Complications of thyroid surgery

Introduction

The first recorded thyroid operation is credited to Albucacis in Spain in around the year 1000. About 170 years later, Frugardi attempted to induce atrophy of a goitre by transfixing it with shoelaces. From this surgical technique evolved slowly via methods including mass ligatures, chemical cautery, morcellation and injection of turpentine, to the more familiar concepts of modern thyroidectomy (Hegner, 1932; Dionigi et al, 2008). Today, surgeons continue to refine the technique via new equipment such as Harmonic scalpels and nerve monitors, and novel techniques such as minimally-invasive and non-cervical approaches (e.g. via the axilla or the mouth).

Mortality after thyroidectomy exceeded 40% in the early 1800s, as a result of haemorrhage, jugular venous thrombosis, mediastinal sepsis and air embolism. This plummeted during the 19th century driven by advances in asepsis, haemostatic forceps and the modern technical concepts of thyroidectomy developed by Lister, Spencer Wells, Billroth, Halsted and Kocher (Hegner, 1932). Indeed, Kocher received the Nobel Prize for Medicine in 1911 in recognition of his contributions to the understanding of thyroid physiology and improvements in the safety of thyroidectomy (Mihai et al, 2008).

Currently, some 2500 thyroid operations are performed annually in the UK by 99 members of the British Association of Endocrine and Thyroid Surgeons (British Association of Endocrine and Thyroid Surgeons, 2009), although the exact total numbers for the UK are not known. Thyroidectomy is performed by a mix of surgeons, ranging from dedicated endocrine surgeons in tertiary centres to general and ear, nose and throat surgeons with or without a specialist interest. In the UK, only a handful of surgeons perform more than 100 cases in a year (British Association of Endocrine and Thyroid Surgeons, 2009); in the USA half of all thyroid operations are performed by surgeons who undertake fewer than five cases per year (Saunders et al, 2003). Consequently, junior doctors often have minimal exposure to thyroid surgery and little experience in postoperative management of such patients. While refinements of technique and perioperative care have minimized mortality and morbidity, significant complications can and do occur after thyroidectomy. Their potential severity and rapidity of progression means that such junior surgical doctors may be called upon in the emergency situation to intervene.

Mr John M Findlay *is Core Surgical Trainee in the Department of General Surgery, Royal Berkshire Hospital, Reading RG1 5AN, and* **Mr Radu Mihai** *is Consultant Endocrine Surgeon and Honorary Senior Clinical Lecturer in the Department of Surgery, John Radcliffe Hospital, Oxford*

Correspondence to: Mr JM Findlay

Thyroid surgery

The anatomy, physiology and operative aspects pertinent to this article have previously been detailed (Gill and Agrawal, 2009; Mihai, 2011). However, it is important to be aware that there are five different operations that can be undertaken (*Table 1*). Rarely, a sternotomy or thoracotomy is required in addition to retrieve a retrosternal or intrathoracic goitre. Other intraoperative decisions are beyond the scope of this article, as is generic postoperative morbidity. This article concentrates on those most severe and common complications which junior doctors may encounter on the wards, but it is important to be aware of less common complications, which are also discussed.

Airway compromise after thyroidectomy

Haemorrhage and haematomas

Postoperative haemorrhage is an uncommon but potentially catastrophic complication. Some 1% of total thyroidectomy patients return to theatre for haemostasis (British Association of Endocrine and Thyroid Surgeons, 2009). The vast majority of such complications occur in the first 6 hours postoperatively, so all patients should be observed for a certain length of time postoperatively. This has an impact on the ability to perform day-case surgery. It is vital to identify such bleeding early, as rapid accumulation of blood may cause airway obstruction. This is caused by progressive oedema of the laryngeal mucosa, driven by increasing venous congestion.

The diagnosis is clinical (*Table 2*), with neck haematomas occupying a spectrum. Immediate distinction must be made between those causing (or with the imminent potential to cause) airway compromise, or those small collections which do not progress and which can be observed and managed conservatively. The presence of doubt mandates immediate senior help.

