Paparella: Volume III: Head and Neck

Section 2: Disorders of the Head and Neck

Part 6: The Thyroid Gland

Chapter 41: Surgical Therapy and Pathologic Features of the Parathyroid Glands

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The diagnosis and treatment of parathyroid disease has become a routine clinical duty for the contemporary otolaryngologist and head and neck surgeon. It is therefore essential for the surgeon who treats this disease to have a clear conceptual understanding of the pathologic characteristics and proper surgical approach to this often confusing subject. The embryology, anatomy and physiology have been discussed in detail elsewhere in this text. The focus of this discussion will be key points that have an impact on clinical management.

Historical Perspectives

The parathyroid glands were first described in the rhinoceros in 1862 by Owen, and Virchow in 1863 was the first to describe this gland in humans, but it was Sandstrom in 1880 who presented the first relatively complete comparative description of the gland and gave it its present name. In 1891, von Recklinghausen recognized the clinical impact of hyperparathyroidism when he described the rather severe, although rare disease of bone, "osteitis fibrosa cystica". Askanazy made the association between bone disease and a parathyroid tumor (an adenoma) in 1903 at autopsy in a patient with von Recklinghausen's disease. In 1925, Mandl, a Viennese surgeon, removed a parathyroid tumor from a patient with hypercalcemia, hypercalciuria, and radiologic changes of osteitis fibrosa cystica. Postoperative regression of bone and biochemical abnormalities were demonstrated, though a recurrence of the disease in retrospect suggests this was a case of chief cell hyperplasia. In the USA, similar progress was being made in the elucidation of parathyroid disease. Dubois is credited with making the first diagnosis of primary hyperparathyroidism in America (1929) in Captain Charles Martell on the basis of bone disease, high serum calcium levels, and negative calcium balance. On the seventh parathyroid exploration, Dr. Edward Churchill of Boston found a retrosternal parathyroid adenoma. The physiologic basis of hyperparathyroidism was detailed by Albright. His group's elegant metabolic studies recognized the presentation of primary hyperparathyroidism in patients with renal stones but without overt bone disease. The combined efforts of these investigators helped to establish the parathyroid gland as the central agent in these distinct clinical pathologic entities. Beginning with these studies and numerous subsequent ones, hyperparathyroidism has been recognized as a common, ubiquitous disease with a wide variety of clinical signs and symptoms.

Epidemiology

As noted in the preceding historic discussion, hyperparathyroidism was once considered to be a rare disease. Through extensive screening programs, it has been

appreciated to be the leading cause of hypercalcemia in nonhospitalized patients and the second most common cause of hypercalcemia (after malignancy) in hospitalized patients. Epidemiologic studies report overall incidence rates of 25 to 28 cases/100,000 population/year. The annual incidence rate increases sharply after the age of 40 years, and after 60 years it reaches almost 200 cases/100,000 population in women and 100 cases/100,000 population in men. More than 60,000 new cases of primary hyperparathyroidism occur each year in the USA; of newly diagnosed patients 85 per cent are between the ages of 30 and 70 years. Most studies suggest that at all ages, females outnumber males by a factor of 2:1.

Clinical Manifestations and Differential Diagnosis

The signs and symptoms of hyperparathyroidism may be separated into those caused directly or indirectly by the hypercalcemia or increased parathyroid hormone levels, to both, or to associated conditions. The majority of patients with hyperparathyroidism are relatively asymptomatic, although symptoms may involve many organ systems. The six "moans" of hyperparathyroidism form a convenient means of recall: (1) stones, (2) bones, (3) groans (abdominal symptoms), (4) ions (asymptomatic hypercalcemia), (5) neurones (neuromuscular and psychiatric presentations), and (6) hormones (part of multiple endocrine neoplasia [MEN] syndromes). Tables 1 and 2 list the common clinical manifestations and associated conditions of hyperparathyroidism.

Parathyroid tumors are palpable in only about 5 per cent of patients with primary hyperparathyroidism. Palpable neck masses are usually coexistent thyroid disease. When present, palpable parathyroid tumors are found in 50 per cent of patients with parathyroid cancer and should alert the clinician. The coexistent thyroid disease in patients with primary hyperparathyroidism (25 per cent of patients) may pose additional therapeutic dilemmas because of the 5 per cent incidence of papillary thyroid cancer.

