Discussion

The more ominous, albeit rate, toxic reactions associated with general anesthesia have included hepatic necrosis and renal failure. Evidence that these reactions are associated with biotransformation of the anesthetic and production of toxic metabolites has accumulated [5,6].

Aliflurane is chemically stable and apparently more resistant to biotransformation than the inhalational anesthetics in current clinical usage. Urinary fluoride ion represented 0.2 per cent of the absorbed dose. Approximately twice this amount can be assumed to have been released, since it is known that approximately 50 per cent of ingested or injected soluble fluoride salts is deposited in bone[7,8] and the rest is excreted promptly in urine, with a half-life of four to five hours. Organic fluorine metabolites accounted for 0.6 per cent of total uptake. Hence, a total of approximately 1 per cent of absorbed aliflurane was converted to measureable metabolites. This compares favorably with methoxyflurane[4] (41 per cent urinary metabolites), fluroxene[9] (10.0 per cent urinary metabolites), halothane[10,11] (12–25 per cent urinary metabolites) and enflurane[3] (2.4 per cent urinary metabolites).

In summary, the biotransformation of aliflurane was studied in ten healthy young male volunteers. It appears to be highly resistant to biotransformation in man. The maximum excretion of fluoride and organic fluorine occurred during the first 24 hours following exposure.

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Intraoperative Obstruction of Endobronchial Tubes

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Since the introduction of the Carlens catheter, in 1949,[1] the benefits of endobronchial anesthesia have been well documented. As with most technical procedures, complications, such as malpositioning of the catheter and respiratory obstruction, have been associated with one-lung anesthesia.[2,3] The latter complication may result in dangerous hypoxemia if allowed to persist. Correction of catheter malpositioning, particularly after a surgical procedure has begun, can present the anesthesiologist with a very difficult technical problem.

Two cases of endobronchial catheter obstruction during thoractomy are reported. In the first case, obstruction occurred during closure of bronchoesophageal fistulas, and in the second, during lobectomy for a tuberculous pulmonary abscess.

Report of Two Cases

Patient 1. A 28-year-old woman was admitted to the hospital because of shortness of breath, persistent cough, and intermittent

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TABLE 1. Arterial Blood-gas Values, Patient 1

	$F_{I_{O_2}}$	P_{O_2} (torr)	P_{CO_2} (torr)	*p*H	HbO_2 (Per Cent)
Preoperatively	0.21	69	28	7.49	95
After left bronchial intubation and anesthetic induction	1	420	36	7.41	100
Lateral position, chest closed	0.6	435	29	7.47	100
Pleura opened, upper lung collapsed	0.6	183	31	7.38	99.1
At hourly intervals	0.6	195	28	7.30	99.1
	0.6	250	59	7.27	100
	0.6	175	40	7.34	98.9
After changing tube, $F_{I_{O_2}}$ increased	1	145	33	7.37	98.7
Chest closed	0.5	116	32	7.42	98.7
In intensive care unit, T piece in place	0.4	156	32	7.40	98.9
After extubation, face mask in place	0.4	120	34	7.45	98.4

hemoptysis of increasing severity. She had a six-year history of bronchiectasis, for which a right lower lobectomy had been done four years prior to admission. There was also a past history of premature ventricular contractions followed by cardiac arrest after induction of anesthesia during another attempt at thoracic surgery. The patient had been resuscitated at that time.

On admission, she was severely dyspneic and febrile. Spirometric studies were consistent with restrictive and obstructive pulmonary disease. Arterial blood-gas analyses showed slight hypoxemia and slightly alkaline *p*H with minimal changes after excercise. Bronchoscopy, bronchogram and esophagogram revealed abundant purulent secretion coming from the right upper lobe. A foreign body (which appeared to be a Tevdeck suture) was found in the posterior segment of the right upper lobe. Obstruction of the bronchus intermedius by granulomatous tissue, multiple fistulas between esophagus and bronchus intermedius, and bleeding from the right upper lobe were also found. After several unsuccessful attempts to remove the foreign body bronchoscopically, the patient was scheduled for right pneumonectomy (right upper lobe, right middle lobe) and closure of bronchoesophageal fistulas.

In the operating room, a single-lumen 7.5-mm endobronchial catheter was inserted into the left bronchus using a flexible fiberoptic bronchoscope after application of 4 per cent lidocaine for topical anesthesia. The location of the catheter was confirmed by auscultation and chest x-rays in both supine and lateral chest positions. Induction and maintenance of anesthesia were accomplished uneventfully using thiopental, N_2O, O_2, and enflurane.

