# PHEOCHROMOCYTOMA AND ANESTHESIA

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# PHEOCHROMOCYTOMA AND ANESTHESIA

Pheochromocytomas are catecholamine-secreting tumors that arise independently or as part of the MEN syndromes (Table 19-9). Patients with von Recklinghausen's disease, tuberous sclerosis, and Sturge-Weber syndrome have increased risk of pheochromocytoma.

## A. Signs and Symptoms.

Hypertension, headache, sweating, pallor, and palpitations are classic signs and symptoms. Hypertensive crises mimic the hemodynamic responses of acute catecholamine administration. Cardiomyopathy, cardiac hypertrophy, (ECG) abnormalities and arrhythmias can occur. The presentation of a pheochromocytoma may mimic thyrotoxicosis, malignant hypertension, diabetes mellitus, malignant carcinoid syndrome, or gram-negative septicemia. Hyperglycemia, but rarely frank diabetes, occurs in most patients owing to catecholamine stimulation of glycogenolysis and inhibition of insulin release.

## B. Diagnosis.

Diagnosis relies on demonstration of excess catecholamine secretion. The most sensitive test for high-risk patients (familial pheochromocytoma or classic symptoms) is a finding of plasma-free normetanephrine greater than 400 pg/mL and/or metanephrine greater than 220 pg/mL. A pheochromocytoma is excluded if normetanephrine is less than 112 pg/mL and metanephrine is less than 61 pg/mL. Determination of elevated urinary free catecholamine levels and their metabolites (i.e., metanephrine, normetanephrine, vanillylmandelic acid) is easy to perform and readily available and is sufficient for patients with a low probability of having a pheochromocytoma. Results of these tests are equivocal in 5% to 10% of patients, in which case a clonidine suppression test may be used (lowers plasma catecholamines in patients without pheochromocytoma but not in patients with pheochromocytoma). A positive glucagon stimulation test result is the safest and most specific provocative

testing method (causes tumor release of catecholamines of at least three times baseline or >2000 pg/mL within 1 to 3 minutes), but the test should be limited to patients with diastolic blood pressure of less than 100 mm Hg. Tumor location can be predicted by the pattern of catecholamine production. CT and magnetic resonance imaging (MRI) are the optimal noninvasive anatomic adrenal imaging studies.

**C. Treatment.**

Treatment **is surgical excision whenever possible**. α-Methylparatyrosine (inhibits the rate-limiting step of the catecholamine synthetic pathway) may decrease catecholamine production by 50% to 80%. Usual doses range from 250 mg twice daily to 3 to 4 g/day. It is especially useful for malignant and inoperable tumors. Side effects include extrapyramidal reactions and crystalluria.

D. **Management of Anesthesia (Table 19-11)**

1. **Preoperative Management**.

   Most pheochromocytomas secrete predominantly norepinephrine. ***The therapeutic goal is normotension, resolution of symptoms, elimination of ST-T changes on the ECG, and elimination of arrhythmias.***

   **a. α-Blockade**

   α-Blockade (with phenoxybenzamine, a noncompetitive α1-antagonist, or alternatively prazosin, an α1-competitive blocker) is used to lower blood pressure, increase intravascular volume, prevent paroxysmal hypertensive episodes, allow resensitization of adrenergic receptors, and decrease myocardial dysfunction. Because of the prolonged effect of phenoxybenzamine on α-receptors, it has been recommended that it be discontinued 24 to 48 hours before surgery or alternatively that only one half to two thirds of the morning dose be administered before surgery to avoid vascular unresponsiveness immediately after removal of the tumor. Hypertension usually occurs with manipulation of the tumor.

b. **β-Adrenergic** Blockade

(Propranolol, Atenolol, Metoprolol). β-Adrenergic blockade is prescribed if tachycardia (i.e., heart rate >120 beats/min) or other arrhythmias result after α2-blockade with phenoxybenzamine.

***Esmolol*** has a fast onset and short elimination half-life and can be administered IV in the immediate preoperative period.

**A nonselective β-blocker** *should never be administered before α-blockade because* blockade of vasodilatory β2-receptors results in unopposed α-agonism, resulting in vasoconstriction and hypertensive crises.

c. **α-Methylparatyrosine**.

In combination with phenoxybenzamine during the preoperative period, α-methylparatyrosine may facilitate intraoperative hemodynamic management.

d. **Calcium Channel Blockers** (Nifedipine, Diltiazem, Verapamil) and **ACEIs (Captopril).**

These agents have all been used to control preoperative hypertension.

***An α1-blocker plus a calcium channel blocker*** is an effective combination for resistant cases.

2. **Intraoperative Management.**

Goals are avoidance of drugs or maneuvers that provoke catecholamine release or potentiate catecholamine action, and maintenance of cardiovascular stability. Hypertension can occur during pneumoperitoneum or tumor manipulation. Hypotension may follow ligature of the tumor's venous drainage. Hypoglycemia after tumor vein ligation can occur owing to increased insulin levels.

3. **Postoperative Management**.

Plasma catecholamine levels do not return to normal for 7 to 10 days, and 50% of patients are hypertensive for several days after surgery. Hypotension is the most frequent cause of death in the immediate postoperative period.

**TABLE 19-11 ■ Anesthetic Considerations in Pheochromocytoma**

| | |
|---|---|
| Preoperative | α-Blockade; phenoxybenzamine, prazosin α-methylparatyrosine<br>β-Blockade for tachycardia (propranolol, metoprolol, atenolol)<br>Calcium channel blockers, ACEIs |
| Intraoperative | Avoid fear, stress, pain, shivering, hypoxia, hypercarbia (stimulate catecholamine release)<br>Catecholamine release during induction, intubation, surgical incision, abdominal exploration, and tumor manipulation<br>Monitoring: arterial catheter, CVP or PA catheter, Foley catheter<br>Significant fluid administration possible to prevent hypotension after tumor removal<br>All anesthetics are used<br>Drugs to avoid: morphine and atracurium (histamine release can provoke catecholamine release), atropine, pancuronium, and succinylcholine (may stimulate the sympathetic nervous system), halothane (sensitizes the myocardium to catecholamine-induced arrhythmias), droperidol, chlorpromazine, metoclopramide, and ephedrine (hypertensive responses)<br>Intraoperative hypertension: treat with sodium nitroprusside or phentolamine<br>Antiarrhythmics to have available: lidocaine, esmolol, amiodarone<br>Intraoperative blood salvage |
| After tumor removal | Hypotension caused by catecholamine decrease: give fluids, decrease anesthetic depth; administer vasopressors if these measures fail<br>Hypoglycemia caused by increased insulin levels: monitor glucose, use dextrose-containing intravenous fluids<br>Glucocorticoid therapy if bilateral adrenalectomy |
| Postoperative | Hypertension may persist for several days; consider persistent hypertension or residual tumor<br>Hypotension is most common cause of death<br>Hypoglycemia may persist<br>ICU monitoring for at least 24 hours |

*ACEI*, Angiotensin-converting enzyme inhibitor; *CVP*, central venous pressure; *ICU*, intensive care unit; *PA*, pulmonary arterial.