



(Print pagebreak 655)

CHAPTER 7.11

Endocrine Surgery

Mark Koransky, MD
Eliza Long, MD
Ralph S. Greco, MD
Frederick G. Mihm, MD
(Print pagebreak 656)

Excision of Thyroglossal Duct Cyst

Surgical Considerations

Description: Thyroglossal duct cysts typically are located in the midline at or below the hyoid bone ([Fig. 7.11-1](#)). Differential diagnoses after initial physical exam include epidermoid cysts or lymph nodes. Cysts should be removed because of an associated high risk of infection with oral flora and a slight (< 1%) risk of either squamous-cell or papillary-thyroid cancer developing in the cyst itself. A transverse skin incision is made over the cyst; and, if the cyst was previously infected and sinus tracts through the skin are present, the skin should be removed along with the cyst. The cyst is identified and followed cephalad to the hyoid bone ([Fig. 7.11-2](#)). Then, the midportion of the hyoid bone is resected to minimize recurrence. There may be many small tracts associated with the cyst that tend to attenuate beyond the hyoid bone. The base of the tract or tracts is resected up to the level of the floor of the mouth at the foramen cecum and ligated with absorbable suture (**Sistrunk procedure**). The wound is irrigated copiously and closed in layers. The wings of the hyoid bone are not reapproximated.

Usual preop diagnosis: Thyroglossal duct cyst

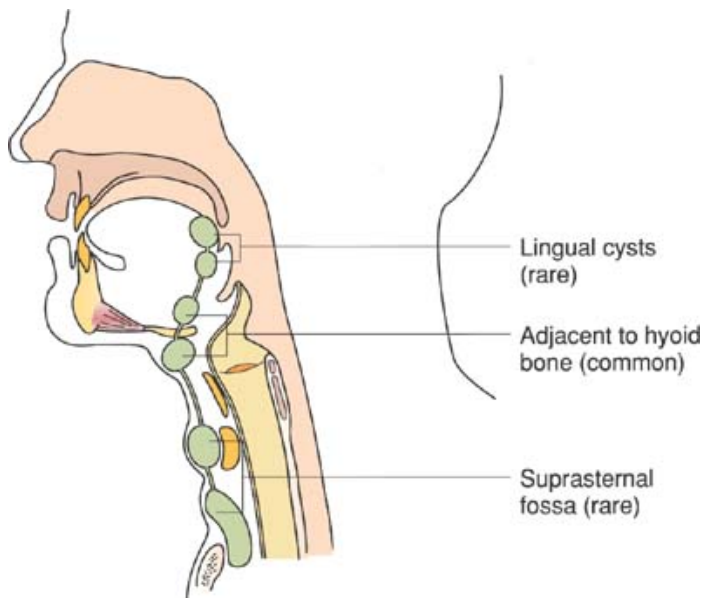


Figure 7.11-1. 1. Location of thyroglossal duct cysts. (Reproduced with permission from Greenfield LJ, Mulholland MW, Oldham KT, et al., eds: *Surgery: Scientific Principles and Practice 3rd edition*. Lippincott Williams & Wilkins, Philadelphia: 2001.)

Summary of Procedures

Position	Supine, with neck in hyperextended position
Incision	Transverse skin incision





Unique considerations

Antibiotics

Surgical time

Closing considerations

EBL

Mortality

Morbidity

Pain score

Surgeon may request assistance, by placing finger at base of tongue to identify the cephalad extent of the needed dissection.

Cefazolin 1 g iv

1–1.5 h

Careful hemostasis. Coughing may be associated with venous congestion and hematoma formation.

5–10 mL

< 0.1%

Bleeding: < 5%

Infection: < 5%

3–4

(Print pagebreak 657)

Patient Population Characteristics

Age range

6 mo–30 yr (40% present at < 10 yr)

Male:Female

1:1

Incidence

1:3000

Etiology

Persistence of undifferentiated epithelial cells in area of hyoid bone that later become squamous-cell epithelium or glandular tissue

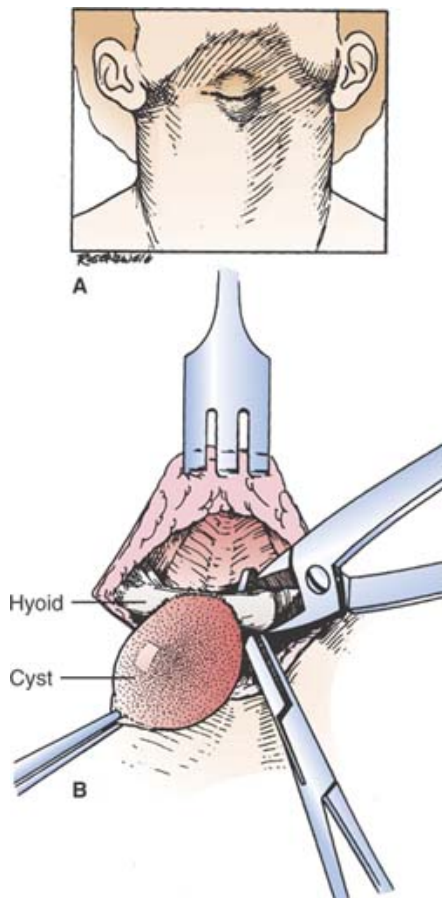


Figure 7.11-2. 2. Thyroglossal duct cyst excision. **(A)** Incision placed over presenting cyst; no skin excised. **(B)** Cyst has been dissected from surrounding tissues, and hyoid is exposed after division of sternohyoid and thyrohyoid muscles at insertion. The bone is encircled with a short right-angle clamp 1 cm from its midpoint, where it is divided with a bone cutter or cautery. (Reproduced with permission from Baker RJ, Fischer JE: *Mastery of Surgery*, 4th edition. Lippincott



Williams & Wilkins, Philadelphia: 2001.)

(Print pagebreak 658)

Anesthetic Considerations

See [Anesthetic Considerations following Thyroidectomy, p. 660.](#)

Suggested Readings

1. Gauger PG: Thyroid gland. In *Greenfield's Surgery, Scientific Principles and Practice*, 4th edition. Mulholland MW, Lillemore KD, Doherty GM et al., eds. Lippincott Williams & Wilkins, Philadelphia: 2006, 1289–309.
2. Organ GM, Organ CH: Thyroid gland and surgery of the thyroglossal duct: exercise in applied embryology. *World J Surg* 2000; 24(8):886–90.
3. Tracy TF Jr, Muratore CS. Management of common head and neck masses. *Semin Pediatr Surg* 2007; 16(1):3–13.

Thyroidectomy

Surgical Considerations

Description: Thyroidectomy is performed through a transverse neck incision ([Fig. 7.11-3](#)) (**Kocher**), usually 6–8 cm long. **Minimally invasive approaches**, including a totally endoscopic approach, have been described, but they remain controversial, as the gland must be removed intact for adequate histological analysis. In the traditional approach, the platysma muscle is divided sharply and subplatysmal flaps are developed superiorly and inferiorly. The two large anterior jugular veins must be avoided and are occasionally a source of blood loss, although rarely of any hemodynamic significance. When the flaps are adequately developed, a large thyroid retractor may be placed to expose the midline prethyroid fascia (median raphe). This is sharply divided in the midline, exposing the strap muscles, which can then be mobilized off the thyroid gland.

After the thyroid gland is exposed, resection can proceed. Resection may be total, subtotal (lobe + isthmus ± partial remaining lobe), or lobar. Degree of resection depends on diagnosis and may be modified based on operative findings. During this portion of the operation, hemostasis is critical to maintain adequate visualization. Resection of a lobe usually begins with ligation of the middle thyroid veins along the midlateral aspects of the thyroid ([Fig. 7.11-4](#)). The superior and inferior poles are then mobilized and ligated with care being taken to identify the superior and ([Print pagebreak 659](#)) inferior parathyroid glands. When the lateral aspects of the thyroid are mobilized, the medial portions may be dissected. Special care must be taken medially along the tracheal-esophageal groove to prevent damage to the recurrent laryngeal nerve, especially in reoperation. As soon as the gland is mobilized to the degree that it is only adherent to the trachea, it may be fully resected using a knife to prevent cautery injury to the trachea. Any enlarged or suspicious lymph nodes should be excised and sent for pathological examination. After excision, hemostasis is secured. Closure involves the use of absorbable sutures to repair the midline fascia and the platysma. The skin may be closed with running monofilament suture or staples. The use of drains remains controversial and has not been shown to decrease the rate of hematoma formation.

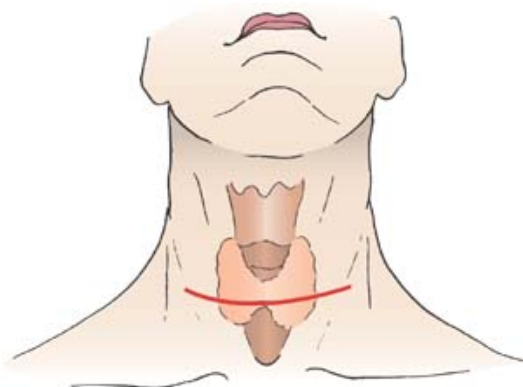




Figure 7.11-3. 3. Transverse incision at base of neck for thyroidectomy. (Reproduced with permission from Baker RJ, Fischer JE: *Mastery of Surgery*, 4th edition. Lippincott Williams & Wilkins, Philadelphia: 2001.)

