ing pressure in the pulmonary vasculature and heart is the transmural pressure, with the intrapleural pressure as reference. A recent report describing a technique for direct measurement of intrapleural pressure brings us one step closer to the true value for this transmural pressure.[8] However, the ultimate solution is obtaining the left ventricular end-diastolic transmural pressure as it relates to intravascular volume and the inotropic state of the myocardium. The pressure immediately surrounding the heart (pericardial) may be different from the intrapleural pressure[9]; however, the clinical significance of this is yet to be shown.

We offer this report as a partial solution to the problem of accurate measurement of pulmonary vascular pressures. Simultaneous airway pressure and pulmonary vascular pressure recording can resolve the difficulty in choosing the correct pressure value during the end of exhalation.

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Induction of Anesthesia in a Patient with an Undiagnosed Pheochromocytoma

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The responses of a patient with pheochromocytoma to three identical anesthetic inductions are reported. The first was performed prior to diagnosis, the second after treatment with α-methylparatyrosine, and the third after resection of the tumor.

Report of a Case

A 54-year-old black man was scheduled for transsphenoidal hypophysectomy for resection of a pituitary adenoma. He had had one episode of severe, throbbing headache, blurred vision, diaphoresis, and dizziness. The patient had a ten-year history of mild diabetes, and had been treated intermittently for hypertension. Roentgenograms of the skull revealed a large sella turcica with erosion of the posterior wall and floor. Results of physical examination were unremarkable. The patient weighed 72 kg. Hemoglobin was 12.4 g/100 ml, and T4 was 3.4 μg/100 ml (normal 4.2–11.5 μg/100 ml). Blood pressure was 135 ± 14/85 ± 11 torr. The hypothyroidism was treated with liothyronine, and the patient was also given dexamethasone preoperatively.

The patient was premedicated with morphine, 5 mg, and scopolamine, 0.5 mg. An arterial catheter was placed. Blood pressure was 160/80 torr, and pulse rate was 55/min. Meperidine, 100 mg, was given iv, followed a minute later by droperidol, 5 mg. Blood pressure increased to 200/100 torr, and pulse rate increased to 90/min. Thiopental, 250 mg, was then given. Ventilation was controlled by mask. Blood pressure abruptly increased to 280/140 torr, and pulse rate increased to 170/min. S-T segment depression developed, followed by bigeminy. The arrhythmia responded to iv infusion of lidocaine, 50 mg. The blood pressure was unresponsive to trimethaphan, but was reduced to 160/100 torr by sodium nitroprusside infusion. The tachycardia was treated with propranolol, 0.75 mg, which reduced the heart rate to 80/min. During this acute episode arterial blood gases were Pa_{O_2} 269 torr, Pa_{CO_2} 52 torr, *p*H 7.33, and base excess +1.0.

The operation was postponed, and the patient, now alert, was transferred to the surgical intensive care unit. The sodium nitroprusside infusion was withdrawn over several hours, and no further episode of hypertension or tachycardia occurred. Mean 24-hour urinary metanephrine excretion was 15 mg (normal, less than 1 mg). The patient was treated with α-methylparatyrosine, 1,500 mg/day, in three doses. The 24-hour metanephrine excretion decreased to 11.3 mg, and blood pressure was reduced to 126 ± 19/74 ± 15 torr.

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Fifteen days after the first anesthetic administration, the patient returned for resection of pheochromocytoma. An arterial catheter was inserted. Prior to induction of anesthesia, blood pressure was 160/80 torr, and pulse rate was 50/min. The same premedication and induction sequence used for the first anesthetic administration was repeated. There was no change in blood pressure or pulse rate after 100 mg meperidine. Droperidol, 5 mg, increased blood pressure to 200/90 torr, and pulse rate to 90/min. After thiopental, 200 mg, blood pressure rose to 250/125 torr, and pulse rate increased to 120/min. No electrocardiographic changes occurred. Anesthesia was maintained with as much as 4 per cent enflurane. Increases in blood pressure were controlled with phentolamine in 1-mg doses. Laryngoscopy, intubation, and operation proceeded smoothly, and a 180-g pheochromocytoma was resected. The patient needed a total of 25 mg phentolamine during the hour between induction of anesthesia and resection of the tumor. The remainder of the anesthesia was uneventful. Postoperative urinary metanephrine excretion was 0.77 mg/ 24 hours.

Fourteen days after the second anesthetic administration the patient returned for a transsphenoidal hypophysectomy. This time the same induction sequence produced a reduction in blood pressure from 150/70 to 115/50 torr; pulse rate remained constant at 60/min. No anesthetic problem was encountered.

Discussion

Induction of anesthesia with meperidine, droperidol, and thiopental usually results in no change or a slight decrease in blood pressure. This patient, however experienced marked hypertension with tachycardia, S-T segment depression, and bigeminy during induction. The increase in blood pressure occurred prior to any airway manipulation or surgical stimulation. Subsequently, pheochromocytoma was diagnosed, and α-methylparatyrosine therapy was instituted to reduce catecholamine synthesis.[1] In spite of a 27 per cent decrease in urinary metanephrine excretion, this patient had hypertension and tachycardia during a second anesthetic induction. However, the peak blood pressure and maximum pulse rate were lower than those seen during the first anesthetic induction. Furthermore, no S-T segment change or premature contraction occurred.

In both anesthetic inductions, the increase in blood pressure began after injection of droperidol, and was maximal after injection of thiopental. No increase in blood pressure occurred following meperidine injection. Sumikawa and Amakata[2] reported a marked increase in blood pressure after the administration of droperidol to a patient with pheochromocytoma. However, others consider droperidol–fentanyl combinations to be indicated in the anesthetic management of patients with pheochromocytoma.[3–7] These investigators generally cite increased cardiovascular stability and freedom from arrhythmia. The rise in blood pressure following droperidol did not occur during the third anesthetic induction, which followed resection of the tumor.

Engelman *et al.*[1] reported that 1,500 mg of α-methylparatyrosine daily for 14 to 28 days reduced catecholamine production by 50 to 80 per cent, compared to our patient's 27 per cent reduction after 13 days of therapy. They found a marked inprovement in cardiovascular stability intraoperatively in patients treated with this agent, although other observers have been less impressed.[3,8] In our patient, while α-methylparatyrosine did not obviate the need for intraoperative phentolamine, it probably modified the peak blood pressure response, reduced the tachycardia, and prevented ectopic rhythms.

In conclusion, a patient with undiagnosed pheochromocytoma had an episode of severe hypertension after administration of droperidol. This response to droperidol was modified, but not eliminated, by pretreatment with α-methylparatyrosine.

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