Approximately five hours after the start of the surgical procedure, rapidly progressive resistance to ventilation was noticed. At the same time, a gas leak developed around the cuffed endobronchial catheter. Oxygen concentration was increased to 100 per cent and the cuff was deflated. Repeated attempts to reposition the endobronchial catheter met with unexpected resistance. A suction catheter could not be passed down the lumen of the tube, confirming the presence of an obstruction. At this point the tube was removed with a sudden snap. A linear tear was found in the endobronchial cuff where it had been inadvertently stapled to the bronchus during the operation. The patient's trachea was reintubated with a conventional endotracheal catheter. The entire episode of obstruction and reintubation of the trachea lasted approximately 5 minutes. Following reintubation of the trachea, the left lung could be ventilated easily. Intraoperative blood-gas values are shown in table 1. The trachea was extubated on the first postoperative day.

The postoperative course was long and stormy, complicated by right-chest empyema necessitating a repeat thoracotomy a week later. Purulent drainage from the right-chest tube and fever persisted, and four days after the second thoracotomy the patient experienced convulsions followed by right hemiparesis. The neurologic complications were felt to be secondary to sepsis.

Patient 2. A 39-year-old man was admitted to the hospital because of cough, fever, weight loss and pedal edema. He was a heroin addict, a chronic alcoholic, and a heavy smoker, with a history of diabetes mellitus, pulmonary tuberculosis, chronic renal failure, and cirrhosis of the liver. He appeared to be lethargic. Examination, including chest x-rays and bronchoscopy, revealed a large cavity in the right upper lobe with extensive infiltrate in the middle lobe. Acid-fast bacilli and gram-positive diplococci were found on sputum culture. Spirometric studies were compatible with severe restrictive disease; arterial blood–gas analyses showed hypoxemia (P_{O_2} 44 torr, P_{CO_2} 31 torr, *p*H 7.51). Only minimal general improvement was achieved with medical treatment, and right upper lobectomy for tuberculous abscess was scheduled.

In the operating room, after preoxygenation, a rapid-sequence

TABLE 2. Arterial Blood-gas Values, Patient 2

	$F_{I_{O_2}}$	P_{O_2} (torr)	P_{CO_2} (torr)	*p*H	HbO_2 (Per Cent)
Preoperatively	0.21	48	34	7.40	83
After induction of anesthesia and intubation with Carlens catheter	1	105	50	7.19	95.8
Lateral position, chest closed	1	155	40	7.30	95
Pleura opened	1	60	43	7.27	85
Upper lung collapsed	1	52	35	7.37	88
After removing Carlens catheter and endotracheal intubation	1	59	35	7.34	88
Chest closed	1	120	27	7.46	98.2
After tracheotomy	1	58	43	7.17	81
15 minutes after tracheotomy	1	90	30	7.44	96.5

induction was done with thiopental, succinylcholine, and 2 per cent lidocaine spray to the vocal cords. The patient's trachea was intubated with a double-lumen Carlens catheter. The catheter was positioned without difficulty and provided adequate selective ventilation to both lungs. The location of the tube was reconfirmed with chest x-rays both in the supine and in the lateral chest position. Four hours after the start of the operation, during surgical manipulation, a large volume of pus was suddenly expelled from the right main bronchus. The copious discharge of pus completely obstructed the right lumen, and extended to the anesthesia circuit and to the left lumen of the Carlens tube despite immediate clamping of the right bronchus and vigorous suctioning. The Carlens tube was removed and replaced with a conventional endotracheal catheter. A tracheotomy was performed at the end of the operation. Intraoperative arterial blood-gas values, indicating hypoxemia and episodes of acidosis, are shown in table 2. Postoperatively the patient had a bronchopleural fistula, which was successfully repaired.

Discussion

Various cases of ventilatory difficulty due to malpositioning of both single- and double-lumen endobronchial catheters have been reported. The catheters are inserted either too far or not far enough, so that: 1) inflation of the bronchial cuff results in obstruction of ventilation with gas trapping[3]; 2) selective ventilation and, when desired, collapse of the diseased lung, cannot be achieved[2,3]; 3) leaks occur around the inflated bronchial cuff due to surgical manipulation or distortion of the shape of the bronchus by tumor.[2,4]

In both our patients endobronchial anesthesia was satisfactory for a number of hours. In the first patient, whose trachea was intubated with a single-lumen endobronchial catheter, inadvertent stapling of the inflated cuff to the bronchial wall caused a gas leak and was also the cause of resistance encountered with removal of the tube. Stapling created increased resistance to gas flow and difficulty in ventilating the patient. We were later able to simulate and recreate this mechanism of obstruction *in vitro*.

Smaller-caliber endobronchial tubes cause greater resistances to gas flow than do conventional endotracheal tubes. Gas flow increases in proportion to the fourth power of the radius, as defined by the Hagen-Poiseuille equation[5]; therefore, increased airway pressure is often necessary to ventilate patients adequately when small-lumen catheters are used. For the same reason, secretions, pus or slight compression deformities of these catheters may cause increased resistance to ventilation and turbulent flow.[5] Suctioning of double-lumen catheters also becomes more difficult and predisposes further to hypoxia. In our second patient, the sudden flow of pus from the right bronchus completely obstructed the right bronchial lumen and partially obstructed the left bronchial lumen.