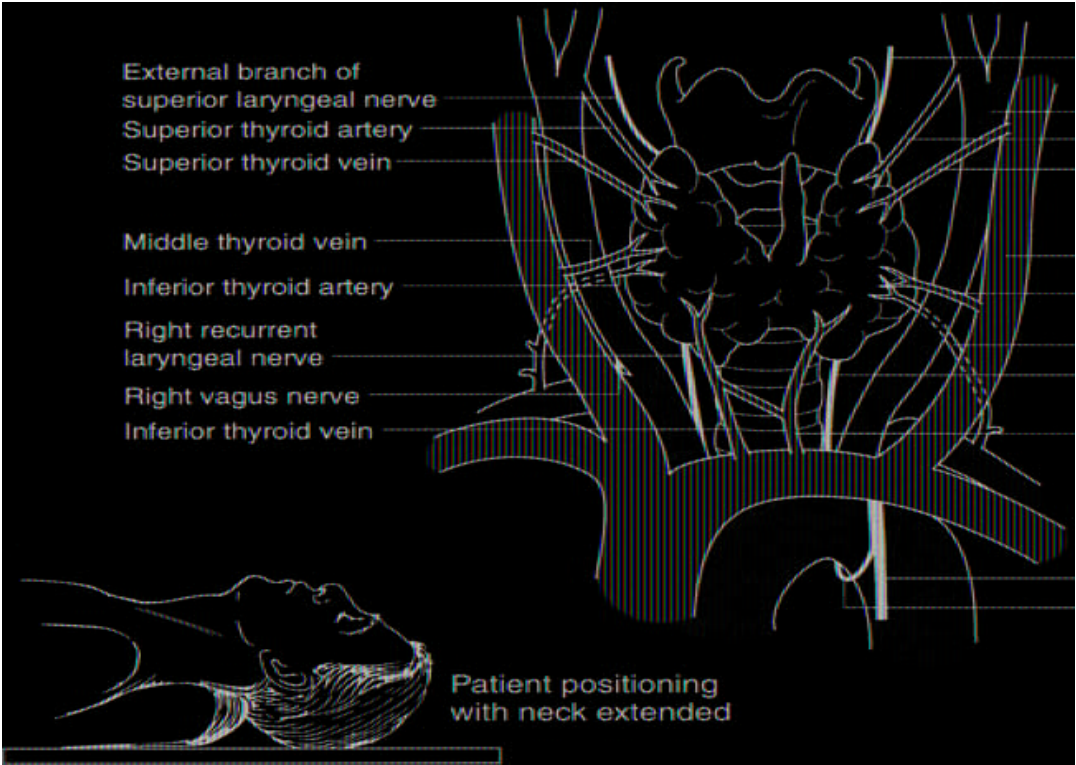


Figure 7.11-4. 4. Vascular relationships to the thyroid gland. (Reproduced with permission from Greenfield LJ, Mulholland MW, Oldham KT, et al., eds: *Surgery: Scientific Principles and Practice*. Lippincott Williams & Wilkins, Philadelphia: 2001.) Inset shows patient positioning with neck extended.

Usual preop diagnosis: FNA findings of definite/suspicious/inconclusive for malignancy; goiter; thyroid cancer (papillary, follicular, medullary, anaplastic); thyroid nodule; hyperthyroidism; Grave's disease

Summary of Procedures

Position	Supine with head elevated to 30° and neck extended (see Fig. 7.11-4 , inset)
Incision	Transverse cervical
Unique considerations	Patients with uncontrolled hyperthyroidism are at ↑ risk for developing thyroid storm during surgery. Hyperthyroidism may be controlled preop with either β-adrenergic blockade or propylthiouracil. Preop iatrogenic hypothyroidism may be associated with ↓ BP and circulatory collapse during induction of anesthesia.
Antibiotics	None routinely used
Surgical time	1–2 h
Closing considerations	Adequate hemostasis; minimize coughing
EBL	50–75 mL
Postop care	Observe for postop respiratory problems 2° recurrent laryngeal nerve injury (bilateral), hematoma, or ↓ Ca ²⁺ Admit × 24 h. 3 Ca ²⁺ the night of surgery and the following a.m. Same-day surgery remains controversial 2° delayed discovery of hematoma (16 h) and hypocalcemia.
Mortality	< 0.5% Hypoparathyroidism (↓ Ca ²⁺): 3–5%





Morbidity

Hematoma: 1–2%
Thyroid storm (usually in association with Graves' disease)
Wound infection: 0.2–0.5%
Recurrent laryngeal nerve damage
 Unilateral (hoarseness): 0.77%
 Bilateral (aphonia, respiratory obstruction): 0.39%
Pain score
3–4

Pain score

(Print pagebreak 660)

Patient Population Characteristics

Age range

15–80 yr

Male:Female

Overall: 1:8

Cancer: papillary (1:2); follicular (1:3); medullary; anaplastic; lymphoma (1:1)

Incidence

Cancer (Uncommon)

Benign (Relatively common)

Etiology

Cancer (50%): papillary (80%); follicular (10%); medullary (5%); anaplastic (1%); lymphoma (1%) Benign lesions (50%): nontoxic goiter (20%); thyrotoxicosis (10%); thyroiditis (5%); benign nodules (5%); other (10%)

Associated conditions

Other endocrine disorders (e.g., pheochromocytoma in association with medullary thyroid carcinoma in patients with MEN 2A and 2B)



Anesthetic Considerations

(Procedures covered: excision of thyroglossal duct cyst; thyroidectomy)



Preoperative

Hyperthyroidism may be 2° Graves' disease (common), toxic multinodular goiter, thyroid adenomas, TSH-secreting tumor (rare), or overdosage of thyroid hormone. Common Sx are fatigue, sweating, intolerance to heat, ↑ appetite, ↑ HR, ↑ BP, ↑ pulse pressure, ↑ T, weight loss or gain, thyroid goiter, and exophthalmos. Some older patients exhibit apathetic thyrotoxicosis, which is often mistaken for hypothyroidism. CHF and AF are common with these patients. Hypothyroidism may be iatrogenic or 2° autoimmune thyroiditis. Common Sx are intolerance to cold, anorexia, fatigue, weight gain or loss, constipation, ↓ HR, ↓ pulse pressure, ↓ DTR, ↓ T, ↓ mentation. Patients presenting for thyroidectomy usually are made euthyroid before surgery and may be taking one or more of the following medications: propylthiouracil, methimazole, potassium iodide, glucocorticoids, or β-blockers. An important aspect of the preop visit is to ensure that the patient is in a physiologically euthyroid state (T, HR, pulse pressure, reflexes).

General:

Beware of tracheal compression with large goiters, → tracheal deviation, stridor.

Respiratory

Tests: CXR; consider preop CT scan of neck to evaluate possible tracheal involvement, especially in patients with large goiters.





	T4	T3ru	T3	TSH
Hyperthyroid				
1°	↑	↑		Normal
hypothyroid	↓	↓	↓ or normal	↓
2°	↓	↓	↓	↓
hypothyroid				

Tests: Thyroid function;
Ca²⁺, Mg²⁺, phosphate;
alkaline phosphatase;
glucose

Endocrine

	T4	T3ru	T3	TSH
Hyperthyroid	↑		↑	Normal or ↓
1° hypothyroid	↓		↓ or normal	↑
2° hypothyroid	↓		↓	↓

(Print pagebreak 661)

Hyperthyroidism:

Respiratory

↑ BMR → ↑ VO₂ → rapid desaturation on induction.
↑ HR, AF (10–40% incidence), palpitations, CHF. A normal resting HR is helpful in determining whether the patient is ready for surgery. If the situation calls for it (e.g., emergency surgery), the patient can be treated with β-blockers to blunt the sympathomimetic effects of the hyperthyroid state. β-blocker therapy can be problematic in patients with CHF (titrate while monitoring filling pressures and CO).

Cardiovascular

Tests: ECG; consider preoperative ECHO for evaluation of LV function.

Neurological

Warm, moist skin, nervousness, anxiety (may require generous sedation), tremor, ↑DTRs.

Musculoskeletal

Higher incidence of myasthenia gravis and skeletal muscle weakness (↑ sensitivity to muscle relaxants), clubbing of the fingers, weight loss, myopathy

Hematologic

Tests: CK, urine myoglobin

Gastrointestinal

Mild anemia, thrombocytopenia

Laboratory

Tests: CBC

Premedication

Weight loss and diarrhea

Tests: As indicated from H&P.

Other tests as indicated from H&P.

Midazolam 1–2 mg iv. Continue antithyroid medications preop.

Hyperthyroid patients must be made euthyroid before **elective** surgery and may be on the following drugs: propylthiouracil, methimazole, potassium iodide, β-blockers, and glucocorticoids.

A life-threatening exacerbation of hyperthyroidism occurring during periods of stress, which is manifested by hyperthermia (> 40°C), tachycardia, widened pulse pressure, anxiety, altered mental state → psychosis → coma, and myopathy (rhabdomyolysis in 50%; severe in 4%). (Thyroid storm has been mistaken intraop for malignant hyperthermia, sepsis, anaphylaxis, and other hypermetabolic reactions, e.g., neuroleptic malignant syndrome.) Thyroid storm is most often associated with Graves' disease that has been incompletely treated prior to surgery.

