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Unsuspected Pheochromocytoma in a Surgical Patient

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Hypertensive patients have been shown to have an increased incidence of cardiac arrest during operation. Additional risks may be involved, depending on the cause of the hypertension. If it is due to pheochromocytoma, there may be a fatal hypertensive crisis, arrhythmia, cerebrovascular accident, or acute left heart failure and pulmonary edema without preoperative management. These potential complications and the fact that pheochromocytoma is a correctable cause of hypertension are important reasons to exclude the presence of this tumor in hypertensive patients about to undergo anesthesia. The following case illustrates problems that may occur when this is not done.

REPORT OF A CASE

A 70-year-old man, seen in January 1967 complaining of hoarseness of 4 months’ duration, had erosion of the right vocal cord posteriorly and lesions along the left vocal cord. Malignancy was suspected, and laryngeal suspension and biopsy under general anesthesia was planned. The preanaesthetic medical evaluation was essentially negative except for a vague history of recent increase in blood pressure. At examination, the blood pressure was 150/90 mm Hg, pulse rate 74/min, and weight 121 kg. The heart and lungs were normal. Routine laboratory results were within normal limits. No electrocardiogram was obtained.

When the patient was placed on the operating table, the blood pressure was 150/90 mm Hg and pulse rate, 75/min. During preparation for induction of anesthesia, the blood pressure increased to 210/90 mm Hg. Atropine (0.4 mg) was given iv, and anesthesia was induced with thiopental (375 mg, iv). A mixture of halothane, nitrous oxide, and oxygen was given for maintenance anesthesia, with spontaneous ventilation. Shortly after induction, cardiac arrest occurred. An endotracheal tube was inserted immediately, controlled ventilation with 100 per cent oxygen was started, and external cardiac massage was instituted. Within a few seconds, a heartbeat was heard and spontaneous respiration resumed. Nevertheless, two additional cardiac arrests occurred during the next few minutes. Ten defibrillations were necessary before cardiovascular stability was achieved. The surgical procedure was cancelled and the patient was transferred to the intensive care unit (ICU). While there, his blood pressure varied between 270/150 and 160/90 mm Hg but stabilized at about 190/90 mm Hg. No treatment was thought necessary, and the cause of his hypertension or of his cardiac arrest was not determined. He was dismissed from the hospital on the eleventh day after cardiac arrest.

Two weeks later, laryngeal suspension with biopsy was completed under local anesthesia. Examination of the biopsy specimen revealed carcinoma of the right vocal cord. Radiotherapy rendered this disease process inactive, and periodic checks confirmed its inactivity for nearly 7 years.

During this interval, general medical care was provided by the patient’s local physician, who prescribed an antihypertensive agent and potassium supplementation in 1973. Four months after the biopsy procedure, the patient had reported having “spells,” lasting as long as 2 hours, when he would feel “mean,” but these were not evaluated further.

In late 1973, an anterior web and granularity of the cords were noted. Laryngeal suspension with biopsy under general anesthesia was considered necessary. During this preanesthetic medical evaluation, the blood pressure was 190/110 mm Hg, pulse rate 70/min, and weight 112 kg. Examinations

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of the heart, lungs, lymph nodes, and abdomen gave normal results. Abnormal laboratory data included ECG evidence of previous myocardial infarction, serum creatinine 1.53 mg/dl, and glucose 105 mg/dl. The thoracic roentgenogram revealed a prominent left ventricle and calcification of the aorta.

Premedication was injection of atropine (0.4 mg), meperidine (50 mg), and diazepam (10 mg). The patient was quite drowsy when placed on the operating table 45 minutes later; his blood pressure was 220/100 mm Hg. Intravenous fluid administration was started, an ECG monitor was attached, and 100 mg lidocaine were given prophylactically. The patient was given 100 per cent oxygen for 5 minutes, after which anesthesia was induced with thiopental (475 mg). A 6-mm oral endotracheal tube was inserted without difficulty after intravenous injection of succinylcholine (60 mg). Maintenance anesthesia was with 1 to 5 percent enflurane in nitrous oxide and oxygen. Systolic blood pressure varied between 150 and 190 mm Hg throughout the 10-minute procedure; pulse rate varied between 90 and 120/min.

After the procedure was completed, 100 per cent oxygen was given, the endotracheal tube left in place, and ventilation was assisted. After approximately 8 minutes, the patient abruptly opened his eyes, moved his upper extremities, and responded to verbal commands. The trachea was then extubated. The systolic blood pressure, which had been between 140 and 160 mm Hg throughout emergence from anesthesia, exceeded the upper limit of the scale (300 mm Hg) at the first check after extubation of the trachea. Pulse rate was 130/min. At this point, multifocal premature ventricular contractions first occurred. Suppression was achieved with lidocaine, 60 mg iv, at which time the systolic blood pressure decreased to 220 mm Hg.