The most common physical signs in patients with hyperparathyroidism are hypertension, arthritis, band keratopathy, and neuromuscular disorders. Classically described bone disease, osteitis fibrosa cystica, includes radiologic "demineralization" as characterized by a moth-eaten appearance on skull radiograph, subperiosteal resorption (best seen on the radial aspect of middle phalanges), and the presence of true bone cysts and brown tumors.

Differential diagnosis involves distinguishing the hypercalcemia of parathyroid dysfunction from other causes. Three common causes of hypercalcemia that should be considered include malignancy, sarcoidosis, and thiazide therapy. Table 3 comprises a more complete list. Of particular note is familial hypocalciuric hypercalcemia, which may be difficult to differentiate from primary hyperparathyroidism and for which surgery is not indicated.

The significance of these facts have translated into clinical controversy as to the appropriate treatment of asymptomatic primary hyperthyroidism. The Mayo Clinic series showed that complications will develop in 26 per cent of asymptomatic patients in 10 years. Additional data support that asymptomatic patients receive the same metabolic benefits as do symptomatic patients and that once complications do arise (mainly renal dysfunction) they may be irreversible once established. The low mortality and morbidity rates of surgical

treatment, as well as its cost-effectiveness, places it as the current treatment modality of choice for parathyroid dysfunction, although some studies suggest that carefully followed untreated patients may not have more serious complications than treated ones.

Table 1. Symptoms Associated With Hyperparathyroidism

General	Polydipsia, weight loss
Renal	Colic, hematuria, polyuria, renal failure
Musculoskeletal	Bone and joint pain, arthralgias, muscle weakness
Gastrointestinal	Constipation, anorexia, nausea, emesis
Neurologic	Depression, lethargy, weakness, recent memory loss, confusion, insomnia, headaches, apathy
Cardiovascular	Hypertension.

Table 2. Associated Conditions of Hyperparathyroidism

Nephrolithiasis or nephrocalcinosis
Hypertension
Hyperuricemia with or without gout
Pseudogout
Arthritis
Pancreatitis.

Table 3. Differential Diagnosis of Hypercalcemia

Malignant tumors involving bone (myeloma, lymphoma, leukemia)
Malignant tumors not involving bone (lung, hypernephroma, extensive squamous cell carcinoma)
Granulomatous disease (sarcoid, tuberculosis, berylliosis)
Endocrine dysfunction (thyrotoxicosis, hypothyroidism, acute adrenal insufficiency)
Excessive calcium, vitamin D, or vitamin A ingestion
Milk - alkali syndrome
Thiazide diuretics
Paget's disease
Familial hypocalciuric hypercalcemia
Idiopathic hypercalcemia of infancy
Laboratory error.

Surgical Anatomy

The normal parathyroid glands weighs 35 to 60 mg. The normal dimensions are 3 to 6 mm in length, 2 to 4 mm in width, and 0.5 to 2 mm in thickness, with the average dimensions of 5x3x1 mm. Glandular weight is accepted as a better measure of size than glandular dimensions. In the operating room, enlargement is determined by size, shape, and color rather than by weight, since the latter determination requires removal of the glands.

The normal gland is well encapsulated by a thin, colorless capsule with a fine capillary network. The normal gland parenchyma is yellow-tan to orange-tan, depending upon the

amount of stromal fat, vascular congestion, and oxyphilic cells. The adult gland is composed of chief and oxyphilic cells and a fibrous stroma with varying amounts of fat cells, depending on the patient's age and amount of body fat. The chief cell is polyhedral and poorly outlined and is 6 to 8 microns in diameter. The cytoplasm is slightly eosinophilic, the nuclear chromatin is well defined and abundant, and the nuclear membrane is sharply outlined, giving a pyknotic appearance. Close to the time of puberty, the oxyphilic cells appear. They increase in number with advancing age, often forming large islands and nodules that may be incorrectly interpreted as neoplastic. The oxyphilic cells are 8 to 12 microns in diameter, with well-demarcated cell membranes, brightly eosinophilic granular cytoplasm, and pyknotic, centrally located nuclei. Ultrastructurally, the cytoplasm is filled with tightly packed mitochondria.