Thyroid storm

General Rx: ↑ FiO₂ fluid resuscitation; electrolyte replacement/correction (↑Ca²⁺); cooling blankets; acetaminophen; maintain diuresis (**maintain euvoolemia**) if rhabdomyolysis; treat





precipitating event (infection, CHF, DKA, pregnancy). **Specific Rx:** propylthiouracil (block synthesis) (200–250 mg po q 4 h); sodium iodide (block release) (1–2.5 g iv); steroids (mechanism unclear)—hydrocortisone (100 mg iv q 8 h), or dexamethasone (4 mg iv q 24 h); β -blockers (use with caution in patients with reactive airway disease, AV block, or CHF)—propranolol (20–120 mg po q h or 0.25–1.0 mg iv q 5 min), and/or esmolol (50–300 mcg/kg/min). Note: Block synthesis **BEFORE** (1 h is adequate) giving iodides to block release, otherwise “iodine escape” will occur later.

Hypothyroidism:

Respiratory

Beware of tracheal compression with large goiters → tracheal deviation, stridor. ↓ ventilatory response to ↑ CO_2 and ↓ O_2 (caution with opioids and sedatives).

Tests: CXR; consider preop CT scan of neck to evaluate possible tracheal involvement, especially in patients with large goiters.

Bradydysrhythmias, diastolic HTN, pericardial effusions, ECG → ↓ voltage, ST-T wave D's, ↑ QT, occasional VT (torsades de pointe—pause-dependent). This type of VT is treated with MgSO_4 , cardioversion; then isoproterenol or pacing to shorten QT.

Cardiovascular

Thyroid replacement must be weighed against the risk of precipitating myocardial ischemia in patients with known CAD.

Diastolic dysfunction, ↓ LV compliance → **cautious** volume expansion.

Tests: ECG; consider preoperative ECHO for evaluation of LV systolic/diastolic function, pericardial effusion/tamponade.

Addison's disease occurs in 5–10% of patients with severe hypothyroidism; some patients may receive a ‘stress dose’ of steroids (hydrocortisone 100 mg iv q 8 h \times 3) in the periop period.

Tests: Cortisol stimulation test

Endocrine

↓ BMR → slow mentation and movement, cold intolerance, ↓ reflexes with “hangup” (delayed relaxation phase)

Neurological

Arthralgias and myalgias

Musculoskeletal

Impaired renal function 2° amyloidosis, urinary retention, oliguria. (50% incidence of ↓ Na^+)

Renal

Tests: Consider BUN; Cr; Na^+

Coagulation abnormalities, anemia

Hematologic

Tests: CBC

GI bleeding, constipation, ileus

Gastrointestinal

Tests: As indicated from H&P.

Laboratory

Other tests as indicated from H&P.

Midazolam 1–2 mg iv. (None in the patient who is clinically hypothyroid and requires emergent surgery.) Hypothyroid patients can safely undergo anesthesia/surgery if they have mild-to-moderate disease. Clinically hypothyroid patients (↓ HR, ↓ T, ↓ pulse pressure, ↓ DTRs) should be given thyroid replacement before elective surgery.

Premedication

Severe hypothyroidism constituting a medical emergency, with mortality of > 50%. Manifestations include stupor or coma, hypothermia (which correlates inversely with mortality), hypoventilation with hypoxemia, bradycardia (HR 50–60), hypotension, apathy, hoarseness, and hyponatremia. **Supportive measures:** early intubation and mechanical ventilation; treat ↓ BP with **cautious** volume expansion (risk of pulmonary edema), inotropes (risk of arrhythmias), pacing (carefully, 60–70 b/min) and r/o pericardial effusion, passive rewarming only, especially if





Myxedema comma

↓ BP (active warming for $T < 30^{\circ}\text{C}$); correct ↓ Na^{+} carefully (risk of Central Pontine Myelinolysis); correct ↓ glucose. **Specific Rx:** L-thyroxine (T_4) (200–500 mcg iv loading dose, 100–300 mcg iv the next day); or tri-iodothyronine (T_3) (20–50 mcg iv q 6–12 h); hydrocortisone (100–300 mg iv q d). T_4 onset is slow (6 h after iv administration) and has to be converted peripherally (slowed in hypothyroid state) to biologically active T_3 . Also, T_4 may be converted in some critically ill patients to biologically inactive rT_3 (“reverse T_3 ”). ↓ TSH level is the earliest sign of response.

(Print pagebreak 662)

Intraoperative

Anesthetic technique: Normally, GETA; infrequently, under local anesthesia. For inadequately treated hyperthyroid patients, it is important to establish an adequate depth of anesthesia to prevent an exaggerated sympathetic response to surgical stimulation. Avoid agents that stimulate the sympathetic nervous system (e.g., ketamine, pancuronium, meperidine). Hypothyroidism may be associated with ↑ sensitivity to anesthetic agents and muscle relaxants.

Induction

Standard induction for euthyroid patients ([p. B-2](#)). If the patient has airway compromise 2° a large thyroid goiter, consider an awake fiber optic intubation ([p. B-5](#)).

Maintenance

Standard maintenance ([p. B-2](#)). Maintain muscle relaxation. Use nerve stimulation to guide relaxant dosing.

Emergence

Airway obstruction 2° recurrent laryngeal nerve damage, tracheomalacia (especially in patients with large destructive goiters), or hematoma can occur. Consider visualizing vocal cord function before extubation.

Blood and fluid

Minimal blood loss
IV: 18 ga × 1
NS/LR @ 5–8 mL/kg/h
Head-up position

Slight head-up position can help make for a bloodless surgical field without substantially increasing the risk of VAE.

Monitoring

Standard monitors ([p. B-1](#)).

+ others as indicated by patient's status.
Maintain normothermia, especially in hypothyroid patients.

Positioning

pad pressure points
eyes

Supine, with head slightly hyperextended, allows for surgical exploration of the neck. In hypothyroid patients, marked ↓ BP and ↓ RR may occur with minimal anesthetic doses.

Complications

Cardiorespiratory depression

(Print pagebreak 663)

Postoperative

Complications

Recurrent laryngeal nerve damage

Bilateral: patient will be unable to speak and will require reintubation. CPAP may temporize situation and make re-intubation less emergent. Unilateral: characterized by hoarseness.

Tracheomalacia or hematoma with airway compromise

Acute airway obstruction may occur immediately postop, and rapid reintubation may be lifesaving. If airway compromise is 2° hematoma, reopen incision and drain remaining blood; if patient still requires artificial airway, consider CPAP or awake re-intubation.

Acute hypocalcemia can present as laryngeal stridor (24–48 h postop),





Acute hypoparathyroid state
(hypocalcemia)

although it most often presents with tingling in the fingertips and in the lips. If untreated and severe, this can progress to tetany and seizures. Administering 1 amp Ca+gluconate given iv over 20 min usually alleviates symptoms. Rx: measure Ca⁺⁺ replace if necessary. CPAP is effective for airway compromise.
Can mimic MH (see Rx above).

Thyroid storm
PCA morphine (see [p. C-3](#))

Vocal cord function

Ability to phonate “e” implies continued vocal cord function.

Pain management

Tests

Suggested Readings

1. Abbas G, Dubner S, Heller KS: Re-operation for bleeding after thyroidectomy and parathyroidectomy. *Head Neck* 2001; 23(7):544–6.
2. Arora N, Dhar P, Fahey TJ 3rd: Seminars: local and regional anesthesia for thyroid surgery. *J Surg Oncol* 2006; 94(8):708–13.
3. Bhattacharyya N, Fried MP: Assessment of the morbidity and complications of total thyroidectomy. *Arch Otolaryngol Head Neck Surg* 2002; 128(4):389–92.
4. Farling PA: Thyroid disease. *Br J Anaesth* 2000; 85:15–28.
5. Fitzgibbons SC, Brams DM, Wei JP: The treatment of thyroid cancer. *Am Surg* 2008; 74(5):389–99.
6. Gauger PG: Thyroid gland. In: *Greenfield's Surgery: Scientific Principles and Practice*, 4th edition. Mulholland MW, Lillemore KD, Doherty GM et al., eds. Lippincott Williams & Wilkins, Philadelphia: 2006, 1289–311.
(Print pagebreak 664)
7. Gaz, RD: Hyperthyroidism. In: *Current Surgical Therapy*, 7th edition. Cameron JL, ed. Mosby Inc, St. Louis: 2001, 653–7.
8. Kearney T, Dang C: Diabetic and endocrine emergencies. *Postgrad Med J* 2007; 83(976):79–86.
9. Miccoli P: Minimally invasive surgery for thyroid and parathyroid diseases. *Surg Endosc* 2002; 16(1):3–6.
10. Schwartz JJ, Rosenbaum SH: Anesthesia and the endocrine system. In: *Clinical Anesthesia*, 5th edition. Barash PG, Cullen BF, Stoelting RK, eds. Lippincott Williams & Wilkins, Philadelphia: 2006, 1129–51.
11. Zeiger, MA: Nontoxic goiter. In: *Current Surgical Therapy*, 7th edition. Cameron JL, eds. Mosby Inc, St. Louis: 2001, 642–4.

Parathyroidectomy

Surgical Considerations

Description: The traditional approach for primary hyperparathyroidism requires **four-gland visualization** with removal of any abnormally large glands. Newer methods of localization and confirmation of adenomas have ushered in an era of minimally invasive parathyroid surgery, using either a **minimally invasive approach**, with a small skin incision and focused operative dissection, or an **endoscopic technique**, with trocar ports and CO₂ insufflation. The minimally invasive approach is aided by the preop localization studies described below. If localization is unsuccessful at identifying the correct gland than the minimally invasive approach is





converted to the standard incision and all four glands are explored until the diseased gland is identified.