In both patients, one-lung anesthesia was abandoned and the endobronchial catheters replaced with endotracheal catheters while the patients were in the lateral position. Reintubation of the trachea under these circumstances is an emergency procedure, which can be technically difficult. The potential for severe hypoxia and spread of infection during this maneuver is also increased. Methylene blue, used to confirm the closure of the fistula in the first patient, had stained the mucosa of the pharynx and larynx, further complicating tracheal reintubation.

In order to maintain adequate pulmonary function in patients with pulmonary disease during thoracotomy, the increased potential for hypoxemia inherent in one-lung anesthesia must be taken into account. For this reason, one-lung anesthesia should not be attempted without the ability to measure arterial blood gases. Hypoxemia has been ascribed to: 1) veno-arterial shunt[2,6–10]; 2) alveolar hypoventilation[2,10]; 3) ventilation of one lung with a large tidal volume, causing a shift of blood to the nonventilated lung[2]; 4) technical catheter problems that rapidly aggravate pre-existing pulmonary dysfunction.

When using endobronchial anesthetic techniques, the anesthesiologist cannot be assured of maintaining a well-positioned catheter *in situ*. Small adjustments and repositioning of the tube, checked by auscultation and x-rays, are often necessary after the patient has been placed in the lateral chest position in order to secure a stable, unobstructed, leak-proof airway. Even then, obstruction, leaks and other airway hazards may develop during the course of the operation,[2,4] as in the two cases presented in this paper. In spite of unexpected problems, both our patients survived with no permanent sequelae attributable to the use of the endobronchial catheters. Difficulties associated with endobronchial anesthesia must be anticipated, as early recognition and correction of airway obstruction are crucial if hypoxemia is to be avoided.

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Anesthetic Management of the Wolff-Parkinson-White Syndrome

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The Wolff-Parkinson-White (WPW) syndrome and its variants are called the pre-excitation syndrome. The anesthetic management of patients with this syndrome is aimed at avoiding tachyarrhythmias. Katz and Kadis[1] advocate minimal circulatory disturbance using a nitrous oxide, oxygen, and narcotic technique. Similarly, on the basis of one case discovered intraoperatively, van der Starre[2] recommended neuroleptanalgesia and avoiding drugs with negative inotropic effects on the heart. Conversely, Kumazawa[3] advised using deep inhalational anesthesia. We have recently anesthetized 13 patients with the pre-excitation syndrome, and our experience supports the latter opinion.

Resumé of Thirteen Cases

Nine of the 15 episodes of anesthesia in these 13 patients were for surgical treatment of arrhythmias (His- or Kent-bundle divisions) refractory to medical therapy. All patients except Patient 4 were known to have the syndrome, and had had episodes of tachyarrhythmias (table 1).

Patients 1 and 2 received morphine (1 mg/kg) for induction of anesthesia with the addition of halothane (0.5–1.0 per cent) and N_2O after endotracheal intubation. They had no arrhythmias. Patient 3 received anesthesia with morphine (1 mg/kg), diazepam, 10 mg, N_2O, and pancuronium. Episodes of supraventricular tachycardia appeared soon after incision of the skin, necessitating cardioversion more than ten times. Patient 4 was managed by a similar anesthetic technique, and cardioversion was done for atrial fibrillation, with a rapid ventricular rate. A few days later, after the heart rate was controlled with propranolol, Patient 4 underwent uneventful repair of an abdominal fistula with morphine, N_2O, and *d*-tubocurarine.

The remaining nine patients received inhalational anesthesia with halothane or enflurane. Blood pressure and heart rate were maintained at or below preoperative values. For all but Patient 10, *d*-tubocurarine was used. Only Patient 5 had an intraoperative arrhythmia, during cardiac manipulation for vena caval cannulation.

Discussion

Excitation of the heart is diagrammed in figure 1*A*. The impulse spreads from the sinus node through the atrium, undergoes physiologic delay at the atrioventricular (A-V) node, then passes through the His bundle to the Purkinje network. Characteristic of the pre-excitation syndrome is premature activation of a portion of ventricular muscle. The common denominator in all forms is the presence of an anomalous conduction pathway that bypasses the A-V node. The classic form of pre-excitation in Wolff-Parkinson-White syndrome is depicted in figure 1*B*. The sinus impulse is conducted simultaneously down an anomalous pathway (bundle of Kent) and the normal pathway. The lack of physiologic delay in the anomalous pathway accounts for the short PR interval. Ventricular excitation is a composite of the two impulses; thus, a fusion beat containing a delta wave accounts for the QRS prolongation. Variations of the pre-excitation syndrome include the presence of some, but not all, of the above electrophysiologic features. The Lown-Ganong-Levine (LGL) syndrome

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