Upon arrival of the patient in the recovery room, systolic blood pressure was 250 mm Hg, but it increased within 2 minutes to more than 300 mm Hg. Premature ventricular contractions were treated with 50 mg lidocaine; 5 mg of diazepam were given. The premature ventricular contractions were again suppressed, and systolic blood pressure decreased to 185 mm Hg, but approximately 4 minutes later the pressure returned to more than 300 mm Hg, with a pulse rate of 140 to 160/min. The patient was awake, oriented, talkative, and without complaint. When the pressure increase persisted, phenolamine, 2 mg, was given iv. The systolic pressure quickly decreased to 140 mm Hg but returned to more than 300 mm Hg within 10 minutes. Over the next 90 minutes, phenolamine was needed four times to bring the systolic blood pressure to below 300 mm Hg. The premature ventricular contractions were suppressed with lidocaine. Propranolol was employed on three occasions to control ventricular tachycardia. Blood samples were drawn for catecholamine determinations, and the patient moved to the ICU.

In the ICU, a lidocaine drip controlled the arrhythmias. Blood pressure was maintained in the range of 250/120 to 200/100 mm Hg with phenolamine. Episodes of ventricular tachycardia were treated with propranolol. When a urinary metanephrine level of 38.7 mg/24 h was reported, treatment with phenoxybenzamine, 10 mg every 4 hours, and propranolol, 10 mg every 4 hours, was started.

Phenolamine therapy was discontinued after 5 days. Phenoxybenzamine and propranolol were continued up to and including the fifteenth day, when a pheochromocytoma, revealed by nephro- togram, was removed with the right adrenal gland.

When the patient was dismissed from the hospital, his blood pressure was 170/80 mm Hg.

**DISCUSSION**

A sudden sustained increase in blood pressure in a patient recovering from anesthesia is rare. In this case, the evidence indicates that the acute increase in blood pressure was a consequence of a pheochromocytoma. Presumably, during recovery from anesthesia, straining on the endotracheal tube prior to its removal or the cough produced by its removal stimulated the release of sufficient amounts of catecholamines from the pheochromocytoma to cause hypertension. This raises the question whether the cause of the cardiac arrest 7 years previously was also the pheochromocytoma. Although little evidence other than persistent hypertension supports the presence of a pheochromocytoma at the time of the previous anesthesia and cardiac arrest, it must be considered a good possibility. Additionally, the "mean spells" the patient reported 4 months after the cardiac arrest are suggestive of a functioning pheochromocytoma. Logicilly, then, one must ask why he did not experience cardiac arrest during the second anesthesia.

A possible explanation lies in the differences in anesthetic technique on the two occasions: 1) preanesthetic medication was heavy; 2) controlled instead of spontaneous ventilation was accomplished through the use of an endotracheal tube; 3) enflurane was used for maintenance anesthesia instead of halothane; 4) lidocaine was given prophylactically. On the other hand, no arrhythmia occurred after the injection of succinylcholine, although arrhythmias have been associated with its use in a patient with pheochromocytoma.3
This case illustrates the need to determine preoperatively the cause of severe hypertension that is untreated or inadequately treated. In a recent study of cardiac arrest in the operating area, 50 per cent of the arrests were in patients who were hypertensive, and 10 of the 12 having an arrest had not received treatment for their hypertension. Certainly, in the case of a hypertensive patient who has had cardiac arrest or a hypertensive crisis during a previous induction of anesthesia, the cause of the hypertension must be sought. In these cases, pheochromocytoma should be considered a possibility and should be specifically ruled out. Although the yield of pheochromocytoma screening procedures applied to all hypertensive patients is less than 1 per cent, preoperative preparation including α- and β-adrenergic receptor blockade in patients with pheochromocytoma decreases the intraoperative incidence of acute increases in blood pressure, arrhythmia, and cardiac arrest.

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A Device to Measure Closing Capacity with Positive Pressure


Measurement of closing capacity (CC) and its relationship to functional residual capacity (FRC) have important implications with regard to pulmonary gas exchange. Closing capacity can be determined only in subjects capable of consciously cooperating with the ventilatory maneuvers involved. Since airway closure may frequently occur in anesthetized or comatose subjects, the measurement of closing capacity may be of value in their evaluation.

In this paper we describe the construction and use of a 5-liter syringe to allow controlled ventilation duplicating the spontaneous ventilatory maneuvers required to measure closing capacity. Results are compared with values obtained during voluntary performance of the standard nitrogen method for measurement of closing capacity.

PROCEDURE

We constructed a 5-liter syringe (fig. 1) using a ½-inch brass cylinder 4 inches in internal diameter and a hand-driven reciprocating Teflon-sealed piston. The position of the piston and thus volume within the syringe was determined by a rack mounted on a connecting rod which drove a pinion gear attached to a potentiometer. Volume per unit of piston movement was calibrated with a 9-liter Collins spirometer.

We studied seven unmedicated healthy subjects with no evidence of pulmonary disease by history, physical examination, or chest roentgenogram. Vital capacities and forced expiratory volumes in one second were