The superior parathyroid glands arise from the fourth branchial pouch and share the embryoanatomic region with the ultimobranchial complex, which gives rise to calcitonin-secreting C-cells of the thyroid gland. They normally descend to positions adjacent to the posterior aspect of the thyroid capsule of the thyroid gland, near the point at which the medial thyroid vessels cross the recurrent laryngeal nerve at the cricothyroid junction (superior or upper parathyroid glands). In Wang's series, the superior parathyroid glands were rarely found to be ectopic on the anatomic basis of parathyroid surgery, yet if they overdescend they may be found in the upper posterior mediastinum. Failure of complete descent places them in the region of laryngeal structures.

The inferior parathyroid glands arise from anterolateral portions of the third branchial pouch with the thymus, and they normally descend to an area near the lower pole of the thyroid gland in the inferior aspect of the neck. The extent of migration during embryogenesis is responsible for the variability in anatomic distribution. When the inferior glands overdescend they may be found in the tracheoesophageal groove or in the anterior mediastinum, not infrequently adjacent to or in the thymus. Ectopic or supernumerary glands are present in 2 to 8 per cent of patients and may be found in any region of the neck and thorax.

Surgical Pathological Features

Primary Hyperparathyroidism

Primary hyperparathyroidism results from an excess of parathyroid hormone. This excessive hormone production is commonly due to the presence of an adenoma (single or multiple), carcinoma, or primary hyperplasia (a hyperplasia for which there is no external stimulus). The accumulated data from many series of patients with primary hyperparathyroidism confirm that approximately 85 per cent of cases have an adenoma as the cause. Hyperplasia and carcinoma are the histologic diagnoses in about 15 per cent and less than 1 per cent of the cases, respectively (Table 4).

Table 4. Relative Frequency of the Three Causes of Primary Hyperparathyroidism at the Massachusetts General Hospital 1930-1976.

Cause		Frequency
Adenoma		598 (81.7%)
Single	591 (80.7%)	
Double	7 (1.0%)	
Hyperplasia		113 (15.4%)
Chief cell	94 (12.8%)	
Clear cell	19 (2.6%)	
Carcinoma		21 (2.9%)*
Total		732.

* Carcinoma accounts for approximately 1 per cent of cases of primary hyperparathyroidism after exclusion of referral cases.

Adenoma

A parathyroid adenoma is characterized by enlargement of one parathyroid gland, occasionally with a recognizable rim of normal tissue and usually a proliferation of chief cells. An adenoma is usually an encapsulated, orange-brown, soft, lobular mass. Mediastinal adenomas will occasionally have a fibrous stalk. Degenerative parenchymal changes may stimulate inflammation, resulting in fibrosis and adherence of the tumor's capsule to the surrounding tissue, more frequently observed with carcinoma. Rarely, an adenoma degenerates into a large cystic mass and presents clinically as a colloid goiter or thyroid cyst.

The use of normal rim in diagnosis is not a reliable criterion, since in primary hyperplasia, hyperplastic nodules may expand to simulate a rim.

Microscopically, the cell composing the bulk of the adenoma is an enlarged chief cell. In standard hematoxylin and eosin (H & E) preparations, the cytoplasm appears pale pink, granular, and occasionally vacuolated. In many cases, only one type of cell is present in the adenoma, but often there is marked variation in size, type, and arrangement of the cells. Individual and group chief cells may be vacuolated and, therefore, mistaken for cells of clear cell hyperplasia. The cells in the latter disorder are much larger and contain numerous small, membrane-lined vacuoles, whereas the vacuolated cells in the adenoma are the result of dissolution of cytoplasmic contents during tissue preparation. Oxyphilic and transitional oxyphilic cells with a brightly eosinophilic granular cytoplasm are present individually and in groups. The transitional oxyphilic cell may be the predominant or only cell type present in a functioning adenoma; a functioning adenoma composed entirely of true oxyphilic cells has been reported. Intracellular fat is uncommon in chief cells and oxyphilic cells of adenomas.

Perhaps the most useful histologic feature of adenomas is pleomorphism. The nuclei of adenoma chief cells are larger and hyperchromatic when compared with normal chief cells. Most adenomas are solid, with cells arranged in large islands or broad bands. A vascular stroma is present, and small foci of fat cells not yet replaced by adenomas may be present. The cells are often arranged around a blood vessel in a pseudoglandular formation and, at

times, seem to form acini. A definitive separation between an adenoma and chief cell hyperplasia can be made only by confirming the presence of a separate normal gland by histologic examination of a biopsy.

