Miller Anaesthesia for Thoracic Surgery

Anesthesia for Thoracic Surgery

INTRODUCTION

The techniques for administering anesthesia for thoracic surgery have undergone a dramatic, progressively refined, and scientifically based evolution. Initially, the general problem of gas exchange with an open thorax was defined and its physiology understood; the problem was solved with the use of controlled positive-pressure ventilation. The introduction of clinically useful muscle relaxants facilitated the use of controlled ventilation. The need for isolating one lung from another became apparent during surgery for lung abscess, bronchopleural fistula, and hemoptysis; a plethora of methods for providing separation of the lungs was developed. Considerable clinical experience subsequently defined and refined the indications and techniques for one-lung anesthesia. Concomitantly, the changes in the distribution of blood flow and ventilation, in the awake state and in the anesthetized and mechanically ventilated state in both the supine and the lateral decubitus positions, became understood. Methods have been developed to manage one-lung ventilation so that arterial oxygenation levels are close to those achieved during two-lung ventilation. The postoperative period can now be almost pain-free and without great risk with the use of epidural narcotics, patient-controlled analgesia, and/or cryoanalgesia (Ch. 69).

This chapter is divided into two sections. The first part moves temporally through the perioperative period and provides the essentials for the management of a patient under-going thoracic surgery. Preoperative considerations include pulmonary evaluation and optimal pulmonary preparation. Intraoperative period considerations are monitoring requirements, choice of anesthesia, respiratory physiology of the lateral decubitus position and one-lung ventilation/anesthesia, and most importantly, the indications and techniques for providing one-lung ventilation/anesthesia. Finally, the postoperative problems of immediate lifethreatening complications, management of mechanical ventilation, therapeutic respiratory care maneuvers, and control of pain are discussed. The second part of this chapter deals with specific anesthetic techniques and problems encountered in a variety of special diagnostic, elective, and emergency thoracic procedures.

THE PREOPERATIVE PERIOD

Preoperative Evaluation

The vast majority of noncardiac, noncardiopulmonary bypass thoracic surgery procedures consist of resectional or repair procedures for cancer and other masses of the lung and bronchi. Consequently, this section emphasizes these lesions and gives special attention to the respiratory and cardiovascular systems. 1, 2, 3, 4, 5 Also see Chapter 23.

Respiratory System

History

The history (Chs. 23 and 25)often raises suspicion of the diagnosis of lung cancer. The average patient with cancer of the lung (carcinoma constitutes approximately 90 percent, adenomas 8 to 10 percent, and benign masses 1 percent of lung tumors) is in the sixth or seventh decade of life, has a history of heavy cigarette smoking and recent weight loss, and resides in an urban area. However, a small percentage of lung carcinomas occur in nonsmokers (<10%) (many of these can be traced to a history of passive or involuntary smoking 6, 7, 8), and lung carcinoma has a higher incidence of occurrence in workers in some chemical industries (such as asbestos, arsenic, chromates, and nickel) than in the general population. Uranium miners have a greatly increased risk for the development of lung carcinoma, especially if they smoke. All age groups are affected, but the disease is rare in persons less than 30 years of age. Five percent of patients are asymptomatic, and in this group the tumor is discovered only by routine roentgenographic examination of the chest. The vast majority of patients, however, have one or more symptoms related to the presence of the tumor. The symptoms may be designated as bronchopulmonary, extrapulmonary intrathoracic, extrathoracic metastatic, extrathoracic nonmetastatic, and nonspecific. On the average, symptoms have been present for 6 to 7 months before the time the patient seeks medical advice; because the first chest roentgenographic

findings frequently antedate the first symptoms by several months, lung carcinoma will be at least 1 year old (and perhaps 2–5 years old) by the time of clinical presentation.

Bronchopulmonary Symptoms.

Bronchopulmonary symptoms arising from involvement of the lung (cough, sputum, chest pain, dyspnea, wheeze) are due to bronchial irritation, ulceration, obstruction, infection distal to the obstruction, or a combination of these processes. In a large series of patients with carcinoma of the lung, 75 percent had cough as one of the major symptoms, and this symptom was severe in 40 percent of patients. However, cough is possibly the most common manifestation of respiratory disease in general. It is so common among cigarette smokers that many of them regard a morning cough as “normal.” The most common stimulus to cough is the formation of sputum in the respiratory tract (see below), and the cough process is an essential element in keeping the tract clear.

The normal adult produces about 100 mL of mucus from the respiratory tract in a day. When excess mucus is formed, it may accumulate, stimulate the mucous membrane, and be coughed up as sputum. Sputum in patients with bronchogenic carcinoma may be formed in response to physical, chemical, or infective insult to the mucous membrane of the airways. Mucoid sputum is clear or white. Black sputum is due to the detritus of cigarette or atmospheric smoke. Purulent sputum can vary from small streaks to gross hemoptysis (see later) and always warrants investigation for carcinoma. Hemoptysis generally manifests itself as episodic blood streaking of the trophils. Failure to clear a recent change in the quality and quantity of sputum within a few days of initiating antibiotic therapy should raise suspicion of a neoplasm. Blood-stained sputum can vary from small streaks to gross hemoptysis (see below) and always warrants investigation for carcinoma. Hemoptysis, generally manifest as episodic blood streaking of the sputum, is present in 57 percent of patients with bronchogenic carcinoma and is the first symptom in many.

Chest pain is present in 40 percent of patients presenting with a new carcinoma. It is usually a mild, constant, dull ache on the side with the tumor. Another important form of chest pain with lung carcinoma is pleuritic pain. It is due to direct tumor extension to the pleura, is characteristically worse on breathing and coughing, and can usually be accurately localized by the patient. Mediastinal tumors can cause pain that is usually aching and retrosternal but poorly localized.

Dyspnea is a common complaint in patients with both chronic lung disease and lung carcinoma (30%). In chronic diseases, it is common to find that the patient begins to complain of dyspnea only after the respiratory reserve is quite severely impaired, whereas in patients with lung carcinoma dyspnea occurs more abruptly and with less objective functional impairment.

Wheezing is described by 10 percent of patients and is frequently localized to one side. It is due to airway obstruction, and, if the obstruction is located in the trachea, severe dyspnea and stridor may develop.

Extrapulmonary Intrathoracic Symptoms.

Other symptoms of chest disease occur as a result of growth of the tumor beyond the confines of the lung. These symptoms are due to involvement of the pleura (effusion), chest wall (pain), esophagus (dysphasia), superior vena cava (superior vena cava syndrome), pericardium (pericarditis), brachial plexus (arm pain, Horner syndrome), and right or left recurrent laryngeal nerve (hoarseness). Approximately 15 percent of patients with carcinoma of the lung have these kinds of extrapulmonary intrathoracic symptoms.

Extrathoracic Metastatic Symptoms.

Symptoms resulting from metastatic spread of the tumor outside the thorax account for a small percentage of the presenting or major complaints of patients with carcinoma of the lung. These extrathoracic metastatic symptoms can be referable, in order of general decreasing frequency, to brain, skeleton, liver, adrenals, gastrointestinal tract, kidneys, and pancreas. The history with regard to these other organs is extremely important because any positive history referable to these organs requires specific organ workup for metastatic disease (for staging, see the section *Pulmonary Function Testing*), and the finding of such metastases precludes surgery.

Extrathoracic Nonmetastatic Symptoms.

Extrathoracic nonmetastatic symptoms are usually due to a paraneoplastic syndrome caused by secretion of endocrine or endocrine-like substances by the tumor. The endocrine-like manifestations include Cushing syndrome, excessive antidiuretic hormone secretion, carcinoid syndrome, hypercalcemia, ectopic gonadotropin secretion, and hypoglycemia. The neuromuscular manifestations consist of carcinomatous myopathies (Eaton-Lambert Syndrome) and various myopathies related to brain dysfunction. Other manifestations can be skeletal (clubbing, pulmonary hypertrophic osteoarthropathy), dermatologic (scleroderma, acanthosis nigricans), vascular (thrombophlebitis), and hematologic.

Nonspecific Symptoms.

Weight loss, anemia, weakness, anorexia, lethargy, and malaise occur in a large number of patients. Vague febrile respiratory (cold-like) syndromes may be present in 22 percent of these patients. In 10 to 15 percent of patients, these symptoms are responsible for the initial visit to the physician.