For the standard open operation, an incision in the lower neck ([Fig. 7.11-5](#)) is made and the platysma is divided. Flaps are created below the platysma superiorly and inferiorly to allow increased working space and expose the prethyroid fascia. The midline between the strap muscles is identified and divided, exposing the thyroid gland. The superior parathyroids are located behind the upper pole, in association with the superior thyroid artery, which often must be taken to locate the parathyroid. The inferior glands are located near the junction of the inferior thyroid artery and the recurrent laryngeal nerve ([Fig. 7.11-6](#)). Hemostasis is crucial to maintain adequate visualization in the surgical field. For the traditional approach, all four glands are located, and biopsies are sent for confirmation of parathyroid tissue. Abnormal glands are removed. If four-gland hyperplasia is found, which may occur with multiple endocrine neoplasia (MEN) 1 or 2A (secondary or tertiary hyperparathyroidism), then all four glands are removed and some tissue is saved for reimplantation in the forearm.

With preop localization studies, dissection may be limited to the area of suspicion, and the abnormal gland or glands removed. Confirmation of successful resection may be made by observing a 50% decrease in the parathormone level, 5 min after removal, as compared with the preop level. This very sensitive assay is rapidly becoming the standard of care in the treatment of primary hyperparathyroidism. The addition of preop methylene blue or radioactive tracers may lend additional assurances that the parathyroid tissue has been identified. If the parathyroid adenoma cannot be found, the surgeon should investigate other areas of the neck, including the retroesophageal space, carotid sheath, posterior triangle, and below the thyroid. Unilateral thyroid lobectomy may be performed when three normal glands have been found and the fourth gland is missing, as the adenoma may be in the thyroid substance. Mediastinal exploration should not be done on primary exploration.

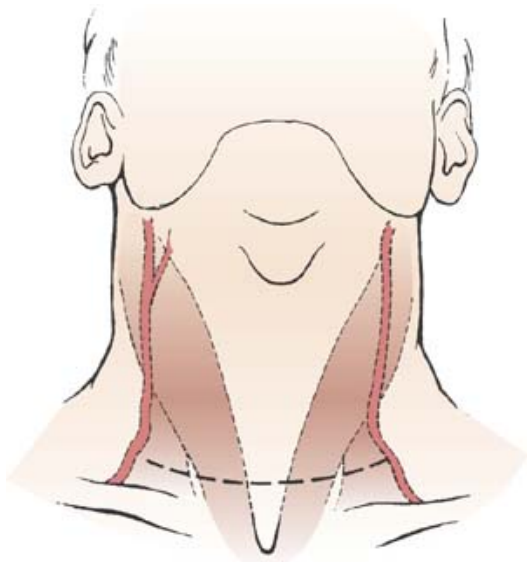


Figure 7.11-5. 5. Skin incision for parathyroid exploration, made 1–2 finger-breadths superior to sternal notch, as far lateral as the external jugular veins on both sides. (Reproduced with permission from Baker RJ, Fischer JE: *Mastery of Surgery*, 4th edition. Lippincott Williams & Wilkins, Philadelphia: 2001.)

(Print pagebreak 665)



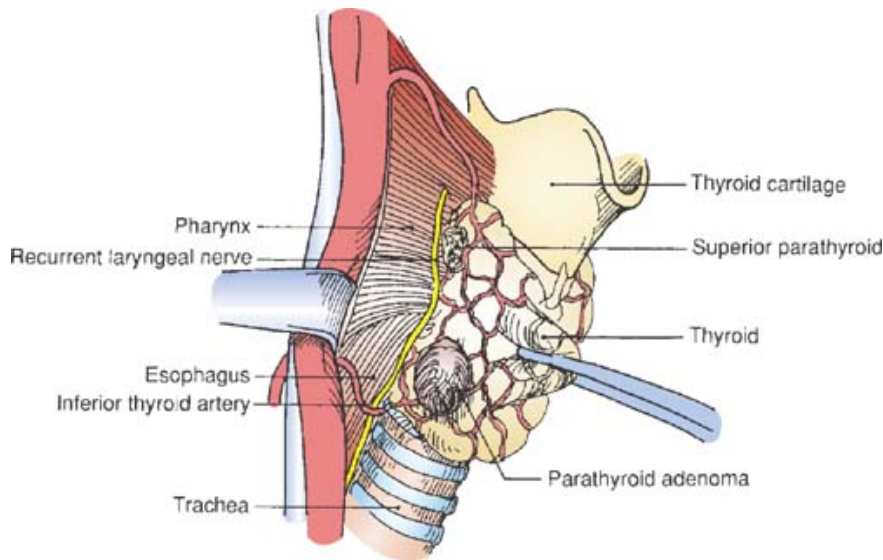


Figure 7.11-6. 6. Identification of upper parathyroid gland on right side. (Reproduced with permission from Scott-Conner CEH, Dawson DL: *Operative Anatomy*, 2nd edition. Lippincott Williams & Wilkins, Philadelphia: 2003).

Variant procedure or approaches: Initial studies of the endoscopic approach have suggested morbidity and rates of cure similar to those of standard procedures, with better cosmetic results. Larger series of randomized trials need to be completed before this approach can be applied broadly.

Usual preop diagnosis: Parathyroid adenoma (multiple in 2–3% of cases); hyperparathyroidism (secondary or tertiary); parathyroid carcinoma

Summary of Procedures

Position	Supine; shoulder roll; reverse Trendelenburg or 30° tilt; head rest (gel donut)
Incision	Transverse cervical (4–8 cm) (see Fig. 7.11-5) Methylene blue (7.5 mg/kg in 500 mL of NS) may be administered in the preop holding area 30 min before surgery → spurious ↓ SpO ₂ Radioactive technetium sestamibi (20 mCi) may be administered 60–90 min before operation. Methylene blue and radioactive tracers may be used independently or simultaneously to aid in identification of parathyroid tissue.
Unique considerations	
Antibiotics	Cefotetan 1 g iv preop
Surgical time	1–2 h
Closing considerations	Coughing may be associated with venous congestion and hematoma formation.
EBL	25–50 mL
Postop care	Monitor serum Ca ²⁺ (nl = 8.5–10.5 mg% total Ca; 1–1.3 mM ionized Ca) ²⁺ Patients typically admitted for 24 h observation to serum Ca ²⁺ levels and monitor for hematoma/airway compromise.
Mortality	< 0.5%
Morbidity	Hypocalcemia: < 15% Hypoparathyroidism: < 5% Hematoma: 1% Infection: 1% Recurrent laryngeal paralysis: < 1%
Pain score	3–4





(Print pagebreak 666)

Patient Population Characteristics

Age range	Increases with age
Male:Female	4:1
Incidence	50–100/100,000 (1.5/100 in elderly)
Etiology	Single adenoma (85%); double adenoma (2–3%); hyperplasia (10–15%); cancer (< 1%) Bone disease (5–15%); duodenal ulcer (5–10%); renal calculi (60–70%); MEN-1 (parathyroid + hyperplasia + pituitary adenoma + pancreatic neuroendocrine tumor); MEN-2A (parathyroid hyperplasia + medullary thyroid cancer + pheochromocytoma); HTN (20–50%)
Associated conditions	

Anesthetic Considerations

Preoperative

These patients typically present with hypercalcemia (hyperparathyroidism), which must be controlled before surgery. Although 25–50% of cases are asymptomatic, many will present with a variety of Sx, including fatigue, muscle weakness, depression, anorexia, nausea, constipation, abdominal and bone pain, HTN, renal stones, and polydipsia. Differential diagnosis for the hypercalcemic patient includes: metastatic disease, multiple myeloma, milk-alkali syndrome, vitamin D intoxication, sarcoidosis, hyperthyroidism, thiazide diuretics, adrenal insufficiency, Paget's disease, immobilization, or an exogenous parathyroid hormone-producing tumor.

Respiratory

Hyperparathyroidism is associated with ↓ clearance of secretions from the tracheobronchial tree → postop atelectasis. Avoid respiratory or metabolic acidosis, which will ↑ the free fraction of Ca → hypercalcemia (↑ BP, ↑ muscle weakness, ↑ HR).

Tests: As indicated from H&P.

HTN (usually resolves with treatment; if severe or episodic, r/o pheochromocytoma); ECG may show tachycardia with ↓ PR and ↓ QT intervals. Patients may be hypovolemic (2° anorexia, N/V, and polyuria) and have ↑ sensitivity to digitalis + resistance to catecholamines.

↓ BP may result from polyuria → hypovolemia. **Preop management** includes correction of intravascular volume and electrolyte abnormalities. Preop treatment of hypercalcemia includes aggressive expansion of intravascular volume, followed by diuresis (**maintain** euvoolemia), to ↑ renal Ca⁺ excretion, usually accomplished with iv NS, and then furosemide. Calcium channel blockers (verapamil, nifedipine, diltiazem) can be used. Hypophosphatemia can impair myocardial contractility and should be corrected; hemodialysis or peritoneal dialysis can be used to lower dangerously elevated serum Ca⁺ levels.

Mithramycin, plicamycin, calcitonin, bisphosphonates, cisplatin, or steroids are not useful for acute Rx of ↑ Ca⁺ (too slow).

Tests: ECG; electrolytes; others as indicated from H&P.

Patient may present with seizures, hyporeflexia, mental status changes (somnolence, depression, memory loss, psychosis, coma), or peripheral neuropathy. Significant improvement often follows correction of hypercalcemia.