Lipoadenoma

Rarely the cause of primary hyperparathyroidism is the lipoadenoma or hamartoma. The microscopic appearance reveals large lobules of fat, with cords and compressed sheets of parathyroid chief and oxyphilic cells. Areas of mucinous stroma may be seen between the cells. Analogies to thymolipomas of the mediastinum have been made.

Hyperplasia

Chief Cell Hyperplasia

Fifteen to 20 per cent of cases of primary hyperparathyroidism are caused by chief cell hyperplasia. About 25 to 35 per cent of individuals with this lesion have familial hyperparathyroidism or MEN syndrome. Grossly, all four glands are enlarged, although not always equally. The combined weight of all four glands may range from 150 mg to more than 10 gm, with the majority around 1 gm. Lobulation, orange-brown color, and homogenous appearance are characteristic.

The microscopic appearance of cords, sheets, and a follicular arrangement of the cells replacing the stromal fat cells characterizes the classic pattern. The cells may be pure chief cells or, rarely, almost pure oxyphilic cells. A mixture of chief cells, oxyphilic cells, and transitional oxyphilic cells is common. The cells are often grouped together in large islands or even nodules; this pattern has prompted the use of the terms *nodular hyperplasia* and *multiple adenomatosis.* Since the adenoma may have an identical cellular pattern and nodularity, it is not possible to determine microscopically whether a single enlarged parathyroid gland is involved by an adenoma or by chief cell hyperplasia.

Each nodule is devoid of fat, and little fat is found in the intervening stroma. The amount of stromal fat in the different glands of the same patient may vary greatly. When all the fat cells have not been replaced, it may be very difficult to distinguish a partially involved hyperplastic gland from a normal gland; it is only when two or more glands in a given patient are obviously hyperplastic that one can easily make the diagnosis of chief cell hyperplasia in the presence of glands with residual fat cells.

Ultrastructural studies have disclosed active-appearing chief cells with large Golgi complexes, secretory vacuoles, and prominent endoplasmic reticulum - virtually identical in appearance to chief cells in adenomas. In most cases, intracellular fat is decreased or nonexistent at the ultrastructural level and by fat stain.

Clear Cell Hyperplasia

The pathologic entity of clear cell hyperplasia is distinct from chief cell hyperplasia. Grossly, the superior glands are usually larger than the lower glands. Total weights of involved glands exceed 1 gm and are usually 5 to 10 gm. They not uncommonly approach

50 gm.

Histologically, clear cell hyperplasia is characterized by a uniform distribution of large cells with amphophilic cytoplasm. In most cases, all four parathyroid glands are completely replaced by these large clear cells, without fat cells in the stroma. Little nodularity or stromal fibrous separation of cells is seen. The cytoplasm of the clear cells is actually not "clear" but composed of numerous vacuoles that are easily recognized by histologic preparation.

Parathyroid Carcinoma

Carcinoma of the parathyroid gland constitutes approximately less than 1 per cent of cases of primary hyperparathyroidism. Clinically the average age at presentation is 44 years with an equal sex ratio. The most common manifestations at presentation are attributed to the elevated calcium levels, averaging 15.2 mg/dL, and its effect on other organs. Bone or pathologic fracture, nephrolithiasis, and a palpable neck mass are important clinical cues.

As described by Schantz and Castleman, these tumors tend to be larger than adenomas (average weight is 12 gm). The microscopic features of parathyroid carcinoma include a trabecular arrangement of tumor cells divided by cellular fibrous bands, nuclear pallisading, mitotic figures, and capsular and blood vessel invasion. All these criteria are not invariably found in each tumor, but several are usually present. Mitosis within parenchymal cells is cause for suspicion of malignancy.

Metastases at the time of presentation are unusual, but when present may be found in local regional lymph nodes, lung, liver, and bone. It is more common to find local visceral invasion. The initial surgical approach should be wide en bloc resection, since the primary therapy determines prognosis. The indications for neck dissection are not clear unless extensive local disease is present. The course of the disease is usually indolent, with approximately 50 per cent survival at 5 years. Multiple recurrences of disease are not uncommon. Recurrence is heralded by reappearance of hyperparathyroidism, and death is usually secondary to complications of hypercalcemia (heart, kidney) rather than widespread visceral involvement.