Airway patency should be assessed based on the ability to speak normally and the absence of stridor. Oxygen saturations will remain normal until the patient is in extremis and so should not guide management. If there is any concern, basic airway procedures must be instituted. Patients should have the head elevated, receive 100% oxygen and have secure intravenous access. If the patient remains stable, the doctor must alert an anaesthetist, make arrangements to transfer the patient

Table 1. Types of thyroid surgery

Types of thyroid surgery	Indications
Total thyroidectomy	Graves disease, multinodular goitre, thyroid cancer
Total thyroidectomy + central compartment lymph node dissection	Thyroid cancer
Subtotal thyroidectomy (rarely performed)	Graves disease
Total lobectomy	Multinodular goitre, small thyroid cancers (<1 cm)
Isthmusectomy	Small isthmic nodules

Table 2. Symptoms and signs of neck haematoma

Neck swelling and pain
Rapid or significant accumulation of blood in any neck drains (<30 ml in 24 hours is normal)
Dysphagia
Respiratory distress and airway obstruction (stridor, hypoxia, tachypnoea, confusion)

urgently back to theatre, and stay with the patient in case of rapid deterioration. If the airway obstruction is critical (or if there is any doubt), the patient's neck must be re-opened immediately on the ward, before returning to theatre (*Table 3*). Ideally this should be performed by a senior surgeon with suitable experience. However, if not immediately available and the patient has an obstructed airway this must be performed by the treating junior doctor (*Table 3*).

Bilateral recurrent laryngeal nerve palsy

The presence of stridor soon after extubation, in the absence of a haematoma or collection, raises the concern of bilateral recurrent laryngeal nerve injury. Urgent flexible laryngoscopy should be performed to assess the position and mobility of the vocal cords. If bilateral recurrent laryngeal nerve palsy is confirmed or suspected, patients should be reintubated and transferred to the intensive care unit. High dose steroids (dexamethasone 8 mg/day) are administered to encourage recovery of a transient neuropraxia, which is a likely mechanism in patients in whom the recurrent laryngeal nerves appeared intact to the operating surgeon. Within 48–72 hours a second attempt should be made to extubate and repeat laryngoscopy be performed. Early tracheostomy should be discouraged as it is seldom necessary. If local ear, nose and throat expertise allows, vocal cord lateralization procedures should be considered.

Tracheomalacia

Although a historically feared complication, emphasized routinely in undergraduate and postgraduate training, tracheomalacia is now almost mythical in modern thyroid surgery. It may be a consequence of prolonged tracheal compression by goitres (particularly retrosternal), with loss of cartilaginous rigidity and unheralded airway collapse and obstruction following removal of large goitres and subsequent extubation. However, contemporary experience of this complication is almost unheard of and limited largely to areas with chronic and endemic goitre (Bennett et al, 2004; Agarwal et al, 2007; Findlay et al, 2011).

Should tracheomalacia be encountered, it should be recognized intraoperatively (by the 'floppy' consistency of the tracheal cartilages) or during extubation with more than 50% of dynamic tracheal luminal collapse (Kandaswarmy and Balasubramanian, 2009). It warrants reintubation, transfer to the intensive care unit and re-evaluation after several days. If severe airway compromise is recognized some hours or days after thyroidectomy, tracheomalacia is not the cause of it and other mechanisms should be looked for (such as neck haematoma or bilateral recurrent laryngeal nerve injury).

Postoperative laryngeal oedema

Direct trauma to the vocal cords and laryngeal mucosa during a difficult intubation can lead to a degree of local oedema that presents with airway obstruction and respiratory distress. This mechanism should be considered in the absence of obvious neck swelling and should be confirmed by laryngoscopy. Having alerted an anaesthetist, intravenous steroids (e.g. dexamethasone) may be required.

Hypocalcaemia

Hypocalcaemia is one of the commonest postoperative problems encountered by junior doctors on the ward, again occupying a spectrum of severity. It is precipitated by inadvertent excision of, or damage (such as devascularization) to, the parathyroid glands with resultant iatrogenic hypoparathyroidism. There are usually four parathyroid glands – two superior and two inferior – situated in close proximity to the thyroid. Comparable in size to a grain of rice, and a similar browny-yellow colour to adipose tissue, the parathyroids are well 'camouflaged' and therefore vulnerable. When only a thyroid lobectomy is performed the contralateral parathyroid glands are safe and remain functional, so these patients are not at risk of developing postoperative hypocalcaemia.