Physical Examination

The basic tools of inspection, palpation, auscultation, and percussion should allow the physician to assess, in a gross way, the overall severity of chronic lung disease, regardless of whether major consolidation, atelectasis, or pleural effusion is present and whether there is any obvious extrathoracic complication of thoracic carcinoma. Because it is mandatory subsequently to use much more sensitive radiologic means of determining resectability, further discussion of physical examination methods for making these determinations is not included here. The detection of bronchospasm, however, is particularly important in that it requires further documentation, is usually reversible with treatment, and has important anesthetic management implications, which are discussed in the section *Preoperative Preparation.*

Common Laboratory Tests

Some of the routine laboratory tests (Ch. 23) that are performed on all patients are especially relevant to the preoperative evaluation of the patient with a lung or bronchial mass. The complete blood count may indicate polycythemia, which may reflect decreased arterial hemoglobin saturation; leukocytosis may be indicative of active pulmonary infection. Gram stain of the sputum provides a qualitative index of infection; cultures and sensitivity studies direct specific antibiotic therapy. Sputum cytology is useful in diagnosing neoplasms. Liver and bone enzymes, blood urea nitrogen and creatinine, and urinalysis can help establish the diagnosis of metastatic lung cancer. However, the chest roentgenogram is, by far, the most useful common laboratory test.

The Chest Radiograph

It has been estimated that when a tumor of the lung is first detected on a chest radiograph, it has completed three fourths of its natural history, 9 and this first radiographic abnormality frequently antedates the first symptoms or signs of the disease by 7 or more months. 10 By the time bronchial carcinoma becomes symptomatic, the chest radiograph is abnormal in 98 percent of all patients, and the abnormality is most suggestive of tumor in over four fifths of these.

The radiographic findings due to carcinoma of the lung (Fig. 48–1) may be the result of the presence of the tumor within the lung 11 (Table 48–1) (70% are centrally located), of changes in the pulmonary parenchyma distal to a bronchus obstructed by the tumor (atelectasis, infection, and cavitation), and of spread of the tumor to extrapulmonary intrathoracic sites (hilar and mediastinal lymph nodes, pleura, chest wall, and diaphragm).

TABLE 48–1. Radiologic Criteria for Differentiating Malignant From Benign Pulmonary Opacities
FIGURE 48–1 In patients with lung carcinoma the chest radiograph findings result from the presence of the tumor within the lung (parenchymal mass), changes in the pulmonary parenchyma distal to a bronchus obstructed by the tumor (atelectasis and infection), and spread of the tumor to extrapulmonary intrathoracic sites (hilar and mediastinal masses and other direct extension pathology). (From Benumof604)

The usual radiographic manifestations of lung carcinoma frequently include the hilar and extrapulmonary intrathoracic manifestations in addition to the pulmonary parenchymal manifestations. In a review of the

radiographs of the chest of 600 patients with carcinoma of the lung, 12 the average lung cancer mass at radiologic presentation was 3 to 4 cm in diameter. A large parenchymal mass was present in 22 percent of patients and a smaller mass in 20 percent; multiple masses were present in only 1 percent. Obstructive pneumonitis, collapse, or consolidation was present in 41 percent. A hilar abnormality, either alone or associated with other abnormalities, was present in 41 percent of the patients. The various extrapulmonary intrathoracic manifestations, of which mediastinal widening, pleural effusion, and raised hemidiaphragm were the more common, were present in 11 percent.

Several radiographic findings can have specific anesthetic implications. These lesions consist of tracheal deviation or obstruction (difficulty with intubation or ventilation); mediastinal mass (difficulty with ventilation, superior vena cava syndrome, compression of pulmonary artery); pleural effusions (decreased vital capacity and functional residual capacity [FRC]); cardiac enlargement (susceptibility to anesthetic agents that depress the heart); bullous cyst (hazard of rupture, compression of adjacent lung); air-fluid levels (abscess with hazard of spread of infection); and parenchymal reticulation, consolidation, atelectasis, or edema (increased ventilation/perfusion [] inequality and transpulmonary shunt). It is important to note that as many as 10 percent of patients with chronic diffuse infiltrative lung disease may have normal chest radiographs. 13

Pulmonary Function Testing

Preoperative pulmonary evaluation (Ch. 24) of patients with cancer of the lung should resolve questions concerning both resectability and operability. The question of resectability requires local tumor, regional node, and distant metastasis (T, N, and M, respectively) staging of the disease and is based on clinical examination, radiographic (including computed tomographic [CT]) studies (T staging), bronchoscopic and mediastinoscopic examination (N staging), and individual organ evaluation and scanning (M staging). Operability addresses the question of how much pulmonary tissue can be safely removed without rendering the patient a pulmonary cripple (the remaining lung may be diseased by a long history of smoking), and this question is best answered by pulmonary function tests. When making an evaluation of operability, the physician must remember that untreated carcinoma of the lung is associated with a mean survival of less than 1 year and that the only currently effective method for curing carcinoma of the lung is surgical excision.

There is a general consensus that in the performance of a pneumonectomy, pulmonary function testing should proceed in three phases 14, 15 (Table 48–2). The first phase evaluates total lung function and consists of analysis of room air arterial blood gases as well as simple spirometry and determination of lung volumes. More recent reports indicate carbon monoxide diffusing capacity and exercise testing may be indicated as well (Table 48–3). Increased risk is present when hypercapnia ($PaCO_2 > 45$ mm Hg) is present on a room air blood gas sample, the forced expired volume in 1 second (FEV_1), and/or the maximum breathing capacity is less than 50 percent of predicted, and/or the residual volume is greater than 50 percent of total lung capacity. If any of these whole-lung pulmonary function values is worse than the stated limits, testing should proceed to the second phase, which evaluates the function of each lung separately; this phase consists of measurement of the ventilation 16, 17, 18 and, more recently, the perfusion 19, 20 of each individual lung (as a fraction of the total) by radioisotopic (133Xe and99Tc) scanning. Combining right-left fractional lung function tests with conventional spirometry should yield a predicted postoperative FEV_1 greater than 0.85 L. 16, 17, 18 For example, if the perfusion of the lung to be removed is 40 percent of the total perfusion and the preoperative FEV_1 is 1.4 L, the predicted postoperative FEV_1 is 0.84 L. That is, predicted postoperative FEV_1 equals preoperative FEV_1 multiplied by contralateral perfusion (expressed as a percentage).

TABLE 48–2. Preoperative Pulmonary Function Tests (PFTs) and Operative Risk of Pneumonectomy

TABLE 48–3. Minimal Pulmonary Function Test Criteria for Variously Sized Pulmonary Resections

If the second-level criterion of acceptable predicted postoperative FEV_1 cannot be met and surgery is still contemplated or desired, the postoperative condition of the patient can be simulated (the third phase of testing) by functionally resecting the vascular bed of the lung to be taken out by temporary balloon occlusion of the major pulmonary artery on that side, with and without exercise. 19, 21, 22, 23, 24 Under these conditions, the distensibility (compliance) of the remaining pulmonary vascular bed is tested, and an increase in mean pulmonary artery pressure to greater than 40 mm Hg, and/or an increase in $PaCO_2$ above

60 mm Hg, and/or a decrease in PaO2 less than 45 mm Hg indicates an inability to tolerate the removal of this amount of lung.

Ventilatory function postpneumonectomy (or after any resection) can also be simulated preoperatively by passing, with the aid of a fiberoptic bronchoscope, a balloon occlusion catheter, which can occlude either lung (or any lobe), and then measuring spirometry of the remaining lung tissue (after careful withdrawal of the bronchoscope). Supplemental oxygen must be administered during bronchial blockade because the blocked segment would still be perfused, and all this perfusion would be right-to-left shunt flow, which would create a risk of hypoxemia.

This pulmonary function testing cascade is logical because it starts out with simple, relatively inexpensive, noninvasive tests and only increases the degree of difficulty, expense, and invasiveness as indicated.

Although less restrictive pulmonary function test criteria for operability for pulmonary resections less radical than pneumonectomy have been published 25, 26, 27, 28, 29, 30, 31, 32, 33, 34, 35, 36, 37 (see Table 48–3), there are several reasons why, at least in some patients, it may be prudent to think of a lobectomy (and lesser procedures) as a functional pneumonectomy. 38 First, during the immediate postoperative period, the function of the lung tissue remaining on the operative side may be significantly impaired by atelectasis and perhaps infection; consequently, these patients may experience significant transient postoperative functional impairment. 39 Patients who are most likely to have a stormy postoperative course with minor resections are those who have had intraoperative exposure problems that required severe and prolonged lung manipulation. Intraoperative exposure problems are most likely to occur when the lung being operated on is large and moving (large tidal volume with positive-pressure ventilation). Second, at the time of thoracotomy more accurate staging of the disease is possible, and it may become apparent that it is necessary to perform a pneumonectomy. 15 Third, the function of the lung on the nonoperated side may be impaired preoperatively 39 and may acutely deteriorate intraoperatively as a result of spillage of blood and/or pus from the operated to nonoperated lung or by inability of the nonoperated lung to tolerate a prolonged period of dependency and compression in the lateral decubitus position. Finally, postlobectomy function studies have shown that although the ventilation and perfusion of the lung remaining on the operated side become significantly greater during the long-term interval (3–51 months), the volume of the remaining lung gradually increases and becomes significantly greater than the increase in either ventilation or perfusion to this lung. 40 The compensatory hyperinflation represents dilation of the preexisting respiratory units without disruption or fragmentation of the elastic tissue as seen in pathologic emphysema; however, the pulmonary hyperinflation decreases the compliance and therefore the ventilation per unit volume of the ipsilateral remaining pulmonary tissue. In addition, the hyperinflated lung stretches and thins out the capillaries in the alveolar walls, decreasing the perfusion per unit volume of the ipsilateral remaining pulmonary tissue.