Parathyroid Cysts

Cysts of the parathyroid glands are unusual lesions, frequently presenting as thyroid nodules. More common in women, the cysts are usually large (1 to 6 cm) and are more commonly associated with the lower glands. The origin of cysts has been attributed to embryologic remnants of pharyngeal pouches or the end result of cystic degeneration of a parathyroid adenoma.

Secondary Hyperparathyroidism

Hypocalcemia caused by intrinsic renal disease, vitamin D deficiency, or intestinal malabsorption of calcium stimulates the parathyroid glands, which in turn, enlarge and secrete increased amounts of parathyroid hormone. The most common cause is renal disease, with secondary hyperparathyroidism and osteodystrophy developing in up to 90 per cent of patients with chronic renal failure. Grossly, all glands are enlarged with the histologic appearance of

hyperplasia, usually of a nodular type, with decreased fat. Progression of bone disease, persistent hypercalcemia, intractable medical therapy, and progressive soft tissue calcifications are the main clinical indications for surgical treatment. Various surgical approaches have been proposed: (1) subtotal parathyroidectomy, (2) total parathyroidectomy, and (3) total parathyroidectomy with autotransplantation. The current trend is to favor total parathyroidectomy with forearm autotransplantation. The reader is referred to Sicard and Wells for an excellent review of this subject. A successful kidney transplant allows for normalization of renal phosphate metabolism, 1,25-dihydroxy-vitamin D_3 production, and regression of hyperplastic parathyroid glands. Persistent postrenal allograft hypercalcemia and hypercalciuria despite medical management requires surgical therapy.

Familial Hyperparathyroidism

Multiple endocrine adenomatosis I (MEN I), also known as Wermer's syndrome, consists of coexisting hyperparathyroidism, pituitary adenomas, and pancreatic islet neoplasms. Originally the parathyroid lesions were called adenomas; however, subsequent review has established the lesion to be parathyroid chief cell hyperplasia. The parathyroid glands are involved in about 90 per cent of cases. The syndrome is transmitted as an autosomal dominant trait with high penetrance and variable expression.

Unlike patients with nonfamilial hyperparathyroid disease, most patients with MEN I have evidence of hyperparathyroidism before the third or fourth decade of life. Therefore hyperparathyroidism may be the initial presentation of the syndrome and warrants careful follow-up or endocrine evaluation, or both. Conversely, new patients with the Zollinger-Ellison syndrome or insulinoma should have serum calcium and parathyroid hormone levels determined. Surgical management consists of subtotal parathyroidectomy with careful marking of the remaining gland for autotransplantation. Of particular note is the increase of supernumerary glands, approximately 15 per cent. Failure to identify a fifth gland during parathyroidectomy (normally in the upper portion of the thymus) will lead to recurrent hypercalcemia postoperatively.

Multiple endocrine adenomatosis type II (MEN II; Sipple's syndrome) is characterized by the familial association of medullary thyroid carcinoma, pheochromocytoma, and hyperparathyroidism. It was noted by Chong and co-workers that there were two phenotypically distinct patient populations with medullary carcinoma and pheochromocytoma; the designation of MEA IIa was given to the population having normal appearance, and MEA IIb was given to those patients with characteristic facies, marfanoid habitus, and submucosal neuromas. Carney and co-workers observed that parathyroid disease was common in MEA IIa but not in MEA IIb.

Parathyroid involvement in MEA IIa consists of chief cell hyperplasia, and glandular involvement is often asymmetric. Approximately 80 per cent of patients will have parathyroid hyperplasia; 20 per cent become hypercalcemic; and symptoms (most commonly nephrolithiasis) will develop in only half of these. Operative management is different from that for MEA I. Subtotal parathyroidectomy (three and one-half glands) has a high incidence of permanent hypocalcemia, and therefore only excision of grossly enlarged glands should be performed. This approach will commonly preserve one or two glands without recurrent hypercalcemia or hypocalcemia developing.

Rare Syndromes

Hereditary neonatal hyperparathyroidism, familial hypocalciuric hypercalcemia, familial parathyroid carcinomas, and familial parathyroid adenoma are three unusual syndromes that must be included for completeness. As already noted, familial hypocalciuric hypercalcemia should be considered in adult patients, since surgical intervention is not indicated.

