from the hospital earlier than any other study group. Some effort should be made to define preoperatively those factors that predict vomiting so that only those who need prophylaxis receive an antiemetic. However, if all patients receive droperidol prophylactically, their average discharge time still may be less than those without prophylaxis (309 min *vs.* 338 min), because patients who vomit take longer to meet criteria for discharge home.

We conclude that droperidol used prophylactically in a dose of 75 mcg/kg iv is very effective in decreasing the incidence and severity of vomiting in children undergoing outpatient surgery for correction of strabismus without significantly delaying discharge home. Their postoperative course is more comfortable yet not more complicated.

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Bronchospasm Following Intraocular Injection of Acetylcholine in a Patient Taking Metoprolol

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Systemic effects produced by locally absorbed ophthalmic agents have been observed in patients with both normal and abnormal cardiopulmonary systems.[1] For instance, 10% phenylephrine eye drops, commonly used in the perioperative period for pupillary dilatation and hemostasis, have produced hypertension and coronary artery spasm.[2–5] Two previous reports of hypotension, one of which occurred in association with bradycardia, were attributed to the instillation of acetylcholine chloride (Miochol® intraocular) into the anterior chamber of the eye during routine cataract surgery.[6,7] We observed a case of bronchospasm following acetylcholine injection into the anterior chamber of the eye of a patient taking metoprolol for hypertension, suggesting an interaction between acetylcholine and a beta-adrenergic blocking drug.

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Report of a Case

The patient was a 76-year-old woman with a long history of hypertension, obstructive pulmonary disease, and stable angina, who was

scheduled for extracapsular cataract extraction and placement of an intraocular lens under local anesthesia. She had ECG evidence of an old asymptomatic inferior myocardial infarction, first noted 21 months prior to admission. Cardiac catheterization performed 1 yr before this admission showed three-vessel coronary artery disease and abnormal inferior wall motion with an ejection fraction of 65%; however, the patient refused the recommended coronary artery bypass procedure. She was being treated with hydralazine, metoprolol, and sublingual nitroglycerin with good results. In addition, she had been receiving Persantine® and aspirin, which were discontinued one week before surgery.

Preoperative physical examination was unremarkable except for a cataract present in the right eye and anxiety regarding her planned procedure. Auscultation of the precordium was significant for an S_4 gallop and a grade II/VI systolic ejection murmur at the lower left sternal border. Breath sounds were clear bilaterally. Chest roentgenogram demonstrated left ventricular prominence without evidence of pulmonary congestion. Arterial blood pressure was 170/80 mmHg and heart rate was 62 bpm at rest. Laboratory tests included normal serum electrolytes and urinalysis. The hematocrit was 42.3%.

Meperidine 50 mg was given orally 2½ h before surgery. Prior to anesthesia, 2.5% phenylephrine and 1% cyclopentolate eye drops were instilled into the right eye. Monitors included a precordial stethoscope, ECG using a V_5 lead position, Accutorr® automatic continuous blood pressure monitor, and axillary temperature probe. Anesthesia was accomplished uneventfully using a retrobulbar and a regional nerve block with a 1:1 solution of 2% lidocaine and 0.75% bupivicaine in the accepted dose range. Because retrobulbar placement of a needle can be painful and stressful, the nerve block was performed under sedation with iv methohexital given in small increments of 5–10 mg to a total dose of 40 mg over a 15-min period. Following retrobulbar block, ophthalmic akinesia was tested and the patient was easily able to cooperate, experiencing no discomfort at this time. Blood pressure and heart rate had been stable during administration of methohexital, as well as for performance of the blocks. The drapes were applied over a face tent for continuous oxygen administration at an $F_{I_{O_2}}$ of 0.3 and a flow rate of 8 $l \cdot min^{-1}$. Cataract surgery proceeded uneventfully, and the patient communicated with the anesthesiologist and surgeons intermittently throughout the procedure in an appropriate fashion. Prior to closing the eye at the end of surgery, two doses of Miochol® (acetylcholine chloride) in a 1:100 solution were administered for myosis, the total being 1.75 ml, which is well within the accepted dose range for ophthalmic procedures. At this time, she complained of difficulty in breathing and became quite distressed. Auscultation of the chest abruptly changed from being clear to diffuse bilateral wheezing and occasional rales. Arterial blood pressure, which had been very stable throughout the procedure, rapidly increased from 160/85 mmHg to 200/110 mmHg, with a concurrent rise in heart rate from 72 to 95 bpm. Her ECG, with the exception of tachycardia, was unchanged. She had no chest pain or change in her level of consciousness. Arterial blood gases showed a pH_a 7.30, Pa_{CO_2} 53 mmHg, Pa_{O_2} 77 mmHg, HCO_3 25 $mEq \cdot l^{-1}$, while breathing from a face tent oxygen at 8 $l \cdot min^{-1}$. Total crystalloids given iv during the 90-min procedure were 300 ml.