Cardiovascular System

Pulmonary Vascular and Right Ventricular Function Testing

The vast majority of patients with pulmonary tumors have had a long history of smoking; consequently, they have varying degrees of chronic obstructive pulmonary disease (COPD). The cardiovascular response to the pathologic alveolar and airway changes in COPD consists of the development of pulmonary hypertension and increased pulmonary vascular resistance (PVR), followed by right ventricular (RV) hypertrophy and dilation.

Increased PVR has important implications for patients undergoing pulmonary resection. Whereas a normal pulmonary vasculature is distensible and capable of accommodating large increases in pulmonary blood flow (to approximately 2 to 2.5 times normal, as would occur through the remaining lung after a pneumonectomy) with only minor increases in pulmonary artery pressure (Fig. 48–2), the relatively rigid and restricted pulmonary vascular bed of chronic lung disease patients cannot accommodate even small increases in pulmonary blood flow without concomitant increases in pulmonary vascular pressure. 41 The inability to tolerate increases in blood flow occurs over the entire range of physiologic cardiac output and may be an important contributing factor to the development of postpneumonectomy pulmonary edema when it occurs. 42

FIGURE 48–2 Mean pulmonary artery pressure (y axis) does not increase until cardiac output (x axis) has been increased 2- to 2.5-fold when the pulmonary vascular bed is normal, whereas mean pulmonary artery

pressure increases linearly with increasing cardiac output is increased when the pulmonary vascular bed is restricted. (From Robin and Gaudio41)

The preoperative pulmonary function testing cascade as outlined in pulmonary function testing phases 1 and 2 in Table 48–2 (which, in reality, is the extent to which the great majority of patients are studied preoperatively) does not allow diagnosis of increased PVR and RV disease. Increased PVR may be noninvasively suspected preoperatively by the presence of the auscultatory and radiographic signs of pulmonary hypertension and by electrocardiographic (ECG) evidence of right atrial and ventricular hypertrophy (Table 48–4). The development of a positive hepatojugular reflex, ascites, and peripheral edema indicates the onset of cor pulmonale. In COPD patients without waking hypoxemia, cor pulmonale can be detected twice as sensitively and frequently by echocardiography (definition criteria are pulmonary hypertension and RV enlargement and/or hypertrophy) as by the preceding clinical methods. 43

TABLE 48–4. Noninvasive Diagnosis of Pulmonary Hypertension, Increased Pulmonary Vascular Resistance, Right Atrial and Ventricular Hypertrophy, and Cor Pulmonale

Measurements of PVR have been made directly by determining mean pulmonary artery and pulmonary artery wedge pressures at various levels of cardiac output produced by varying treadmill exercises. Thus, using the patient's own cardiac output, pulmonary vascular compliance can be determined. PVR measurements made in this way have been good indicators of risk for pneumonectomy. 44, 45 Operative risk was considered to be increased if PVR was greater than 190 dyne/s/cm. 5 However, if the risk, expense, and time to pass a pulmonary artery catheter have been accepted, it is logical to take one further step and measure pulmonary vascular pressures during temporary unilateral pulmonary artery balloon occlusion in states of rest and exercise (see pulmonary function testing, phase 3). This maneuver specifically tests the compliance of just the pulmonary vascular bed that will remain after pneumonectomy. Performing this procedure during exercise is the most realistic preoperative approximation of pulmonary vascular and right ventricular function to be expected in the ambulatory postpneumonectomy patient. 14, 21, 22, 23, 24 Also, echocardiography is increasingly being used to estimate right ventricular alterations and pulmonary hypertension 43 (see earlier).

In addition to the preoperative condition of the pulmonary vasculature, the intraoperative anesthetic and surgical experience can introduce numerous other causes for further acute increases in PVR, including episodes of hypoxia, 46, 47 acidosis, 48 increased airway resistance during spontaneous exhalation 49 (which causes air trapping, increased alveolar pressure, and compression of the small intra-alveolar vessels), positive end-expiratory pressure (PEEP), 50, 51 and sepsis. 52 In addition, resection of a significant amount of the pulmonary vascular bed further increases PVR, especially if the remaining pulmonary vascular bed is diseased to begin with and the cardiac output is increased. 41, 53, 54 Finally, the remaining (nonresected) lung may become further diseased either intraoperatively (surgically traumatized) or postoperatively. 38, 39 Thus, PVR in patients undergoing thoracic surgery may be additively and sequentially increased preoperatively, intraoperatively, and postoperatively.

Left Ventricular Function Testing

The possible independent causes that may contribute to left ventricular (LV) dysfunction in patients with lung disease consist of coronary artery or valvular disease, 55, 56 systemic hypertension, 55, 56 presence of carboxyhemoglobin, 57 systemic hypoxemia and acidosis, 58 marked alterations in intrathoracic pressure, 59, 60 and RV dysfunction. 55, 61, 62, 63, 64 Considering the usual age, the long and heavy smoking history, and the frequently sedentary life-style of patients undergoing thoracic surgery, it is not surprising that coronary artery disease is by far the most likely independent cause of LV dysfunction. Myocardial ischemia leading to infarction may occur throughout the perioperative period, although peaks of incidence occur during operation and on the third day after operation. The first peak is caused by intraoperative changes in hemodynamics and the second peak is caused by episodes of hypoxia, uneven administration of pain medication, and withdrawal or alteration of drug therapy. 65

There are only two proven preoperative clinical predictors of perioperative cardiac morbidity (defined as occurrence of myocardial infarction, unstable angina, congestive heart failure, serious dysrhythmia, or cardiac death during the intraoperative or in-hospital postoperative periods); these predictors are recent (<6 months) myocardial infarction and current congestive heart failure. 66 The classic historical intraoperative predictors of morbidity—emergency surgery, prolonged (>3 hours) operations, and thoracic or upper

abdominal surgery—also appear to be independent predictors of perioperative morbidity, whereas choice of anesthetic is not. The dynamic intraoperative predictors of perioperative cardiac morbidity are intraoperative hypotension and tachycardia. Hypertension remains a controversial predictor.

If a history of angina or the ECG is suggestive, then further preoperative evaluation of coronary artery function is necessary (suggestive evidence includes Q waves [previous infarction], left bundle branch block, ST segment elevation [transmural ischemia], ST segment depression [subendocardial ischemia], T-wave inversions, and positive U-wave [left main coronary artery disease]). The first step should be noninvasive exercise testing. ECG and thallium scans (in that order) appear to be the best such exercise tests at this time. An exercise study provides information about the functional level of the patient. Unfortunately, the degree of exercise stress may be limited by low ventilatory reserve as well as by low cardiac reserve. If the exercise ECG is normal, surgery should proceed; if the exercise ECG indicates ischemia, a thallium exercise test is indicated. 67 If the thallium exercise test is negative, the planned pulmonary resection should proceed; if the thallium exercise scan is positive for ischemia, coronary angiography should be performed. 68 However, if for any reason there is a strong suspicion that the patient is indeed having significant angina, even though exercise testing is negative or equivocal, coronary angiography is indicated. Consideration should always be given to coronary angiography in the patient with proven previous myocardial infarction, especially if the patient currently has angina. Echocardiography is increasingly being used to estimate LV function.