Cardiovascular

Neurological





Musculoskeletal

Hematologic

Endocrine

Renal

Gastrointestinal

Laboratory

Premedication

These patients may have muscle atrophy and weakness, osteopenia, arthralgia, pathologic fractures (careful laryngoscopy and positioning), osteitis fibrosa cystica. Response to NMBs may be enhanced 2° \uparrow Ca^{++} muscle weakness.

Patients also tend to be hypophosphatemic and may show signs of hemolysis, Plt dysfunction, impaired ventricular contractility, and leukocyte dysfunction.

Tests: CBC with Plt count, PO_4^{2-}

Primary hyperparathyroidism is most commonly due to benign parathyroid adenoma (90%), or hyperplasia (9%) and rarely carcinoma. It may be associated with MEN syndrome. MEN-1 consists of tumors of the parathyroid, pancreatic islets, and pituitary. MEN-2 includes pheochromocytoma, mucosal neuromas, parathyroid tumors, and medullary thyroid carcinoma.

***NB: Anesthetizing a patient with an unrecognized pheochromocytoma could result in a fatality.**

Tests: As indicated from H&P.

Patients may have renal dysfunction 2° nephrolithiasis, nephrocalcinosis, renal tubular disorders, and glomerular disorders. Polyuria 2° \uparrow Ca^{++} electrolyte disturbances.

Tests: Cr; BUN; electrolytes

These patients may have constipation, anorexia, N/V, epigastric pain.

Symptoms not related as well to absolute Ca^{++} level but to rate of change. Serum Ca^{++} 12 mg/dL: likely asymptomatic; 12–14 mg/dL: mild symptoms; >16 mg/dL: life-threatening. Albumin (\uparrow albumin by 1 g/dL will \uparrow total serum Ca^{++} by 0.8 mg/dL); electrolytes; Mg^{++} phosphate (usually low).

All medications to lower hypercalcemia should be continued unless Ca^{++} levels have normalized. If patient has been treated with steroids in the preop period, administer a stress dose (hydrocortisone 100 mg iv q 8 h \times 24 h) before induction of anesthesia and continue into the early postop period. Standard premedications ([p. B-1](#)) are usually appropriate in this patient group, unless patient has mental status changes.

(Print pagebreak 667)

Intraoperative

Anesthetic technique: GETA, with head slightly hyperextended, allows for surgical exploration of the neck. Cervical plexus blocks may be appropriate in selected patients; however, phrenic nerve block \rightarrow respiratory compromise.

Induction

Maintenance

Emergence

Blood and fluid requirements

Monitoring

Standard induction ([p. B-2](#)). If patient is clinically hypovolemic, restore intravascular volume before induction and titrate induction dose of sedative/hypnotic agents.


Standard maintenance ([p. B-2](#)), with muscle relaxant titrated to effect, using peripheral nerve stimulator. Avoid hyperventilation or hypoventilation (acidosis will \uparrow Ca^{++} levels, while alkalosis will \downarrow Ca^{++} levels). Maintain adequate hydration and UO throughout the procedure.

No special considerations (see postop complications, below).

Minimal blood loss	Avoid Ca^{++} -containing iv solution (e.g., LR), use
IV: 18 ga \times 1	Normosol, NS.
NS @ 5–8 mL/kg/h	Others as indicated by patient status.
Standard monitors (p. B-1)	Patients should be positioned carefully as they tend to be osteopenic and are prone to pathologic bone fractures. Slight head-up

and pad all pressure points



Positioning	eyes	position may ↓ blood loss and improve surgical visibility without significantly ↑ risk of VAE.
Complications	Hypocalcemia	See postop complications (below).
(Print pagebreak 668)		
 Postoperative		
Complications	Hypocalcemia Hypocalcemic tetany Seizure Laryngospasm Recurrent laryngeal nerve injury	Hypocalcemia may occur in the immediate postop period. Sx include paresthesias, muscle spasm, tetany, laryngospasm, bronchospasm, and apnea. Rx: includes 10–20 mL Ca ⁺⁺ gluconate 10% over 10 min. Follow levels and repeat therapy until the clinical signs of hypocalcemia are controlled. CPAP is effective for airway obstruction.
	Hypophosphatemia Laryngeal edema 2° surgical trauma Stridor Hematoma with airway compromise	Recurrent laryngeal nerve dysfunction can be monitored by having the patient vocalize the letter “e.” Unilateral vocal cord dysfunction results in hoarseness, while bilateral vocal cord dysfunction results in aphonia. CPAP is effective for airway obstruction.
	Pneumothorax	Dx: pleuritic chest pain, dyspnea, ↑ RR, ↓ breath sounds, ↑ resonance, hypoxemia; CXR. Rx: O ₂ chest tube and reintubation as necessary.
Pain management	PCA (p. C-3)	
Tests	Serial measurements of: Ca ⁺⁺ Phosphate Mg ⁺⁺ Others as clinically indicated CXR to rule out pneumothorax	The lowest Ca ⁺⁺ level usually is seen after 4–5 d postop. Follow clinical Sx of hypocalcemia: Trousseau's sign (carpopedal spasm in response to application of a BP cuff at a pressure > SBP for 3 min); Chvostek's sign (contracture of the facial muscles produced by tapping on the facial nerve).

Suggested Readings

1. Abbas G, Dubner S, Heller KS: Re-operation for bleeding after thyroidectomy and parathyroidectomy. *Head Neck* 2001; 23 (7):544–6.
2. Carling T, Udelsman R. Focused Approach to Parathyroidectomy. *World J Surg* 2008; Apr 5:[Epub ahead of print]
3. Doherty GM: Parathyroid gland. In *Greenfield's Surgery: Scientific Principles and Practice*, 4th edition. Mulholland MW, Lillemore KD, Doherty GM, eds. Lippincott Williams & Wilkins, 2006: 1310–33.
4. Gauger PG, Thompson NW: Persistent or recurrent hyperparathyroidism. In *Current Surgical Therapy*, 7th edition. Cameron JL, eds. Mosby Inc, St. Louis: 2001, 668–71.
5. Miccoli P: Minimally invasive surgery for thyroid and parathyroid diseases. *Surg Endosc* 2002; 16(1):3–6.
6. Mihai R, Farndon JR: Parathyroid disease and calcium metabolism. *Br J Anaesth* 2000; 85:29–43.





7. Schwartz JJ, Rosenbaum SH: Anesthesia and the endocrine system. In *Clinical Anesthesia*, 5th edition. Barash PG, Cullen BF, Stoelting RK, eds. Lippincott Williams & Wilkins, Philadelphia: 2006, 1129–51.

8. Stalberg P, Delbridge L, van Heerden J et al: Minimally invasive parathyroidectomy and thyroidectomy—current concepts. *Surgeon* 2007; 5(5):301–8.

Adrenalectomy

Surgical Considerations

Description: Adrenalectomy can be performed via a number of different approaches, each with its own merits. Traditionally, the adrenal glands have been removed with an open incision through either the transperitoneal or extraperitoneal (flank) approach ([Fig. 7.11-8](#)). With some exceptions (see below), laparoscopic approaches are becoming the favored methods. Indication in malignant disease or metastasis remains controversial and is currently being evaluated. Relative contraindications include large adrenal adenocarcinomas (> 5 cm), (*Print pagebreak 669*) malignant pheochromocytoma, invasive adrenal mass, large adrenal mass (> 8–10 cm), or other contraindications to abdominal laparoscopic surgery (e.g., multiple previous abdominal surgeries). Each approach is discussed individually, but their basic principles remain the same. The anatomy of the retroperitoneum is shown in [Fig. 7.11-7](#).

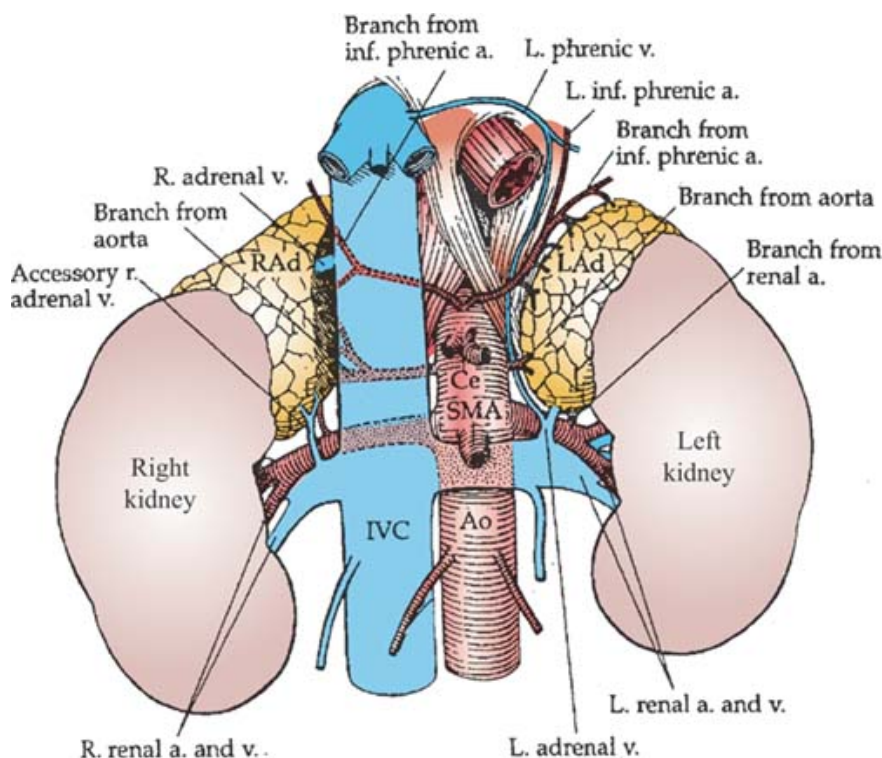


Figure 7.11-7. 7. Anatomic relationships of the adrenal glands. Note the origins of the three main arteries: inferior phrenic, aortic, and renal branches. Note also the single draining veins (except for a small accessory right adrenal vein): the right-located superior and medial, and the left-found inferior and medial. (Ao = aorta; Ce = celiac; LAd = left adrenal gland; RAAd = right adrenal gland.) (Reproduced with permission from Baker RJ, Fischer JE: *Mastery of Surgery*, 4th edition. Lippincott Williams & Wilkins, Philadelphia: 2001.)

