posteriorly and occlude the pulmonary arteries. Since the slightest compression of the mass in various directions by the surgeon's hand during the initial resection resulted in severe hypoxia, it may be concluded that positioning the patient in the lateral or sitting position would not have significantly relieved the problem or rendered extracorporeal oxygenation unnecessary. The only position that might possibly have been beneficial would have been the prone position, allowing the mass to fall anteriorly, but the resection would have been technically difficult, if not impossible, with the patient in this position. Partial aspiration of the cyst did not relieve the hypoxia, and since during resection of the tumor it was noted that the cyst did not constitute a significant portion of the tumor mass, it may reasonably be assumed that complete aspiration of the cyst also would not have significantly relieved the compression of the pulmonary artery or have obviated the need for external oxygenation.

The EEG was an essential part of the monitoring in this patient. Sudden and severe hypoxia was immediately diagnosed from analysis of the EEG and was confirmed by simultaneous blood–gas sampling showing low Pa_{O_2} values (fig. 2). This monitoring allowed immediate adjustment of surgical compression or cardiopulmonary bypass flow rates. For example, it was determined that when the surgeon's hands were severely compressing the pulmonary arteries, as was unavoidable during parts of the resection, the flow rate had to be maintained at 2,000 ml/min for adequate oxygenation, whereas at other moments a flow rate of only 500 ml/min was adequate. When enough of the tumor had been removed that oxygenation was achieved with zero flow rates, cardiopulmonary bypass was discontinued. It is our belief that this child could not have survived the induction of anesthesia and the early part of the surgical dissection if extracorporeal oxygenation had not been used.

In summary, a 14-year-old girl had a dermoid cyst removed from her anterior mediastinum with arterial oxygenation maintained by cardiopulmonary bypass to counteract severe compression of the pulmonary artery by the tumor during anesthesia.

Use of Enflurane for Pheochromocytoma Removal

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The surgical mortality associated with pheochromocytoma has been markedly reduced in recent years because of improvements in medical, surgical and anesthesiologic management.

Crout and Brown[1] described a rational approach for the anesthetic management of pheochromocytoma. Pretreatment with phenoxybenzamine (Dibenzyline) an alpha-adrenegic receptor blocker, controls blood pressure and expands plasma volume. Beta-adrenergic receptor blockers are used only if needed to control sinus tachycardia or ventricular arrhythmias. The use of methoxyflurane as the anesthetic agent prevents ventricular arrhythmias. However, since methoxyflurane is nephrotoxic, the search for a better anesthetic agent for these patients continues.

The following is a report of a pheochromocytoma in which the regimen described by Crout and Brown was followed, with enflurane (Ethrane) used as the anesthetic agent.

REPORT OF A CASE

A 28-year-old Caucasian man was admitted to the hospital with a six-month history of intermittent episodes of headache, nervousness, sweating, palpitations, and diarrhea. The physical examination was essentially negative except for a pulse rate of 110/min and a blood pressure of 160/100 torr.

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The chest x-ray was unremarkable and the electrocardiogram showed sinus tachycardia and no ventricular arrhythmia. Ptosis of the right kidney was demonstrated by intravenous pyelography. The diagnosis of pheochromocytoma was made on the basis of 24-hour urine collections which contained 1,276 and 1,386 μg of total catecholamines on two separate occasions, and 21 mg of metanephrine and 13 mg of vanillylmandelic acid.

In the hospital, the blood pressure was 120/70 torr and pulse rate 100–110/min. On the third day after admission headache and palpitations developed and the blood pressure was recorded as 140/100 torr. Six days before operation the following schedule of phenoxybenzamine was started: 10 mg, *b.i.d.*, 2 days; 20 mg, *b.i.d.*, 2 days; 40 mg, *b.i.d.* 2 days. The last dose was given at 8:00 P.M. on the night before operation. Propranolol was not used since the pulse rate was 70–80 and no ventricular arrhythmia was detected by electrocardiogram.

Premedication consisted of pentobarbital sodium (Nembutal), 100 mg, morphine sulfate, 5 mg, and atropine sulfate, 0.4 mg. The patient was awake on arrival to the operating room. Monitors included a central venous pressure line in the right subclavian vein, an arterial line in the left radial artery, Foley catheter, electrocardiogram, sphygmomanometer, precordial and esophageal stethoscopes. The blood pressure was 130/90 torr and the pulse rate 90/min before induction.

Induction of anesthesia was achieved with 200 mg thiopental in divided doses, followed by nitrous oxide, oxygen, and enflurane by face mask. After satisfactory anesthesia had been obtained, the trachea was sprayed with 4 per cent lidocaine, and intubation was facilitated by the use of 100 mg succinylcholine following 3 mg *d*-tubocurarine. No change in blood pressure or arrhythmia was seen during intubation. Anesthesia was maintained with 1–2 per cent enflurane in 50 per cent nitrous oxide in oxygen. Intermittent doses of pancuronium were used to provide adequate relaxation; a total of 6 mg was given in 2½ hours of surgery.