In the recovery room, she continued to have evidence of bronchospasm and repeat analysis of arterial blood gases indicated worsening of hypoxia and hypercarbia with a pH_a 7.19, Pa_{CO_2} 67 mmHg, Pa_{O_2} 59 mmHg, HCO_3 27 $mEq \cdot l^{-1}$. Initial postoperative chest roentgenogram showed hyperexpansion and a fluffy alveolar pattern consistent with pulmonary edema superimposed on reactive airway disease. She was treated successfully with diuretics, endotracheal intubation, and controlled ventilation. Bronchospasm was treated with aminophylline. The trachea was extubated 4 h after treatment was initiated in the recovery room. Postoperative EKG was unchanged from the one obtained on the previous day. Cardiac enzymes were normal and failed to rise over the course of her hospitalization. Ten hours after the operative procedure, the patient was alert and communicating appropriately. At that time arterial blood gasses were pH_a 7.39, Pa_{CO_2} 42 mmHg, Pa_{O_2} 74 mmHg, HCO_3 24 $mEq \cdot l^{-1}$ on supplemental oxygen by nasal cannula at 3.0 $l \cdot min^{-1}$.

Discussion

Systemic side effects due to absorption of acetylcholine from the eye have been limited to the cardiovascular system in previous case reports. In our patient, however, bronchospasm was her predominant initial problem, with subsequent development of pulmonary edema and cardiac failure.

Prior to surgery she had been taking metoprolol for control of hypertension. Although this drug has more selectivity for B_1 receptors than does propranolol, metoprolol also can exert effects on B_2 receptors in the lung.[8] Chronic therapy with metoprolol may have made our patient more sensitive to both the phenylephrine instilled into the eye initially, as well as the acetylcholine used at the conclusion of the procedure. Mathe *et al.*[9] tested isolated strips of human bronchi for their responses to several drugs. He found that propranolol would block the bronchodilator effects of epinephrine completely. In addition, after pretreatment with beta-adrenergic blocking drugs, epinephrine produced constriction, although at doses 10 times the minimum dilator dose in the absence of beta adrenergic blockade.[9] Thus, alpha-adrenergic stimulation from absorption of intraocular phenylephrine, especially in the presence of beta blockade, would favor the breakdown of 3′5′ cyclic AMP in the bronchi of the lung, tipping the equilibrium toward bronchoconstriction in this patient.

Acetylcholine also has both cardiovascular and pulmonary effects mediated indirectly via stimulation of parasympathetic nerve fibers and directly by interaction at the neuromuscular junction. Cardiovascular actions include vasodilatation, depression of cardiac contractility, especially of atrial muscle, and a negative chronotropic effect. In the pulmonary system, acetylcholine produces pulmonary vasodilation, increases tracheobronchial secretion, stimulates bronchospasm, and alters the response of chemoreceptors in the aortic and carotid bodies.[8,10] Our patient may have been more susceptible to the adverse pulmonary effects of acetylcholine due to primary disease, as well as chronic therapy with metoprolol. She never showed any of the typical cardiovascular responses to systemic acetylcholine, such as bradycardia or hypotension. Perhaps these direct effects of the drug were superseded by the hypercarbic stimulus produced by bronchospasm, which occurred abruptly only minutes after a second dose of Miochol® was injected into the anterior chamber of the eye. In the presence of bronchospasm, hypoxia ensued, which would have worsened car-

diac performance in a heart already compromised by poor circulation. The preexisting beta-adrenergic blockade would have compounded the problems, since these drugs decrease cardiac muscle responsiveness and ability to compensate in the face of a failing heart. Also, pulmonary vasodilation and negative inotropic effects of acetylcholine would have decreased further the heart's ability to respond to changes in pulmonary compliance, leading to the development of pulmonary edema.

In summary, bronchospasm has not been reported previously as the primary event following systemic absorption of acetylcholine injected in accepted doses for myosis during routine cataract procedures. Because the drug is used widely for this purpose, it is important for both the anesthesiologist and the ophthalmologist involved in its administration to be aware of potential problems, especially in patients with a history of bronchospastic disease or severe coronary artery disease.

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Paradoxical Diuresis in Some Neurosurgical Patients Under Balanced Anesthesia

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Reduced urinary output and electrolyte excretion, especially sodium, occurs in patients subjected to anesthesia, possibly due to an approximate anesthesia induced 40% decrease in renal blood flow and 20–40% decrease in glomerular filtration rate.[1–3] Positive-pressure ventilation also decreases renal blood flow and glomerular filtration rate.[4,5]

Inappropriate secretion of antidiuretic hormone (ADH) occurs in certain cases of mediastinal tumors, expanding intracranial lesions, cerebral trauma, and coma. As a result hyponatremia, supranormal extracellular fluid volume, increase in glomerular filtration rate, and low blood urea nitrogen concentration occur.

We observed that some patients undergoing craniotomies for brain tumors have profound diuresis during induction of anesthesia. We, therefore, designed a prospective study to determine whether there was diuresis and, if so, what was the mechanism and does it correlate with the anatomic location or pathology of the intracranial lesion.

Method

Twenty-one consecutive patients undergoing neurosurgical procedures consented to participate in the study approved by the institutional review board. Five patients undergoing anterior or posterior cervical fusions constituted the control group. Sixteen patients underwent craniotomy for brain tumors. Patients undergoing craniotomies were restricted to fluid intake of 1,500–1,800 $ml \cdot day^{-1}$ and were receiving dexamethasone. They all had normal renal function as judged by their blood urea nitrogen (BUN) and creatine levels. All patients were premedicated with diazepam, 10 mg po, and atropine, 0.4 mg, or glycopyrrolate, 0.2 mg im. An indwelling urinary catheter was inserted at the time of premedication. After arriving in the operating room, an iv catheter was inserted into a peripheral vein for fluid administration and a radial artery catheter inserted for blood pressure monitoring and blood sampling. Prior to induction of anesthesia, the bladder was emptied completely and urine

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