If significant coronary artery disease is present, the patient needs coronary artery bypass grafting before or at the time of pulmonary resection. For lesser degrees of coronary artery disease, pulmonary resection for carcinoma of the lung should be performed after appropriate medical therapy for coronary insufficiency has been initiated. If the patient needs coronary artery bypass grafting and a limited resection can encompass the cancer, both procedures can be performed under the same anesthetic, but the coronary artery bypass grafting should be done before pulmonary resection. 69, 70 After bypass, if the patient is stable, has good myocardial function, and is not bleeding, a pulmonary wedge resection can be done. For patients who require coronary artery bypass grafting and have pulmonary lesions that require segmentectomy, lobectomy, or pneumonectomy, there is a good possibility that the prolonged nature of the pulmonary procedures will increase the operative mortality (and therefore should not be done), although a small number of successful combined procedures have been reported. 70, 71 In one series of 21 patients, the pulmonary mass was discovered on the preoperative chest x-ray film (for cardiac surgery) and therefore before the occurrence of any symptoms and by definition constituted a fortuitously early diagnosis. Not surprisingly, resection at the time of cardiac surgery (17 wedge resections, 4 lobectomies) resulted in a 95 percent 5-year survival. 72

In cases that require large resections in compromised patients, coronary artery bypass grafting should be done first, and pulmonary resection should be delayed until the patient has gained weight and muscle mass (usually 4–6 weeks). The risk of general anesthesia for a noncardiac operation in the patient with previous coronary artery bypass grafting is similar to that in patients without proven coronary artery disease. 73, 74 Although it is not possible to estimate the true effects of delay in pulmonary resection in terms of tumor spread in a possibly immunocompromised patient (especially after general anesthesia), 75 it seems reasonable that in the latter group (those requiring bypass grafting and major pulmonary resection), the operative risk of combined procedures probably exceeds the risk of tumor spread.

Preoperative Preparation

Thoracic surgical patients are at high risk for the development of postoperative pulmonary complications. In most of the medical literature, “postoperative complications” refers to the development of atelectasis and/or pneumonia. 76 The incidence of pneumonia usually parallels that of atelectasis, and the onset of pneumonia lags behind the onset of atelectasis because atelectasis provides the ventilatory and mucociliary stasis conditions necessary for the development and growth of the organisms that cause pneumonia. 77, 78

There are three major reasons why thoracic surgery promotes postoperative pulmonary complications; these reasons originate in the preoperative, intraoperative, and postoperative periods (Fig. 48–3). First, the incidence of postoperative respiratory complications after any surgery is positively correlated with the degree of preoperative respiratory dysfunction, and most thoracic surgical patients come to surgery with some degree of preoperative lung dysfunction. Compared with nonsmokers, smokers have a sixfold increase in the incidence of pulmonary complications after major operative procedures. 79, 80 In patients with chronic lung disease as compared with normal healthy patients, there is a 20-fold increase in the incidence of

postoperative pulmonary complications. 81 Preoperative pulmonary function testing will identify the patients at high risk because of poor preoperative lung function. The impact of this first factor (presence of preoperative respiratory dysfunction) can be significantly reduced by preoperative prophylactic respiratory preparation measures (see later). 82, 83, 84, 85, 86
FIGURE 48–3 There are preoperative, intraoperative, and postoperative reasons why thoracic surgery impairs postoperative lung function (see text for details). (From Benumof604)

The second reason thoracic surgery promotes postoperative pulmonary complications is that the performance of surgery can impair lung function in any patient. During actual surgery, nondependent lung function may be impaired by resection of functional lung and/or by trauma to the remaining nondependent lung (due to various nondependent lung manipulations), and dependent lung function may be impaired as a result of the development of atelectasis and edema formation. This second factor can be minimized by appropriate intraoperative management (such as one-lung ventilation, PEEP, or continuous positive airway pressure [CPAP]—see later).

The third cause of increased pulmonary complications in these patients is that thoracotomy and upper abdominal incisions are most painful and cause patients to resist deep breathing and coughing in the postoperative period, leading to retained secretions, atelectasis, and pneumonia. 82, 87, 88, 89, 90, 91, 92, 93, 94 It is painful for these patients to deep breathe and to stretch either the chest or the abdominal wall (i.e., the incision); consequently, they fail to cough (which requires a deep breath), and they retain secretions (promoting the development of atelectasis and infection). The third factor can be minimized by appropriate postoperative pain management (e.g., epidural narcotics) (Ch. 69).

Preoperative Respiratory Preparation Maneuvers

The preceding data indicate that patients undergoing thoracic surgery are particularly susceptible to postoperative respiratory complications 79, 80, 81, 82, 83, 87, 88, 89, 90, 91, 92, 93, 94 and that prophylactic measures do decrease such complications. 82, 83, 84, 85, 86, 93, 94 Consequently, preoperative evaluation should be followed by preoperative preparation efforts (Ch. 23) directed toward optimally managing any preexisting pulmonary disease. 95 In general, a full preoperative respiratory preparation regimen involves a five-pronged attack on airway disease. The five elements of the preoperative regimen are stopping smoking, dilating airways, loosening and removing secretions, and taking measures to increase motivation and education and to facilitate postoperative care (Table 48–5 and Fig. 48–4).

TABLE 48–5. Preoperative Respiratory Care Regimen
FIGURE 48–4 A full, aggressive, preoperative respiratory preparation regimen consists of a five-pronged attack: *(1)* require the patient to stop smoking, *(2)* dilate the airways, *(3)* loosen secretions, *(4)* remove secretions, and *(5)* increase patient participation. Using these five maneuvers in the numbered sequence allows them to complement one another in improving secretion removal. (From Benumof604)

The five treatment modalities are instituted and proceed in parallel fashion. Before discussing each element separately, it is important to point out that the desirable results of these maneuvers should be, for the purpose of understanding their interaction, achieved in a sequential manner (see Fig. 48–4). The logic behind this concept is as follows. First, stopping smoking eliminates the stimulus for the production of airway secretions and bronchoconstriction. Next, the airways should be dilated to facilitate secretion removal. Similarly, thick, tenacious, and adherent secretions must be loosened in order to be removed. Once the airways are dilated and the secretions loosened, then it makes sense to use physical maneuvers to remove the secretions. Finally, patients should assist, as much as possible, in their preoperative preparation and postoperative respiratory care. The studies cited below indicate that using the maneuvers in this sequence (dilating the airways, loosening the secretions, removing the secretions) allows the maneuvers to complement one another in improving mucociliary transport function. Of course, it is recognized that patients who do not have bronchospasm will not benefit from bronchodilator treatment, and patients who do not have secretions will not benefit from measures to enhance secretion removal. 96

Discontinuing Smoking

An improvement in mucociliary transport and small airway function and a decrease in airway secretions and reactivity occur over several weeks after cessation of smoking. 97, 98 Consequently, preoperative cessation of smoking for more than 4 to 8 weeks is associated with a decrease in the incidence of postoperative

respiratory complications. 97, 98 Although stopping smoking for only 24 hours will do nothing to decrease the amount of secretions (at least 1–2 weeks is required), 98, 99 airway irritability, and incidence of postoperative respiratory complications, a number of important benefits accrue in the first 1 to 2 days of abstinence. 97, 98, 99 Cessation of smoking for as short a time as 12 to 48 hours has been shown to decrease carboxyhemoglobin levels significantly (increasing hemoglobin available for oxygen transport), to shift the oxyhemoglobin dissociation curve to the right (increasing the availability of oxygen to the tissues), 100 and to reduce nicotine-induced tachycardia; 98, 99 in addition, cessation of smoking for a few days may greatly improve ciliary beating. 98, 99 All these acute effects may confer a critical benefit to the marginal patient. 84

For a few select patients, the risks of stopping smoking for 1 or 2 days may outweigh the benefits. These risks include excessive anxiety, 101 causation of a hypersecretory and bronchospastic state, 100 and increased incidence of deep vein thrombosis; 98, 99 nevertheless, a good argument can be made that these potential benefits of continuing smoking during the 1 or 2 days before surgery can just as easily be accomplished with anxiolytics, bronchodilators, and anticoagulants.

Dilating the Airways

The next preparatory step should be to dilate the airways (Ch. 14). ?2-Sympathomimetic drugs, such as albuterol, terbutaline, and metaproterenol, are administered to patients who have a demonstrable reversible bronchospastic airway component to their respiratory disease. Patients who have increased airway responsiveness and therefore are candidates for preoperative bronchodilation are smokers, 102 atopic individuals, 102 patients with airway symptoms of allergies, 102 patients with COPD, 103, 104, 105, 106, 107, 108 and asthmatics. 109, 110 The sympathomimetics (so-called first messengers) are believed to act on their target cells in the lungs by increasing the activity of adenyl cyclase, an enzyme in the cell membrane, which catalyzes the formation of cyclic adenosine monophosphate (cAMP) from adenosine triphosphate (ATP). cAMP functions as a “second messenger” in the cell, where it acts on smooth muscle to decrease tension and motility. In addition, adrenergic compounds (epinephrine, ephedrine, isoproterenol) can also increase ciliary activity; this may help in removing secretions. 111, 112

cAMP is broken down by a cytoplasmic enzyme, phosphodiesterase, whose activity can be inhibited by methylxanthines such as theophylline and aminophylline. Thus, the methylxanthines also increase cAMP but by a mechanism different from that of ?2 -adrenergic receptor agonists. Because the methylxanthines and ?2 -agonists act by different mechanisms, theophylline is often given to patients with bronchospasm already receiving ?-adrenergics, and thus they work in synergy to increase intracellular concentrations of cAMP 113, 114 (Table 48–6). In addition, aminophylline improves the contractility of the diaphragm and renders it less susceptible to fatigue. 115 Recently, however, there has been considerable concern about the short-term toxicity of inhaled ?-adrenergic agents when used in the presence of methylxanthines; specifically, myocardial ischemia might occur as a result of the drugs‘ combined effect on the heart, with resultant (and possibly fatal) ventricular arrhythmias. 116, 117 Therefore, appropriate caution should be exercised when combining inhaled ?2 -agonists with methylxanthines. 118 Adrenocorticosteroids may also be given, by either oral, parenteral, or aerosol routes, to the subgroup of patients with COPD who have especially reactive airways. 119 Steroids are not considered to be bronchodilators but probably act by decreasing mucosal edema and preventing the release of bronchoconstricting substances.

