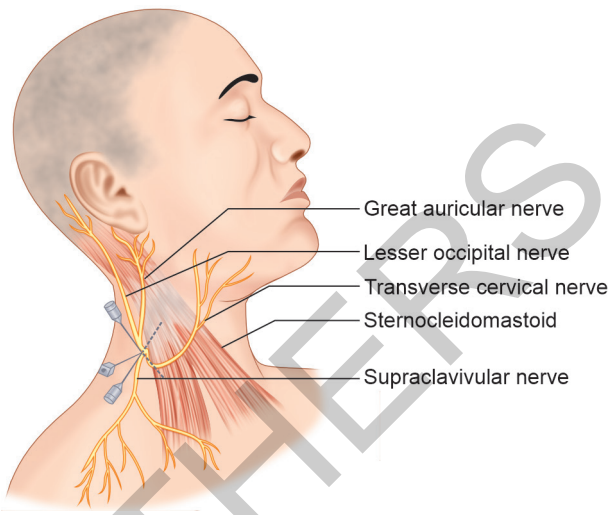

Fig. 2: Superficial cervical plexus block—nerves and landmarks.

Q What are the implications of pregnancy in hyperthyroidism?

Ans. During pregnancy, the increase in TBG causes an increase in total serum thyroid hormones which can lead to an erroneous diagnosis, so free T_4 and T_3 levels should be used to determine thyroid status.

Both PTU and methimazole cross the placenta and can affect fetal thyroid function, especially at higher doses. PTU is less teratogenic than methimazole. Most women with Graves' disease can be treated medically during pregnancy, with a target T_4 level at or slightly higher than the upper normal limit (and the lowest effective dose) to ensure normal thyroid hormone levels in the fetus. Iodides cause fetal hypothyroidism and are contraindicated, so is radioactive iodine.

Graves' disease generally improves in the second and third trimesters of pregnancy, allowing reduction or discontinuation of antithyroid drug therapy, although it can exacerbate in the postpartum period.

During pregnancy, ultrasonography (USG) monitoring is used to check for the presence of fetal goiter. Fetal goiter can indicate excessive antithyroid drug treatment in the mother or fetal Graves' disease. Maternal complications of Graves' disease in pregnancy include preeclampsia and preterm delivery.

Women receiving thyroxine therapy for hypothyroidism should have their dose increased by up to 50% during pregnancy. Avoiding maternal (and fetal) hypothyroidism is extremely important because of potential damage to fetal neural development, an increased incidence of miscarriage, and preterm delivery.

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