Symptoms and signs of hypocalcaemia are detailed in *Table 4* and can be recognized within 12–24 hours of total thyroidectomy. Calcium is an ubiquitous intracellular regulator of cyclic AMP-mediated systems, and consequently hypocalcaemia impairs the functioning of both cell

Table 3. Re-opening a neck wound on the ward

Use sterile gloves (provided they are rapidly available)
Remove skin sutures or staples (either cut the end of the suture and pull, or use a staple remover)
Insert a finger through the next layer: the sutured platysma. This may be enough to decompress the haematoma. If not possible, cut the stitches with scissors or a stitch cutter
Divide the midline sutures holding the strap muscles so that the trachea is exposed (you should feel the rings of the trachea)
The goal is to evacuate the haematoma and prevent airway loss, not to achieve haemostasis. Do not be tempted to clip possible bleeding points – these will be dealt with after return to theatre

Table 4. Symptoms and signs of hypocalcaemia

Circumoral and peripheral paraesthesia
Tetany
Carpopedal spasm
Chvostek's sign (facial muscle spasm on tapping the facial nerve)
Trousseau's sign (fingers, hand and forearm muscle spasm on inflation of a blood pressure cuff to occlude systolic blood flow for > 3 minutes)
Confusion and altered mental state
Prolonged QT interval on electrocardiogram

organelles and the cell as a whole. Approximately 99% of total body calcium is stored in bone, with just 1% in the extracellular fluid compartment; 50% of this is free and physiologically active, with the remainder complexed with albumin, proteins (largely albumin) and non-protein anions. Acute severe hypocalcaemia is a life-threatening condition because of the cardiorespiratory system's intrinsic reliance upon calcium. Abrupt withdrawal of parathyroid hormone-mediated mobilization of calcium from bone stores may precipitate bronchospasm, myocardial depression, a prolonged QT interval (evident on electrocardiogram) and the potential to develop arrhythmias (particularly ventricular tachycardias).

Immediately after total thyroidectomy the risk of hypocalcaemia is approximately 33%, but the majority (73%) of these patients will recover normal calcium homeostasis within 1 week. Between 1 and 10% of all patients require long-term calcium supplementation (British Association of Endocrine and Thyroid Surgeons, 2009; Youngwirth et al, 2010). The risk is higher in patients with low vitamin D levels, those with Graves' disease, and those undergoing central compartment lymph node dissection for thyroid cancer (Roh et al, 2009). However, it can be reduced by auto-transplanting inadvertently removed glands into the sternocleidomastoid muscle (Kikumori et al, 1999).

The risk of hypocalcaemia can be better predicted by measuring serum parathyroid hormone concentration within 1–12 hours after thyroidectomy, or by using the trend in calcium levels within the first 24 hours (Wang et al, 2011). It remains contentious exactly whether treatment should be instituted based on the onset of symptoms or the absolute serum concentration of calcium. However, in general biochemical hypocalcaemia should be treated, irrespective of the presence or absence of symptoms (Adler et al, 2008). Consequently, many units offer routine calcium supplementation for 2 weeks postoperatively and patients are discharged with information about how to recognize symptoms of hypocalcaemia and how to decide on reducing the daily dose of calcium.

Oral calcium tablets (e.g. Calcichew 1 g four times a day) can be used to treat the vast majority of patients. If instituted early enough oral calcium therapy is sufficient to treat even extreme hypocalcaemia so that intravenous calcium gluconate is seldom required. In acute severe hypocalcaemia 10 ml of 10% calcium gluconate can be injected slowly through a large bore cannula. Vitamin D supplementation is not beneficial in the immediate postoperative period (it can inhibit the proliferation of any remaining parathyroid tissue) but can be used as treatment for permanent hypoparathyroidism (Roh et al, 2009).

Recurrent laryngeal nerve injuries

Traditionally the risk of recurrent laryngeal nerve injury has been quoted to be 1–2%, based on historical data from large surgical units with extensive experience or from cohort studies in whom postoperative laryngoscopy was performed only in patients with significant voice changes. An increased awareness and routine use of postoperative laryngoscopy has demonstrated that temporary recurrent laryngeal nerve palsy can occur in up to 1 in 10 patients (Frattini et al, 2010).

Implementation of the 2009 guidelines from the British Association of Endocrine and Thyroid Surgeons recommend postoperative laryngoscopy in all patients has the potential to provide a more up-to-date and realistic estimate of the recurrent laryngeal nerve injury rate. With a recurrent laryngeal nerve injury the ipsilateral vocal cord lies adducted; the contralateral cord may be unable to compensate, and the voice becomes breathy and hoarse. Owing to a lack of laryngeal sensation, patients may also aspirate fluids or saliva. Management involves speech therapy, sometimes consolidated by medialization of the affected vocal cord (Laccourreye et al, 1999).