TABLE 48–6. Two Basic Different Approaches to the Use of Bronchodilators in Asthma and COPD

At present there are two basic different approaches (plans) to the use of bronchodilators in asthma and COPD patients; the essential difference between the two approaches is whether the inhaled ?2 -agonists and/or anticholinergics are considered the front-line drugs with inhaled steroids considered the second-line drugs or vice versa (i.e., inflammation is considered most important 120, 121, 122) (see Table 48–6). Although the patient‘s subjective feeling of relief is an important end point, the effect of bronchodilator drug treatment should be quantified by pulmonary function tests.

Loosening the Secretions

The next step in preparing patients for thoracic surgery should be to thin and loosen thick adherent secretions. The most important method of thinning and loosening secretions is by hydration. When tracheal mucus transport velocity is quantitatively measured by radioactive tracer methods, it can clearly be shown

that dehydration decreases and rehydration increases velocity. 123 The most common method of hydrating secretions is by use of a jet humidifier or ultrasonic nebulizer to produce a heated, sterile water aerosol that is delivered by a close-fitting mask for 20 minutes to a patient breathing spontaneously with large tidal volumes. Concurrently, continuous systemic hydration must be ensured by adequate oral intake or intravenously.

Pulmonary infection, if present, is treated according to the results of culture and sensitivity tests; broad-spectrum antibiotics such as ampicillin or a cephalosporin frequently have the required specificity and potency. If the antibiotic treatment clears the infection to any extent, it may also decrease the tenacity, viscosity, and volume of secretions.

Removing the Secretions

The next preoperative respiratory preparation step is the actual removal of secretions, and this is accomplished by a combination of postural drainage (several different positions may be required), coughing, and chest percussion and vibration (common methods include tapping with cupped hands and use of electric vibrators) for 15 to 20 minutes several times a day. 101, 124, 125

When removal of tracheobronchial secretions is quantitatively measured with radioactive tracer methods in patients with COPD, chest physiotherapy (chest percussion and vibration along with postural drainage) with cough is effective in increasing both central and peripheral airway clearance and sputum yield, whereas cough alone is effective in increasing only central airway clearance and sputum yield. 126 Thus, chest physiotherapy moves peripheral bronchial secretions to more central airways for expectoration by coughing. The reason cough alone cannot clear peripheral airways is that an effective cough must attain a high enough airflow rate so as to shear secretions away from the airway wall. In patients with chronic lung disease, flow rates are low (especially peripherally), and the shearing of secretions by cough may well be limited to the trachea and perhaps just the first two airway generations. 127 Obviously, with either chest physiotherapy or cough it will be much easier to expel the secretions if the airways have already been dilated and the secretions loosened.

Chest physical therapy is relatively contraindicated in patients with lung abscesses, metastases to the ribs, a history of significant hemoptysis, and inability to tolerate the postural drainage positions. It is generally agreed that intermittent positive-pressure breathing regimens do not have sufficient efficacy to warrant the excessive cost of routine use. 128, 129, 130, 131

The forced expiration technique (FET) is increasingly being regarded as more effective in removing secretions than a cough. 132 The FET comprises a forced expiration starting from midlung volume (50% of inspiratory reserve lung volume) to a low lung volume, usually the RV, followed by a period of relaxation and diaphragmatic breathing. This forceful expiration maneuver differs from a cough because it is performed without closure of the glottis and without the accompanying compressive phase that characterizes cough. The transpulmonary pressure is less during the FET than during cough, resulting in less airway compression and permitting improved proximal and distal clearance of bronchial secretions as compared with a conventional cough (documented by radioaerosol techniques). 132 The FET is now being increasingly used as an alternative to cough during chest physiotherapy. 132

Measures to Increase Motivation and Postoperative Care

The last step of preoperative preparation consists of general measures designed to increase motivation and education and to facilitate postoperative respiratory care. These measures include stabilization of all other medical conditions, psychologic preparation, improvement of nutrition, exercise, weight loss in the obese, use of oxygen (where appropriate), antibiotics, instruction about realistic expectations of postoperative pain, and education in postoperative respiratory care procedures (including incentive spirometry, chest physiotherapy, postural drainage, and ambulation and exercise programs).

Preoperative Use of Digitalis

The preoperative use of digitalis in the thoracic surgical patient deserves special comment. Resection of pulmonary tissue reduces the available pulmonary vascular bed for perfusion and can cause postoperative RV and right atrial enlargement. Thus, it is not surprising that the incidence of postoperative arrhythmias

(due to atrial stretching) increases progressively with age and amount of lung resected. In addition, there is a higher incidence of atrial arrhythmias after left than after right pneumonectomy because a greater degree of manipulation of the atrium occurs during the former operation. Although the postoperative incidence of arrhythmias has provided the basis for the prophylactic use of digitalis in thoracic surgical patients without evidence of congestive heart failure, this practice is still controversial. 133, 134, 135, 136 Widely accepted indications for preoperative digitalization in patients without cor pulmonale undergoing thoracic surgery include congestive (left-sided) heart failure and supraventricular arrhythmias with a rapid ventricular response. 137

The indications for preoperative digitalization are more straightforward in patients with cor pulmonale. However, it is important to note that these patients have a propensity to develop hypoxemia, hypercarbia, and acidosis, and they are therefore at an increased risk of developing digitalis toxicity; thus the drug should be used with caution. 138, 139 If these patients do receive digitalis preoperatively, it is important to normalize the serum potassium in order to decrease the risk of arrhythmias. Digitalis should probably be withheld on the day of surgery to help prevent confusion with digitalis intoxication if arrhythmias occur postoperatively. 140

THE INTRAOPERATIVE PERIOD

Monitoring Requirements

Monitoring requirements (Chs. 30, 31, 32, 33) differ among individual patients for two reasons. First, patients undergoing thoracic operations have varying degrees of preexisting cardiorespiratory disease. Second, the very nature of thoracic procedures (e.g., one-lung ventilation) causes further derangements in respiratory and cardiovascular function in the perioperative period. On the basis of these two considerations and their interactions, individual patients can and should be categorized into a progressively sophisticated and complex tier system with regard to what monitoring is necessary to make possible accurate and rapid diagnosis and therapy during anesthesia. It should be apparent from this monitoring approach that an individual with severe pulmonary disease who is to undergo a minor surgical procedure may well require as extensive a monitoring system as a patient with normal lungs who is to have extensive thoracic surgery.

Table 48–7 presents three major categories for patients undergoing thoracic surgery and recommends the monitoring required for each category. The first category (tier I) includes healthy patients without special intraoperative conditions, such as a young patient undergoing pleurodesis. This tier contains the minimal, yet essential, monitoring required for any patient undergoing a thoracic procedure.