Open transperitoneal approach: This approach allows for easy access to both adrenals and is preferred for large tumors. A midline or bilateral subcostal incision ([Fig. 7.11-8](#)) is used, with the patient in the supine position. The left adrenal is accessed by incising the lateral peritoneal attachments of the spleen. The spleen and tail of the pancreas are rotated medially, exposing Gerota's fascia, which is then incised at the upper pole of the kidney, exposing the adrenal gland. A combination of blunt and sharp dissection is used to mobilize the gland and expose the adrenal vein, which is ligated between ties, and the gland is removed. The right gland is exposed by retracting the liver cephalad and depressing the hepatic flexure inferiorly. The peritoneum is then incised lateral to the duodenum and the inferior vena cava (IVC) is exposed. The right kidney is pulled down and the right adrenal vein





entering the IVC is identified. The adrenal vein is ligated and the gland is removed.

Open extraperitoneal approach (flank): Before the advent of laparoscopic surgery, this approach was favored to minimize pain and improve postop recovery of adrenalectomy through use of smaller incision size and by remaining extraperitoneal. In general, this approach is best used for unilateral, smaller tumors. The patient is placed in the prone jackknife position and a dorsal curved flank incision is made, exposing the 12th rib. The rib is resected and Gerota's fascia is identified and incised, and the adrenal gland is resected.

Laparoscopic anterior transperitoneal approach: This approach may be used for bilateral adrenalectomy with the patient in the supine position. The surgical plan is similar to that of the transabdominal open approach, but is associated with longer operating times and additional trocar sites for placement of more retractors to mobilize the intraabdominal organs.

(Print pagebreak 670)

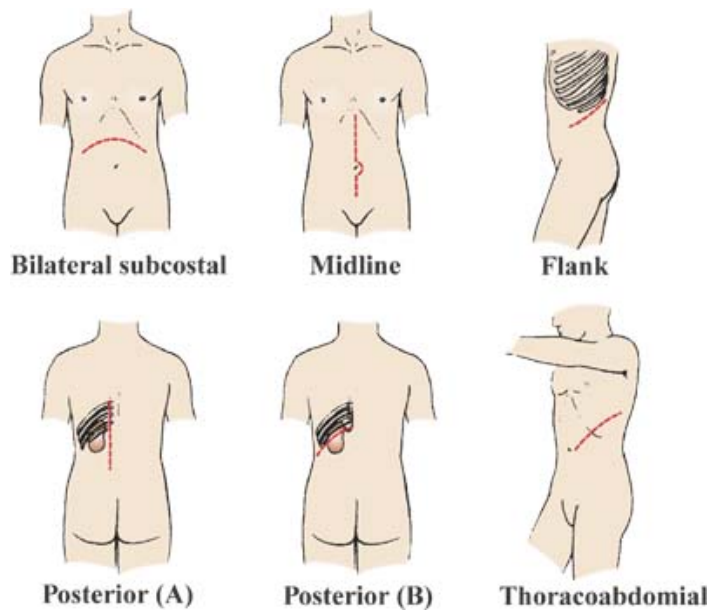


Figure 7.11-8.8. Potential incisions for adrenalectomy. (Reproduced with permission from Baker RJ, Fischer JE: *Mastery of Surgery*, 4th edition. Lippincott Williams & Wilkins, Philadelphia: 2001.)

Laparoscopic lateral transperitoneal approach: This is the preferred approach for adrenalectomy in most centers. The patient is positioned in the lateral decubitus position with the operative site up, and is immobilized with a carefully placed beanbag to minimize nerve compression injuries. The patient is prepped for either an open or laparoscopic approach (so that conversion to an open procedure may be done swiftly, if necessary). If both adrenal glands are to be removed, the right is done first, because there is a slightly higher risk of converting to an open procedure because of the proximity of the adrenal vein to the IVC.

For **right adrenalectomy**, pneumoperitoneum (15 mmHg) is established, with a Veress needle placed in the midclavicular line below the right costal margin. A 5 mm liver retractor is placed, as are two operating trocar sites (usually a 5 mm and a 10 mm or 12 mm port) (Fig. 7.11-9). The liver is retracted cephalad and the retroperitoneum incised with hook cautery. The right renal vein is dissected out carefully and ligated with clips. Dissection of the gland then proceeds from medial to lateral and inferior to superior. The gland is removed through a laparoscopic retrieval bag.

The **left adrenal gland** is approached in a similar fashion, with the Veress needle being placed in the left midclavicular line just below the costal margin. After placement of additional trocars, the peritoneum lateral to the spleen and the splenorenal ligament are incised. With the patient in the decubitus position, gravity will help pull the spleen medially, exposing the anterior surface of the kidney (Fig. 7.11-10). The inferior and medial edges of the gland are dissected first, and the left adrenal vein is identified. After the vein is clipped, the remainder of the gland may be dissected free and the gland removed as above.

Usual preop diagnosis: Hyperaldosteronism; hypercortisolism; pheochromocytoma; metastatic tumor; metastasis; lymphoma; angiomyolipoma; adrenal adenoma; adenocarcinoma

Summary of Procedures





	Open Adrenalectomy	Laparoscopic Adrenalectomy
Position	Transperitoneal: supine; extraperitoneal: prone jackknife	Transperitoneal anterior: supine; transperitoneal lateral: decubitus
Incision	Transperitoneal: midline or bilateral subcostal (Fig. 7.11-8); extraperitoneal: dorsal flank oblique or curved posterior	Transperitoneal anterior: 4–5 trocar incisions (Fig. 7.11-9); transperitoneal lateral: 3–4 trocar incisions
Special instrumentation	Transperitoneal: Denier or Bookwalter retractor; extraperitoneal: none	Transabdominal anterior/lateral: video set-up
Unique considerations	BP monitoring essential for pheochromocytoma patients	
Antibiotics	None	
Surgical time	1–2 h	1–3 h
Closing considerations	Hemostasis; hemodynamic stability	
EBL	100–250 mL	
Postop care	Continue monitoring BP; PACU → ward; ± ICU, based on hemodynamic stability	
Mortality	< 0.5% Overall: 1–12% Bleeding: < 10% Pancreatic fistula: < 1% DVT: 0.8% PE: 0.5%	
Morbidity	Renovascular HTN: < 1% Peroneal nerve palsy: < 1% Venous thrombosis with embolism (Cushing's): 2%	
Pain score	6–8	5–6

(Print pagebreak 671)

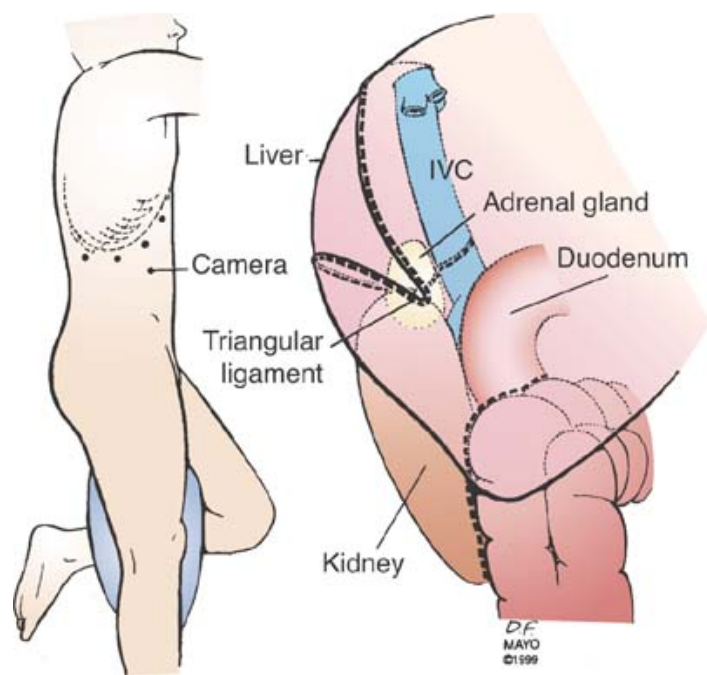


Figure 7.11-9. 9. Port placement for laparoscopic right adrenalectomy. Relevant anatomy is displayed through the right lobe of the liver. (Reproduced with permission from Baker RJ, Fischer JE: *Mastery of Surgery*, 4th edition. Lippincott Williams & Wilkins, Philadelphia: 2001.)