Superior laryngeal nerve injuries

The external branch of the superior laryngeal nerve is encountered in relation to the superior thyroid artery, as it travels to innervate the cricothyroid muscle. Dividing the artery close to the gland helps reduce the risk of injury. Trauma to the external branch of the superior laryngeal nerve results in an inability to lengthen the vocal fold and, therefore, to create a higher pitched sound and vocal projection. The clinical presentation may be quite subtle in many patients, with increased tendency for vocal fatigue and decreased pitch range being the most common symptoms. For the singer or the professional voice user, however, paralysis of the external branch of the superior laryngeal nerve may be career threatening because of the loss of the upper register of the voice (Lombardi et al, 2006; Pagedar and Freeman, 2009). While the true incidence of injury is not known (because of the subtlety of its functional consequences) some degree of dysphonia occurs in a significant proportion of patients. Treatment is limited to speech therapy.

Voice changes in patients with normal recurrent laryngeal nerve function

In recent years it has become increasingly recognized that voice changes occur in some 30% of patients undergoing thyroid surgery despite having a normal recurrent laryngeal nerve function. Voice fatigue, loss of voice projection, and a more monotone voice can be identified when using patient-administered voice quality questionnaires (e.g. voice handicap index) or by using computerized voice analysis. In addition, many such patients have swallowing difficulties.

Infection and collection

Infection may be superficial (limited to the wound) or deep. As a 'clean' operation, thyroidectomy wound infection rates are minimal (Efremidou et al, 2009). However, significant deep collections may occur (particularly in the potential spaces afforded by the removal of large goitres), and can cause local compression of, for example, the airway. Wound cellulitis (indicated by erythema, warmth, tenderness and discharge) is managed with appropriate antibiotics. However, deep neck abscesses usually require drainage. Seromas (sterile collections of serious fluid) may also require drainage, possibly under ultrasound guidance.

Rare complications

Thyrotoxic storm

Modern medical management of hyperthyroidism has rendered thyrotoxic storm (extreme thyrotoxicosis) exceptionally

rare. However, it may occur following surgery on thyrotoxic patients and be life-threatening, requiring a low threshold of suspicion (*Table 5*). Treatment must be instituted rapidly, with timely involvement of endocrine specialists and critical care physicians. Such treatment is supportive and involves beta blockade (for example, with propranolol 80 mg), supplemented by dexamethasone, methimazole and Lugol's iodine (Kearney and Dang, 2007).

Pneumothorax

This can occur if there has been extensive retroclavicular dissection.

Chyle leak

Injury to thoracic duct can occur during left lateral radical neck dissection (for cancers with extensive metastatic disease) but it is rare after thyroidectomy for large retrosternal goitres. Small volume chyle leak (<100–200 ml) can be treated conservatively by maintaining a low-fat diet for 1–3 weeks. Large volume leaks (>500 ml/day) mandate re-exploration and ligation of the injured duct. Clinical experience helps decide the care of individual cases.

Table 5. Symptoms and signs of thyrotoxic storm

Pyrexia (>38.5°C)
Tachycardia
Hypertension
Tremor
Confusion
Nausea and vomiting
High output cardiac failure

Conclusions

Modern surgical and perioperative technique has rendered significant morbidity and mortality rare. Despite this, complications of thyroid surgery do occur and may progress rapidly with disastrous consequences. As many surgical junior doctors deal with thyroid surgical patients, and are likely to be first port of call in the case of complications, it is important to have a working knowledge of them, in order to deal safely with emergencies. However, a low threshold of suspicion and timely intervention (to the level discussed in this article) means that they can be dealt with simply safely. BJHM

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KEY POINTS

- Significant complications following thyroid surgery are rare, but may be life-threatening.
- Junior doctors, despite having minimal experience, are often required to deal with these.
- The most serious complication is airway compromise, which may be caused by haemorrhage, collection, bilateral recurrent laryngeal nerve injury, laryngeal oedema or tracheomalacia.
- Recent moves towards routine laryngoscopy and nerve monitors have revealed a greater incidence of subtle nerve damage than previously realized.
- Postoperative hypocalcaemia is common, but usually reversible and manageable with temporary oral calcium supplements.