TABLE 48–7. Tiered Monitoring System Based on Amount of Pre-existing Lung Disease and Presence of Special Intraoperative Conditions

Because failure to check equipment properly before the induction of anesthesia is responsible for 22 percent of critical incidents that occur during anesthesia, 141 the function of the anesthesia machine and ventilator must be assessed preoperatively in an orderly and complete fashion.* 142 Failure of the oxygen delivery system to the patient must be signaled by audiovisual alarms from an inspired oxygen monitor. Pulse oximetry will also serve as a relatively late indicator of inappropriately decreased inspired oxygen fraction (FIO_2) to the patient. Observation of the respiratory rate and movements of the rebreathing bag, ventilator bellows, and chest wall permits rough estimation of minute ventilation and, along with constant use of a stethoscope to hear breath sounds, provides for rapid diagnosis of a breathing circuit disconnect or apnea. However end-tidal carbon dioxide ($ETCO_2$) monitoring allows precise breath-by-breath analysis and diagnosis of all of these respiratory functions (apnea, minute ventilation, gas exchange) and is therefore now an essential monitor. In addition, because the slope of the alveolar plateau correlates positively with airway resistance, $ETCO_2$ can function as a rough continuous monitor of airway mechanics. The stethoscope is also used to assess airway mechanics (detection of bronchospasm), along with an “educated” hand on the rebreathing bag (manual detection of total lung compliance and resistance). Adequacy of gas exchange is further assessed by observing the color of shed blood, looking for cyanosis (nailbeds, lips, mucous membranes, skin), and listening to the character of the breath sounds. More importantly, pulse oximetry allows precise, on-line, beat-by-beat, continuous monitoring of arterial oxygenation and is therefore now an essential monitor. Pulse oximetry also indicates desaturation when blood pressure or blood flow is very low and is therefore an on-line monitor of severe changes in cardiovascular function. Because pulse oximetry is a crude monitor in this regard, cardiovascular function must be monitored by frequent use of the blood

pressure cuff or, increasingly commonly, an automated oscillometric blood pressure monitor and by continuous ECG. With constant minute ventilation, acute changes in ETCO2 are proportional to acute changes in cardiac output (i.e., pulmonary blood flow). Muscle relaxation is assessed by simple observation for motor activity and by a peripheral nerve stimulator. Body temperature is continuously measured by a probe. These recommendations for monitoring are routine for anesthesia for nearly all kinds of surgery.

It should be emphasized that if a serious question arises about the adequacy of arterial oxygenation and/or minute ventilation while the pulse oximeter and ETCO2 monitor, respectively, are being used, arterial blood gases should be drawn (see tier II); thus, pulse oximetry and ETCO2 monitoring should not be regarded as a total substitute for arterial blood gases. However, venous blood from the back of a warmed hand or large neck vein obtained with no tourniquet or a short-time low-pressure tourniquet has a PCO2 near enough to that of arterial blood (usually 4–8 mm Hg) to be useful for most clinical purposes. 143, 144 In addition, under these sampling conditions a sufficiently high oxygen tension and saturation of venous blood (>40 mm Hg and 75%, respectively) provides a good indication that arterial hypoxemia is most probably absent. As an alternative sampling site, capillary earlobe blood (obtained by needle prick or cut) is usually acceptable for clinical management, because this blood has a PaCO2 ± 2.0 percent of simultaneous arterial samples. 145

The second tier in Table 48–7 represents an increase in risk, caused by either special unfavorable intraoperative conditions for relatively healthy patients or significant preexisting cardiopulmonary disease in patients who will not experience special intraoperative conditions. An example of the former circumstance is a patient with mild lung disease having a lobectomy. An example of the latter circumstance is a patient with moderate to severe interstitial lung disease who requires an open lung biopsy. Anyone with some lung disease undergoing one-lung ventilation for a major thoracic procedure must be considered to be in a tier II category. For this second monitoring level, in addition to the preceding essential system (tier I), the following monitoring modalities are required: a respirometer to permit accurate measurement of tidal volume and minute ventilation (the ventilator bellows can be very inaccurate); arterial blood gas analysis to ensure adequate ventilation and oxygenation; an inspiratory pressure gauge to calculate and to follow changes in whole-lung and individual-lung dynamic and static compliance; accurate measurements of intake (intravenous fluids) and output (blood loss, urine output, irrigation fluids, estimation of third space loss); central venous pressure measurement to assess intravascular volume status (depending on expected fluid shifts, condition of the patient‘s heart); and an automated oscillotonometric blood pressure device or directly transduced arterial pressure for more continuous and accurate systemic blood pressure measurements.

Finally, a third tier of monitoring requirements is constructed for patients with significant preexisting cardiopulmonary disease who will experience further compromising intraoperative conditions. An example of such a patient is one with cor pulmonale undergoing lobectomy or pneumonectomy. For this high-risk group of patients, the anesthesiologist should consider the following additional monitoring: the adequacy of tissue oxygenation continuously assessed by in-line arterial or mixed venous PO2 and PCO2; measurement of mixed venous oxygen saturation (), which is especially helpful as an index of global well-being in that a major decrease is probably never a good event (is decreased by either a decrease in cardiac output, an increase in oxygen consumption, or a decrease in arterial oxygen content [CaO2]); an esophageal ECG obtained by modifying an esophageal stethoscope and used to differentiate atrial arrhythmias 146 ; a V5 ECG lead to detect myocardial ischemia by analysis of the ST segment of the ECG, which is especially important in patients with coronary artery disease; and a pulmonary artery catheter with a thermistor to permit continuous measurement of intravascular filling pressures and rapid determination of cardiac output (and therefore myocardial work indices and assessment of myocardial contractility). Advanced monitoring mainly confined to research at this time includes measurement of right-to-left transpulmonary shunt, ventilation dead space, airway resistance, and aortic pulse pressure contour for beat-to-beat estimation of cardiac output and myocardial contractility.

Two of the preceding monitoring recommendations deserve special discussion and emphasis: arterial cannulation and pulmonary artery catheterization. Direct arterial cannulation (usually of the radial artery) permits frequent arterial blood gas analysis (and perhaps continuous in-line measurement of arterial PO2 and PCO2); this alone is sufficient justification for arterial line placement for patients with double-lumen endotracheal tubes and/or serious respiratory disease. In addition, an arterial line permits continuous measurement of systemic blood pressure; similarly, this alone is frequently sufficient justification for arterial line placement in patients with serious cardiovascular compromise. When it is used with a paper readout, the “trend” in arterial pressure is demonstrated. An electrical mean pressure can be calculated, allowing a more

precise measurement of perfusion pressure. Finally, an increase in positive-pressure-induced variation in systolic blood pressure may be an early indicator of hypovolemia. 147

Normally, the central venous pressure is an adequate index of intravascular volume status. With progressive pulmonary disease and/or LV dysfunction, however, the left- and right-sided cardiac circulations must be assessed separately, because the central venous pressure may no longer reflect left-sided filling pressure (pulmonary artery wedge pressure [Ppaw]). In addition, with significant pulmonary disease and/or tachycardia, the pulmonary artery diastolic pressure will be elevated above the pulmonary artery wedge pressure, and a significant pulmonary artery diastolic-to-wedge pressure gradient will be present. In this situation only the wedge pressure can be used to reflect left-sided cardiac volume/ compliance status. Finally, cardiac output is easily determined by using pulmonary artery catheter thermodilution techniques. For these reasons, the use of a pulmonary artery rather than a central venous catheter should be considered if pulmonary hypertension and/or cor pulmonale and coronary artery disease are present, especially if extensive perioperative fluid shifts or blood loss is anticipated.

Special Pulmonary Vascular Monitoring Considerations Related to Thoracotomy in the Lateral Decubitus Position

Pulmonary artery catheters usually (in >90% of cases) float to and locate in the right lung. 148 Consequently, during a right thoracotomy (left lateral decubitus position), the pulmonary artery catheter will be in the nondependent lung and therefore either in a collapsed lung if one-lung ventilation is used or possibly in a zone 1 or 2 region of the lung if large tidal volume two-lung ventilation is used. Conversely, when a left thoracotomy is performed (patient in the right lateral decubitus position), the pulmonary artery catheter will be in the dependent lung and will probably be in a zone 3 region. Thus, it is theoretically possible that the pulmonary artery catheter might function differently or yield different pulmonary vascular pressure and cardiac output data during right versus left thoracotomies and during two-lung versus one-lung ventilation.