(Print pagebreak 672)



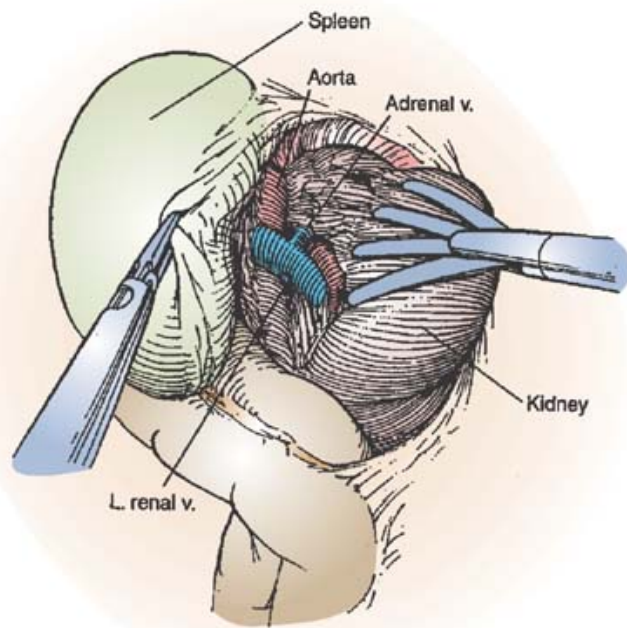


Figure 7.11-10. 10. Exposure of left adrenal after mobilization of the spleen. (Reproduced with permission from Baker RJ, Fischer JE: *Mastery of Surgery*, 4th edition. Lippincott Williams & Wilkins, Philadelphia: 2001.)

Patient Population Characteristics

Age range	13–75 yr
Male:Female	1:2.8
Incidence	Pheochromocytoma: 0.4–2% of all hypertensive patients Primary hyperaldosteronism: 4% of all hypertensive patients Cushing's syndrome: 6/1 million
Etiology	Adenoma or adenocarcinoma (90%); ectopic ACTH (15% of patients with Cushing's); Cushing's syndrome (hyperadrenocorticism) (10–15%); Conn's syndrome (hyperaldosteronism). Idiopathic hyperplasia is usually treated medically.
Associated conditions	HTN (75–80%); diabetes mellitus (10–15%)

Anesthetic Considerations

Preoperative

Cushing's syndrome: Hyperadrenocorticism can be due to adrenal hyperplasia, adrenal carcinoma, pituitary hypersecretion (Cushing's disease), hypersecretion from exogenous tumor, or exogenous steroid administration (most common). Adrenalectomy is the traditional treatment for hyperadrenocorticism 2° adrenal carcinoma. These typically moon-faced patients present with one or more of the following: HTN 2° glucocorticoids, renin (usually not severe); renal calculi; osteoporosis; glucose intolerance; personality changes; and myopathy. In addition, a fragile vasculature predisposes these patients to easy bruising and difficult vascular access.

(Print pagebreak 673)



Respiratory

Cardiovascular

Renal

Neurological

Musculoskeletal

Hematologic

Laboratory

Premedication

Patient may be obese with all attendant problems of morbid obesity (see [Preoperative Considerations in Operations for Morbid Obesity, p. 502](#)).

Tests: As indicated from H&P.

HTN, hypervolemia, dysrhythmias 2° hypokalemia

Tests: ECG; orthostatic vital signs; consider ECHO or MUGA scan, to evaluate LV function, if indicated from H&P.

Excess steroid production → Na⁺retention, K⁺excretion and glucose intolerance (hyperglycemia).

Tests: UA; creatinine; glucose; urine concentrations of catecholamine metabolites; others as appropriate

Psychiatric changes, headache

Striae, muscular wasting, buffalo hump, truncal obesity, thin skin, easy bruisability, osteopenia (compression fractures), weakness

Polycythemia

Tests as indicated from H&P.

Midazolam (1–2 mg iv), consider withhold if morbidly obese

Conn's syndrome: Hyperaldosteronism can be primary (Conn's syndrome—adrenal adenoma or hyperplasia) or secondary (caused by excess renin secretion related to renal dysfunction). These patients are typically hypokalemic and alkalotic → muscle weakness, paresthesias, tetany and polyuria. They may also be hypervolemic → CHF, hypernatremic and hypertensive (diastolic).

Respiratory

Cardiovascular

Renal

Musculoskeletal

Laboratory

Premedication

Respiratory muscle weakness.

Tests: As indicated from H&P.

HTN, dysrhythmias, T wave, + U wave

Tests: ECG; orthostatic vital signs; consider ECHO or MUGA scan, to evaluate LV function, if indicated from H&P.

Renal HTN, polyuria, polydipsia, K⁺Na⁺ Correction of hypokalemia requires > 24 h supplemental K infusion (e.g., 5–20 mEq/h).

Tests: UA; creatinine; glucose; urine concentrations of catecholamine metabolites; others as appropriate

Conn's syndrome: muscle weakness, tetany, sensitivity to muscle relaxants, osteoporosis

Tests as indicated from H&P.

Spironolactone often given to inhibit excess aldosterone effects.

Midazolam (1–2 mg iv); hydrocortisone 100 mg q 8 h.

Intraoperative

Anesthetic technique: GETA (± epidural for postop analgesia). If postop epidural analgesia is planned, placement of catheter prior to anesthetic induction is helpful in establishing correct placement in the epidural space and assuring a bilateral block (accomplished by placing 5–7 mL of 1% lidocaine via the epidural and eliciting a segmental block). Epidurals cannot be used for a posterior approach since they are in the operative field.

Induction

Gentle iv induction (titration to effect with STP or etomidate) and muscle relaxation (vecuronium 0.1 mg/kg). Patient should be adequately anesthetized prior to any stimulation. Unopposed parasympathetic response to laryngoscopy may → bradycardia/asystole.

Volatile anesthetic (isoflurane), opiate, muscle relaxant. N₂O may cause bowel distention and is best avoided. Local anesthetic (2% lidocaine 3–5 mL q 60 min) can be injected into the epidural catheter to provide both anesthesia and optimal surgical exposure (contracted bowel and profound muscle relaxation). A continuous infusion of local anesthetic (e.g., 2% lidocaine or 0.25% bupivacaine) at 3–5 mL/h may enhance hemodynamic stability. Some anesthesiologists will not use the epidural catheter intraop because chemical “sympathectomy” is more difficult to reverse. If epidural opiates are used for postop analgesia, a loading dose (e.g., hydromorphone 0.4–1.0 mg) should be

Maintenance





administered at least 1 hour before the conclusion of surgery. Systemic sedatives (droperidol, opiates, benzodiazepines, etc.) should be minimized during this type of anesthetic as they increase the likelihood of postop respiratory depression. Depends on ease of the surgical procedure and the hemodynamic stability of the patient intraop. If patient is hemodynamically unstable, hypothermic, or has a large 3rd-space fluid requirement, consider postop ventilation.

Emergence

Blood and fluid requirements

Anticipate large fluid loss.
IV: 14–16 ga × 2
NS/LR @ 10–15 mL/kg/h
Warm all fluids.
± Cell Saver

As blood loss can be significant, blood should be immediately available. If procedure does not involve cancer, cell-saving devices can be utilized. Guide fluid management by EBL, known volume deficits, UO, and filling pressures/CO if available.

Monitoring

Standard monitors ([p. B-1](#))
UO

Others as clinically indicated. Forced air warmer useful for maintaining body temperature.

Complications

Labile HTN

Surgical manipulation of the adrenal may cause ↑↑ BP and dysrhythmias. Rx: Alert surgeon and control BP with esmolol/SNP.

Positioning

and pad pressure points
eyes

Strict attention to patient positioning, padding and taping are important in patients with glucocorticoid excess because of osteopenia and thin, easily traumatized skin.

(Print pagebreak 674)

Postoperative

Complications

Pneumothorax
Hypoglycemia
Hypoadrenocorticism after tumor resection
Cushing's syndrome:
Hypoventilation 2° to obesity
(hypoxemia, hypercarbia)
HTN

Dx: pleuritic chest pain, dyspnea, ↑ RR, ↓ breath sounds, hypoxemia. CXR. Rx: O₂ chest tube and reintubation as necessary.

Consider glucocorticoid and mineralocorticoid replacement—hydrocortisone 100 mg q 8 h.

Pain management

Epidural analgesia ([p. C-1](#)).
PCA ([p. C-3](#))

Patient should be recovered in an ICU or ward accustomed to treating the side effects of epidural opiates (e.g., respiratory depression, breakthrough pain, nausea, pruritus).

Tests

CXR; ECG; electrolytes; glucose

Anesthetic Considerations for Pheochromocytoma

Pheochromocytoma: Tumors of chromaffin tissue origin release massive amounts of catecholamines (norepinephrine > epinephrine) and are responsible for the patient's clinical presentation. The tumor is usually found unilaterally in one of the adrenal glands, but also can be found anywhere in the body that chromaffin tissue arises (*Print pagebreak 675*) (e.g., urinary bladder, sympathetic chain). There is an increased incidence of pheochromocytoma in certain diseases (multiple-endocrine neoplasia II, neurofibromatosis, tuberous sclerosis, Sturge-Weber syndrome, von Hippel-Lindau disease).