Operative Strategy, Localization, and Reoperation

The main problem in parathyroid surgery is the variation in the location of normal and abnormal glands. Success rates of 95 per cent have ben achieved in primary surgical procedures and should be attainable by surgeons who fully appreciate the embryology, anatomy, and gross identification of parathyroid tissue. Additionally, the team approach utilizing experience, judgment, and skills of the surgeon, pathologist, and endocrinologist enhances therapy results. The primary goal of the surgeon should be the attainment of a permanent euparathyroid state, without complications, and the removal of as little parathyroid tissue as possible. In the cases caused by adenomas or single gland enlargements (80 per cent of case), the main controversy concerns the number of glands to be identified. Wang suggests that the exploration can be halted if, on the first side to be explored, the adenoma and a normal gland are identified. Other surgeons suggest that it is necessary to explore both sides of the neck. The difference when an experienced parathyroid surgeon and pathologist are present is not significant, nor is the difference in complication rate.

Identification and histologic confirmation of all glands is mandatory, although some authors have recommended ipsilateral subtotal thyroidectomy if three normal glands are found after exploration, since intrathyroidal glands are present in up to 6 per cent of cases. Thyroid lobectomy should be performed only after careful search for the fourth gland in the anterior superior mediastinum, tracheoesophageal groove, retroesophageal space, or carotid sheath. After identification of four glands, frozen sections should be obtained to confirm parathyroid tissue. If the glands are normal in appearance, their location should be marked with surgical clips and the anterosuperior mediastinum evacuated and the operation ended. If the glands are clinically enlarged and hyperplastic, a subtotal parathyroidectomy (three and one-half glands, leaving approximately 150 mg of parathyroid tissue) should be performed, with cryopreservation of the removed parathyroid tissue. Some investigators have proposed total parathyroidectomy and autotransplantation. This approach remains controversial. The previously outlined surgical plan will achieve successful primary management.

Some comments in regard to preoperative localization are necessary. It is evident that careful history, physical examination, and laboratory studies followed by a systematic surgical approach will permit success in 95 per cent of patients treated initially for primary hyperparathyroidism. Use of selective angiography, with or without selective venous sampling, high-resolution ultrasound, computed tomography, nuclear scintigraphy, and magnetic resonance imaging, only adds to the total medical costs and appears to be unwarranted in the management of first-time cases. In the management of recurrent or persistent hyperparathyroidism, some feel that these studies are useful. Brennan and colleagues have summarized their experience in 106 patients. Approximately 14 per cent had persistent hypercalcemia following reoperation, the causes of which are detailed in Table 5.

Table 5. Results of Reoperation for Primary Hyperparathyroidism (N = 106)

Cause	N	%
Hypercalcemia	15	14
Probable FHH	4	
Not primary HPT	3	
Parathyroid Ca	3	
MEA I	2	
Primary HPT	2	
Familial HPT	1	
Hypocalcemia	55	53
Eucalcemia	36	34.

FHH, familial hypocalciuric hypercalcemia;
HPT, hyperparathyroidism;
MEA I, multiple endocrine neoplasia, type I.

Complications of Parathyroid Surgery

Transient hypocalcemia following parathyroidectomy, although common, usually does not require calcium or vitamin D supplementation. Serum calcium levels should be checked 12 hours after surgery and then every 8 to 12 hours until stabilization at 8.0 mg/dL or greater. If significant circumoral numbness, paresthesias, or a positive Trousseau sign develops, immediate calcium supplementation should be initiated after obtaining a blood sample for documenting serum calcium levels. Prompt institution of supplementation is important to avoid the extreme symptoms of hypocalcemia, such as tetany, laryngeal spasm, and respiratory arrest. Certain groups of patients may require prolonged calcium and vitamin D supplementation. They include patients with primary hyperparathyroidism with significant bone disease and elevated serum alkaline phosphatase levels, patients undergoing total parathyroidectomy and autotransplantation, and uremic patients with secondary hyperparathyroidism. Additionally, hypomagnesemia can occur in patients with primary hyperparathyroidism and can be responsible for persistent and refractory hypocalcemia, which will resolve after magnesium supplementation.

The recurrent laryngeal nerve is at risk during parathyroid surgery, although permanent injury is rare (less than 1 per cent). Bilateral recurrent nerve injury may necessitate immediate intubation or tracheotomy.