Indeed, with the pulmonary artery catheter tip located in the right lung, the cardiac output is lower during right thoracotomy with one-lung ventilation (right lung collapsed) than during left thoracotomy with one-lung ventilation (left lung collapsed) in patients who were otherwise similar 149 (Fig. 48–5A). Consequently, it is possible that when the pulmonary artery catheter is located in the collapsed lung, where blood flow patterns may be distorted or the function of the thermistor interfered with (so that it is not free in the lumen of the vessel), the measured output is indeed lower. This hypothesis is supported by the concurrent finding that continuously measured is also decreased during right thoracotomies as compared with left thoracotomies when the pulmonary artery catheter is located in the collapsed nondependent lung. The decrease in may have been caused by stagnant blood flow and therefore not truly representative of whole-patient . 149 When the nondependent lung is ventilated with varying levels of PEEP (in contrast to nondependent lung collapse), there is no difference in the cardiac output measured simultaneously from thermistors located in the nondependent and dependent lungs. 150 This finding implies that when the pulmonary artery catheter tip is in the nondependent lung and the nondependent lung is ventilated, blood flow to the nondependent lung is undistorted and/or there is no interference with the function of the thermistor.
FIGURE 48–5 Conditions during thoracotomy in the lateral decubitus position when pulmonary artery catheter data may be inaccurate. (A) During right thoracotomy with a pulmonary artery (PA) catheter located in the collapsed right lung (one-lung ventilation [1 LV]), the cardiac output (CO) may be lower than when the right lung is ventilated. The thermistor in the collapsed lung may be exposed to abnormal flow patterns or vascular wall interference. (B) When the PA catheter is in the nondependent lung and the nondependent lung is exposed to CPAP or PEEP, the pulmonary artery wedge pressure (Ppaw) may be inaccurate. Nondependent lung CPAP or PEEP may cause zone 1 conditions in the nondependent lung. The Ppaw is probably always reasonably accurate when the PA catheter is in the dependent lung, even if the dependent lung is exposed to PEEP. (From Benumof604)

When the pulmonary artery catheter is in the nondependent lung and the nondependent lung is ventilated with large tidal volume, PEEP, or CPAP, the wedge pressure may not reflect left atrial pressure (Pla) 151 (Fig. 48–5B). When the pulmonary artery catheter is in the dependent lung and presumably in the zone 3 region, wedge pressure should accurately reflect Pla, even when PEEP is applied to the dependent lung. 151

In summary, the lateral decubitus position is important with regard to pulmonary artery catheter monitoring in three situations. First, when the nondependent lung is collapsed and the catheter is in the nondependent lung, the measured cardiac output and mixed venous blood PO2 () may be decreased compared with more normal conditions or the “real” value. Second, when the nondependent lung is ventilated with PEEP and the catheter is in the nondependent lung, Ppaw may not equal Pla. Third, when the catheter is in the dependent lung, Ppaw will be a faithful index of Pla, even if PEEP is used.

Finally, after pneumonectomy, inflation of the balloon of the pulmonary artery catheter to obtain Ppaw can result in considerable occlusion of the remaining cross-sectional area of the pulmonary circulation. This occlusion acutely decreases the preload on the left ventricle and increases the right ventricular afterload, resulting in reduced cardiac output and reduced Pla. Although the Ppaw under these circumstances still accurately reflects the Pla, both these values have been artificially lowered by the blocked pulmonary circulation; hence, they result in a falsely low Ppaw reading. 152 This falsely low value for the left ventricular filling pressure is misleading and may result in fluid management that contributes to the development of pulmonary edema and to the excessively high mortality reported in postpneumonectomy patients. Advancing the catheter carefully without inflating the balloon and wedging it into a smaller peripheral vessel can minimize the reduction in the cross-sectional area of the pulmonary vasculature. Thus, a more accurate value for Ppaw, reflecting the true Pla, can be obtained.

Choice of Anesthesia and Arterial Oxygenation During One-Lung Ventilation

Effect of Anesthetics on Hypoxic Pulmonary Vasoconstriction

Thoracic surgery may be greatly facilitated by causing selective atelectasis of the lung being operated on (one-lung ventilation/anesthesia conditions). The normal response of the pulmonary vasculature to atelectasis is an increase in PVR (in just the atelectatic lung). The mechanism of the increase in PVR is thought to be due almost entirely to hypoxic pulmonary vasoconstriction (HPV). 153, 154, 155 The selective increase in atelectatic lung PVR diverts blood flow from the atelectatic lung toward the remaining normoxic or hyperoxic ventilated lung. Figure 48–6 shows the theoretically expected effect of HPV on PaO2 as the amount of lung that is made hypoxic increases. When the percentage of lung that is hypoxic is between 30 and 70 percent, which encompasses the one-lung ventilation/anesthesia condition, there is a large difference between the PaO2 expected with a normal amount of HPV and that when there is no HPV. In fact, in this range of hypoxic lung, HPV increases the PaO2 from levels that might cause arrhythmias to much higher and safer values. Thus, HPV is an autoregulatory mechanism that protects the PaO2 by decreasing the amount of shunt flow that can occur through hypoxic lung.
FIGURE 48–6 Effect of hypoxic pulmonary vesoconstriction (HPV) on PaO2. As the amount of lung that is made hypoxic is increased (x axis), the arterial oxygen tension (PaO2) decreases (y axis). In the range of 30 to 70 percent of hypoxic lung, the normal expected amount of HPV increases PaO2 from arrhythmogenic levels to much higher and safer levels. Normal cardiac output, hemoglobin concentration, and mixed venous oxygen tension () are assumed. (Data from Marshall and Marshall209)

General anesthesia with controlled ventilation is the safest method of anesthetizing patients for the vast majority of elective thoracic procedures. Inhibition of HPV in the nonventilated nondependent lung by the anesthetic should be prevented. All the inhaled and many of the injectable anesthetics have been studied with regard to their effect on HPV; halothane has been studied most extensively (Table 48–8). 156, 157, 158, 159, 160, 161, 162, 163, 164, 165, 166, 167, 168, 169, 170, 171, 172, 173, 174, 175, 176, 177 The experimental preparations may be divided into four basic categories: (1) *in vitro;* (2) *in vivo*–not intact (pumped perfused lungs, no systemic circulation or neural function); (3) *in vivo*–intact (normally perfused lungs, normal systemic circulation); and (4) humans (volunteers or patients). It appears from this breakdown of experimental preparations that inhibition of HPV by halothane is a universal finding in the *in vitro* and *in vivo*–not intact preparations. However, in the more normal or physiologic *in vivo*–intact and human studies, halothane has caused no or only a very slight decrease in HPV response. Thus, it appears that a fundamental property of halothane is its inhibition of HPV in experimental preparations that can be controlled for other physiologic influences (e.g., pulmonary vascular pressure, cardiac output, mixed venous oxygen tension, carbon dioxide level, temperature) that can have an effect on the HPV response. In the more biologically complex *in vivo*–intact models, other factors that greatly diminish the inhibitory effect of halothane on HPV seem to be involved. Important methodologic differences between the *in vitro* and *in vivo*–not intact preparations and the *in vivo*–intact and human models that could account for the observed differences in halothane effect on HPV are the presence (or absence) of perfusion pulsations; perfusion fluid

composition; size of perfusion circuit 171 ; baroreceptor influences, absence of bronchial blood flow (which abolishes all central and autonomic nervous activity in the lung) 178 ; chemical influences (e.g., pH, PO2); humoral influences (e.g., histamine and prostaglandin release from body tissues); lymph flow influences; and, very importantly, unaccounted for or uncontrolled changes in physiologic variables (e.g., cardiac output, , and pulmonary vascular pressures) that might have directionally opposite effects on HPV, and use of different species. 179, 180, 181

TABLE 48–8. Effect of Halothane on Hypoxic Pulmonary Vasoconstriction in Various Experimental Preparations

Ether has been the next most studied drug, and it appears that the quantitative effect of ether on HPV is also dependent on the type of experimental preparation used. Thus, the *in vitro* and *in vivo*–not intact models show much more inhibition of HPV by ether than the *in vivo*–intact and human models 155, 157, 158, 161, 164, 182, 183 (Table 48–9). Although the number of studies involving halogenated drugs other than halothane, namely isoflurane, 160, 172, 173, 177, 184, 185, 186, 187, 188, 189, 190, 191 enflurane, 155, 160, 192, 193, 194 methoxyflurane, 156, 157, 158, 195, 196 fluroxene, 172, 173 and trichloroethylene 161 has been too small to permit recognition of an experimental preparation result pattern, most of these anesthetics have demonstrated inhibition of HPV (at least in the *in vitro* models) (see Table 48–9). Nitrous oxide seems to cause a small, somewhat consistent inhibition of HPV 158, 168, 172, 173, 197, 198, 199 (see Table 48–9). None of the injectable anesthetics studied to date have an effect on HPV 157, 158, 169, 172, 194, 200, 201, 202, 203, 204, 205 (see Table 48–9).

TABLE 48–9. Effect of Ether, Isoflurane, Enflurane, Methoxyflurane, Fluroxene, Trichloroethylene, Nitrous Oxide, and Injectable Anesthetics on Hypoxic Pulmonary Vasoconstriction

To summarize previous animal studies, it appears that a fundamental property of inhaled anesthetics is to decrease HPV. However, in intact animal preparations some biologic or physiologic property seems to remove or greatly lessen the inhibitory effect of anesthetic drugs on HPV. It may be that the cause(s) of the difference in effect of anesthetic drugs on regional HPV from preparation to preparation, anesthetic to anesthetic, and species to species (see later) are closely related to the fundamental mechanism of HPV, which is still unknown.