These patients require extensive preoperative preparation, consisting of α-blockade (phenoxybenzamine 40–400 mg/d × 2 wk) and should ideally be monitored closely by the anesthesiologist who is to provide the intraoperative management. In contrast to its use in essential HTN, β-blockade is not indicated to control hypertension in patients with pheochromocytoma and is dangerous when used alone. When used alone, β-blockade may cause serious hypertension, ↓ CO, intense vasoconstriction → CHF, organ ischemia, and shock (unopposed α-agonism). Patients with tachydysrhythmias may require some β-blockade only after institution of the α-





blockers to control reflex tachycardia. Titration of adrenergic blockade may take 2–4 wk prior to surgical removal of the tumor and should be titrated to relief of all episodic symptoms (HAs, sweating, palpitations) and control of blood pressure. Inadequate preop preparation will increase the perioperative morbidity of patients with pheochromocytomas. The adequacy of medical therapy is assessed by the absence of symptoms of catecholamine excess, BP in the normal range. “Resetting” of autoregulation, including cardiac “recovery” from catecholamine-induced myocarditis and organ recovery from ischemia, may be an important process that prepares the patient to tolerate the surgery and post-tumor removal period. It is anticipated that some degree of postural hypotension will be observed during titration of phenoxybenzamine. At a minimum, a BP of = 160/90 (usually much better than this can be achieved) on two measurements in the 36 hours preceding surgery, SBP dropping by > 15% on standing, but not less than an absolute BP of 80/45; no ST-T wave changes, and resolution of episodic symptoms, for 2 weeks prior to surgery is the goal. There is no rush to surgery.

Respiratory

Cardiogenic pulmonary edema

Cardiovascular

Paroxysmal/continuous HTN, tachydysrhythmias, orthostatic hypotension, hypovolemia, myocardial dysfunction, cardiomyopathy, ventricular ectopy, ↓ intravascular volume, ↓ sensitivity of a and b (cardiac)-receptors to normal levels of catecholamines, CHF, acute myocarditis.

Tests: ECG; ± CK/TP enzymes, orthostatic vital signs; ECHO to evaluate LV function and mitral valve

HTN from excess catecholamine state may damage kidneys; hyperglycemia.

Renal

Tests: UA; creatinine; glucose; urine concentrations of catecholamine metabolites; others as appropriate

Neurological

Sx include: Tremulousness, headache, anxiety, nervousness, personality changes, psychosis (rare), paresthesia in arms, hypertensive retinopathy, dilated pupils.

Musculoskeletal

Weight loss, weakness, fatigue

Hematologic

Polycythemia 2° hemoconcentration (common) or tumor erythropoietin production (rare)

Laboratory

Tests as indicated from H&P.

Premedication

Midazolam (0.025–0.05 mg/kg iv). Preop steroid replacement if bilateral adrenalectomy is contemplated.

Anesthetic technique: GETA: Consider establishing invasive hemodynamic monitoring (arterial and PA lines) PRIOR to induction (this is not difficult, can be done safely, and provides all hemodynamic information to the anesthesiologist from the start; and also lets the anesthesiologist pay attention to the patient at the start of the surgery, rather than being distracted with placing invasive lines after induction) using iv fentanyl (50–200 mcg) and generous local anesthesia. Femoral arterial pressure monitoring is preferred over radial artery, because of the concern over monitoring “central” arterial pressures in a patient who may experience high catecholamine levels during surgery with intense vasoconstriction. If postop epidural analgesia is planned (open surgical procedures), placement of catheter prior to anesthetic induction is helpful in establishing correct placement in the epidural space and assuring a bilateral block (accomplished by placing 5–7 mL of 2% lidocaine **without epinephrine**) via the epidural and eliciting a segmental block. Consider administering epidural narcotic (Dilaudid 0.4–1.0 mg) prior to skin incision. Epidurals cannot be used for a posterior approach since they are in the operative field. Epidurals are not indicated for laparoscopic procedures. Epidurals block neurally mediated sympathetic responses and may → ↓ tumor release of catecholamines, but (*Print pagebreak 676*) do not block catecholamine release resulting from direct surgical manipulation of tumor. Local anesthetic-induced sympathetic block after tumor resection may compound ↓ BP at this important stage in the operation.

Induction

Gentle iv induction (titration to effect with propofol) and muscle relaxation (vecuronium 0.1 mg/kg). Deep mask GA prior to laryngoscopy. Consider double DL with LTA spray (4% lidocaine).

Volatile anesthetic (isoflurane), opiate, muscle relaxant. Systemic sedatives (opiates, benzodiazepines, etc.) should be minimized during this type of anesthetic as they increase the likelihood of postop respiratory depression.

Intraop HTN Rx:

1. Na nitroprusside 0.5–3.0 mcg/kg/min ± esmolol 50–300 mcg/kg/min
2. Fenoldopam 0.1–1.5 mcg/kg/min
3. Nitroglycerin 0.5–10 mcg/kg/min

The surgeon may need to stop manipulating the tumor in order to control BP. Lidocaine





Maintenance

may be needed for ventricular ectopy (especially with epinephrine-secreting tumors).

Hypotension (after tumor removal) Rx:

1. anticipation → fluid loading prior to tumor removal
2. fluid resuscitation based on PA pressures, TEE, stroke volume response
3. inotropes (epinephrine) if ↓ CO
4. vasopressor (phenylephrine, NE,) if ↓ MAP with normal/ ↑ CO

Good communication with surgical team is very important, especially when the ↑↑ adrenal gland is being mobilized, and when the tumor veins are being ligated.

Depends on ease of the surgical procedure and the hemodynamic stability of the patient after tumor removal. If patient is hemodynamically unstable, hypothermic, or has a large 3rd-space fluid requirement, consider postop ventilation, as with any other surgical case. Most cases done laparoscopically can be extubated.

Emergence

Blood and fluid requirements

Prepare for large fluid loss (unusual).

IV: 14–16 ga × 2

NS/LR @ 10–15 mL/kg/h

Warm all fluids.

± Cell Saver

As blood loss can be significant, blood should be immediately available. If procedure does not involve cancer, cell-saving devices can be utilized. Guide fluid management by UO, filling pressures, CO, TEE.

Others as clinically indicated (e.g., PA catheter for patients with pheochromocytoma). Forced air warmer useful for maintaining body temperature. The use of TEE may be helpful in establishing fluid status and other hemodynamic parameters.

Monitoring

Standard monitors ([p. B-1](#)) UO Arterial line (femoral artery) CVP/±PA catheter ± TEE

Surgical manipulation of the adrenal will cause ↑↑ BP ± dysrhythmias. Rx: Alert surgeon and control BP with SNP/fenoldopam/TNG ± esmolol.

↓↓ BP common after removal of tumor. May be severe and life-threatening. Rx: anticipate event; fluids, phenylephrine or epinephrine infusions depending on need for inotropic support.

Complications

↑↑↑ BP and dysrhythmias

↓↓BP (postexcision – most dangerous part of anesthetic)

Positioning

and pad pressure points
eyes

(Print pagebreak 677)

Postoperative

Complications

Pneumothorax

Hypoglycemia

Dx: pleuritic chest pain, dyspnea, ↑ RR, ↓ breath sounds, hypoxemia. CXR. Rx: O₂ chest tube and reintubation as necessary.

Hypoadrenocorticism after tumor resection (usually only with bilateral tumors)

Consider glucocorticoid and mineralocorticoid replacement → hydrocortisone 100 mg q 8 h.

HTN

Patient may have other cause for HTN.

Hypotension

Continue vasopressor/inotropes, fluids as indicated. Patient should be recovered in ICU overnight.

Myocardial dysfunction

Pulmonary edema

Patient should be recovered in an ICU or ward accustomed to treating the side effects of epidural opiates (e.g., respiratory depression, breakthrough pain, nausea, pruritus).

Pain management

Epidural analgesia ([p. C-2](#)). PCA ([p. C-3](#))

Tests

CXR; ECG; electrolytes; glucose





Suggested Readings

1. Brunt, LM: Laparoscopic adrenalectomy. In: *Current Surgical Therapy*, 7th edition. Cameron JL, eds. Mosby Inc., St. Louis: 2001, 1460–7.
2. Cooper ZA, Mihm FG: Blood pressure control with fenoldopam during excision of a pheochromocytoma *Anesthesiology* 1999; 91(2):558–60.
3. Kebebew E, Siperstein AE, Clark OH et al: Results of laparoscopic adrenalectomy for suspected and unsuspected malignant adrenal neoplasms. *Arch Surg* 2002; 137(8):948–53.
4. Mihm FG: Pheochromocytoma: decreased perioperative mortality *Anesth Clin North Am* 1998; 16(3):645–62.
5. Olson JA: Adrenal gland. In: *Greenfield's Surgery, Scientific Principles and Practice*, 4th edition. Mulholland MW, Lillemore ED, Doherty GM eds. Lippincott Williams & Wilkins, Philadelphia: 2006, 1334–53.
6. Prys-Roberts C: Phaeochromocytoma—recent progress in its management. *Br J Anaesth* 2000; 85:44–57.
7. Schwartz JJ, Rosenbaum SH: Anesthesia and the endocrine system. In: *Clinical Anesthesia*, 5th edition. Barash PG, Cullen BF, Stoelting RK, eds. Lippincott Williams & Wilkins, Philadelphia: 2006, 1129–51.
8. Sieber FE: Evaluation of the patient with endocrine disease and diabetes mellitus. In: *Principles and Practice of Anesthesiology*, 2nd edition. Longnecker DE, Tinker JH, Morgan GE Jr, eds. Mosby-Year Book, St Louis: 1998, 303–22.
9. Smith CD, Weber CJ, Amerson JR: Laparoscopic adrenalectomy: New gold standard. *World J Surg* 1999; 23(4):389–96.
10. Witteles RM, Kaplan EL, Roizen MF: Safe and cost-effective preoperative preparation of patients with pheochromocytoma. *Anesth Analg* 2000; 91:302–4.