A hypertensive episode occurred when the surgeons explored the right adrenal gland; the blood pressure rose to 220/120 torr and the pulse to 120/min. This was treated with 5 mg phentolamine as a bolus plus an infusion of phentolamine containing 10 mg/100 ml of 5 per cent dextrose in water. The blood pressure returned to 120/80 torr. When dissection of the tumor was started, a second hypertensive episode occurred; it responded again to phentolamine. During both hypertensive episodes a total of 13.5 mg phentolamine was used.

A final hypertensive episode occurred at the end of dissection. This time it was treated with a sodium nitroprusside infusion (50 mg in 1,000 ml of 5 per cent dextrose in water); a total of 750 μg was given, and the blood pressure decreased to 120/80 torr. Because the pulse had increased to 140/min, 1 mg propranolol, iv, was given after the last hypertensive episode, followed by return to normal sinus rhythm. No other arrhythmia was noted during the operation.

A well encapsulated tumor, 6 cm in diameter, was removed from the right adrenal gland. No hypotension occurred after removal of the tumor. The blood loss, estimated to be 400 ml, was not replaced. Total fluid replacement consisted of 1,000 ml of 5 per cent dextrose in lactated Ringer's solution, 900 ml of lactated Ringer's solution, 50 ml of salt-poor albumin, and 250 ml of plasma protein fraction. Central venous pressure remained between 5 and 10 cm H_2O. Arterial blood gases were normal throughout the procedure. Postoperatively, the patient's condition remained stable, and he was discharged from the hospital a week later.

Discussion

Crout and Brown reported a series of 12 cases of pheochromocytoma in which the patients were anesthetized with methoxyflurane and 50 per cent nitrous oxide without development of arrhythmia.[1] They suggested that methoxyflurane was an ideal agent for pheochromocytoma since it does not appreciably lower the arrhythmia threshold of the myocardium to catecholamines.[2] It is well established that methoxyflurane may produce renal damage,[3] and Brown[4] has stated that two of his patients developed high-output renal failure postoperatively. Cooperman *et al.* reported a series of 14 cases of pheochromocytoma in which they used halothane.[5] Ten of the patients had ventricular arrhythmias during anesthesia and were treated with a beta-adrenergic blocker.

Joas and Craig reported three cases in which they used fluroxene as the anesthetic agent.[6] All three patients had electrocardiographic evidence of nodal rhythm or premature atrial contractions which did not require treatment. No ventricular arrhythmia was seen. Based on these data they proposed that fluroxene might be the agent of choice for the surgical removal of a pheochromocytoma. However, counter to this conclusion are the observations that some arrhythmias were present, fluroxene is flammable in concentrations above 4 per cent, and the anesthetic provides poor muscular relaxation.

One of the advantages of the ether type of anesthetic, as opposed to the alkanes, is the decreased incidence of arrhythmias with or without epinephrine.[7] Methoxyflurane, enflurane and fluroxene are examples of ether-type agents. It is our clinical impression that enflurane does not alter the arrhythmia threshold of

the myocardium to catecholamines in man. Thus it seemed to us that enflurane would be a good anesthetic for the surgical removal of a pheochromocytoma. There are no data in man as to the effect of injecting catecholamines during enflurane anesthesia. Virtue *et al.*[8] and McDowell *et al.*[9] have reported ventricular arrhythmias in dogs challenged with epinephrine during enflurane anesthesia. However, in dogs, ventricular arrhythmias are also produced by injection of epinephrine during methoxyflurane anesthesia.[10] This may reflect a species difference.

Enflurane is nonexplosive and produces excellent muscular relaxation. It has been suggested that enflurane is capable of producing renal damage in renal transplants,[11] but there is no evidence that it produces renal damage in the presence of normal renal function. Our experience in this one case suggests that further evaluation of enflurane for surgical removal of pheochromocytomas is warranted.

Note Added in Proof

R. R. Johnston and E. I. Eger, II, reported (Abstract of Scientific Papers, 1974 ASA Annual Meeting, page 53) epinephrine arrhythmic thresholds in man of 2.06 μg/kg during halothane anesthesia and 7.5 μg/kg during enflurane anesthesia.

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Determinants of Etidocaine Concentration in the Blood

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The systemic toxicity of a local anesthetic agent is directly related to its concentration in blood and organs such as brain and heart. A number of studies have been conducted to determine the factors that influence the absorption of local anesthetic agents such as lidocaine, prilocaine, and mepivacaine.[1–3] The blood levels and, thus, the potential toxicities of these specific compounds are affected by site of injection, total dosage administered, the presence of a vasoconstrictor agent in the anesthetic solution, and the pharmacologic characteristics of the drug itself. Etidocaine (Duranest) is a new local anesthetic agent

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