Effect of Anesthetics on Arterial Oxygenation During One-Lung Ventilation

An often-made extrapolation of the much more numerous *in vitro* and *in vivo*–not intact HPV studies is that anesthetic drugs might impair arterial oxygenation during one-lung anesthesia by inhibiting HPV in the nonventilated lung. One of the previously cited studies on the effect of isoflurane on regional canine HPV was especially well controlled and showed that when all nonanesthetic drug variables that might change regional HPV are kept constant, isoflurane inhibits single-lung HPV in a dose-dependent manner. 186 Additionally, the study is valuable because the authors offer the reader an easily comprehensible quantitative summary of the relationship between dose of isoflurane administered and degree of inhibition of the single-lung canine HPV response. If the summary can be extrapolated or applied to the clinical one-lung ventilation situation (at least as an approximation), insights can be gained into what might be expected with regard to arterial oxygenation when such patients are anesthetized with isoflurane. To put this insight into clinical focus, it is necessary first to understand what should happen to blood flow, shunt flow, and arterial oxygenation as a function of a normal amount of HPV when two-lung ventilation is changed to one-lung ventilation in the lateral decubitus position (LDP). Once the stable one-lung ventilation condition has been described, it is possible, by using the data from the previously mentioned study, to see how isoflurane administration would affect the one-lung ventilation blood flow distribution, shunt flow, and arterial oxygen tension (PaO2).

Two-Lung Ventilation: Blood Flow Distribution

Gravity causes a vertical gradient in the distribution of pulmonary blood flow in the LDP for the same reason that it does in the upright position. Consequently, blood flow to the dependent lung is significantly greater than blood flow to the nondependent lung. When the right lung is nondependent, it should receive approximately 45 percent of total blood flow as opposed to the 55 percent that it received in the upright and supine positions. When the left lung is nondependent, it should receive approximately 35 percent of total blood flow as opposed to the 45 percent that it received in the upright and supine positions (closed-chest

data with normal pulmonary artery pressure). 206, 207, 208 If these blood flow distributions are combined (both the right and left lungs being nondependent an equal number of times), average two-lung ventilation blood flow distribution in the lateral decubitus position would consist of 40 percent of total blood flow perfusing the nondependent lung and 60 percent of total blood flow perfusing the dependent lung (Fig. 48–7, left-hand panel).

FIGURE 48–7 Effect of 1 MAC isoflurane anesthesia on shunt during one-lung ventilation (1LV) of normal lungs. This diagram shows that for two-lung ventilation the ratio of the percentages of blood flow to the nondependent and dependent lungs is 40:60 (left-hand side). When two-lung ventilation is converted to one-lung ventilation (as indicated by atelectasis of the nondependent lung), the HPV response decreases the blood flow to the nondependent lung by 50 percent, so that the nondependent/dependent lung blood flow ratio is now 20:80 (middle). According to the data of Domino et al186 administration of 1 MAC isoflurane anesthesia should cause a 21 percent decrease in the HPV response, which would decrease the blood flow reduction from 50 to 40 percent. Consequently, the nondependent/dependent lung blood flow ratio would now become 24:76, representing a 4 percent increase in the total shunt across the lungs (right-hand side). (From Benumof603)

One-Lung Ventilation: Blood Flow Distribution, Shunt Flow, and Arterial Oxygen Tension

When the nondependent lung is nonventilated (made at-electatic), HPV in this lung will increase its PVR and de-crease its blood flow. In the absence of any confounding or inhibiting factors to the HPV response, a single-lung HPV response should decrease the blood flow to that lung by 50 percent. 209 Consequently, the nondependent lung should be able to reduce its blood flow from 40 to 20 percent of total blood flow, and the nondependent/dependent lung blood flow ratio during one-lung ventilation should be 20:80 (see Fig. 48–7, middle panel).

All the blood flow to the nonventilated nondependent lung is shunt flow, and therefore one-lung ventilation creates an obligatory right-to-left transpulmonary shunt flow that was not present during two-lung ventilation. If no shunt existed during two-lung ventilation (ignoring the normal 1–3% shunt flow due to the bronchial, pleural, and thebesian circulations), we would expect the ideal total shunt flow during one-lung ventilation (i.e., with intact HPV) to be 20 percent of total blood flow. With a normal hemodynamic and metabolic state, the PaO2 should be approximately 280 mm Hg. 210 Other much more complicated models have been constructed to describe what to expect in terms of shunt during one-lung ventilation when varying degrees of shunt exist during two-lung ventilation. 211

Effect of Isoflurane on One-Lung Ventilation Blood Flow Distribution, Shunt Flow, and Arterial Oxygen Tension

Domino et al 186 found the percentage inhibition of regional HPV response to equal 22.8 (percentage alveolar isoflurane) minus 5.3 (see Fig. 48–7, top equation). 186 The top equation and right-hand panel of Fig. 48–7 show that isoflurane anesthesia at one minimum alveolar concentration unit (1 MAC) would inhibit the nondependent lung HPV response by approximately 21 percent, which would decrease this response from a 50 to 40 percent blood flow reduction in this lung; this in turn would increase nondependent lung blood flow from 20 to 24 percent of total blood flow, causing shunt to increase by 4 percent of the cardiac output and PaO2 to decrease a moderate amount to 205 mm Hg ($FIO_2 = 1.0$). A decrease in PaO2 and an increase in shunt of this magnitude are small and may not be detectable given the usual accuracy of clinical methodology. In fact, in clinical one-lung ventilation studies involving intravenously anesthetized patients with this level of shunting, administration of 1 MAC isoflurane (and halothane) anesthesia during stable one-lung ventilation conditions causes no detectable decrease in PaO2. 177, 212 In one of these clinical studies, 212 stable one-lung ventilation conditions in the lateral decubitus position were established in patients who were anesthetized with only intravenous drugs. While stable one-lung ventilation was maintained, inhaled anesthetics were administered (halothane and isoflurane end-tidal concentrations were greater than 1 MAC for at least 15 minutes) and then discontinued (halothane and isoflurane end-tidal concentrations decreased to near zero). In the other study, 177 steady-state one-lung ventilation conditions in the LDP were established in patients who were anesthetized with only inhaled drugs (halothane and isoflurane end-tidal concentrations were greater than 1 MAC for more than 40 minutes). While one-lung ventilation was continued, inhaled anesthesia was then discontinued and intravenous anesthesia administered (halothane and isoflurane end-tidal concentrations decreased to near 0). There was no significant difference in PaO2 during inhaled anesthesia with either halothane or isoflurane as compared with intravenous anesthesia during one-lung ventilation in either of the two experimental sequences. In

addition, there were no significant changes in physiologic variables, such as cardiac output, PVR, and , that might secondarily alter nondependent lung HPV. Thus, irrespective of whether inhaled anesthesia is administered before or after intravenous anesthesia during one-lung ventilation, inhaled anesthesia does not further impair arterial oxygenation. These findings are consistent with the interpretation that 1 MAC halothane or isoflurane in patients with a moderate level of shunting does not inhibit HPV enough to cause a significant decrease in PaO2 during one-lung ventilation in the LDP. Almost identical results have been obtained during one-lung ventilation with nitrogen (while the other lung was ventilated with 100 percent oxygen) of volunteers who were alternately anesthetized with isoflurane and intravenous drugs 191 and enflurane and intravenous drugs. 193

Recommended Anesthesia Induction and Maintenance Drugs and Techniques

Summary of Advantages of Anesthetic Drugs

Inhaled Anesthetics.

General anesthesia with controlled ventilation (Chs. 3– 7) is the safest method of anesthetizing patients for the vast majority of elective thoracic procedures. Although a variety of general anesthesia techniques can be used, the volatile halogenated anesthetic drugs are good choices for several reasons. First, the halogenated drugs have a salutary effect on airway irritability. The mechanism of this action is controversial, but as previously discussed, there is evidence that these drugs can block specific forms of bronchoconstriction, 213, 214 as well as having a nonspecific bronchodilating effect related to the depth of anesthesia. 215 Obtundation of airway reflexes in patients who have reactive airways (i.e., smokers) and who may have their airways directly manipulated by the surgeon is a highly desirable property of the general anesthesia produced by these drugs. Second, the use of volatile halogenated drugs allows delivery of a high inspired oxygen concentration without loss of anesthesia. Although a nitrous oxide-oxygen-narcotic-relaxant anesthesia technique can be used, nitrous oxide necessitates a significant decrease in FIO2 and increases the chance of developing hypoxemia (especially if one-lung ventilation is used). 216 Unless very high doses of narcotics are used, airway reflexes and reactivity may remain at a high level. Third, because the volatile halogenated drugs can be rapidly eliminated, concern about postoperative hypoventilation in extubated patients may be diminished. Relatively high doses of intravenous anesthetics, such as narcotics, ketamine, and barbiturates, may cause the patient to require a period of postoperative ventilation. Fourth, in the usual clinical doses (near 1 MAC), the halogenated anesthetic drugs provide a reasonable degree of cardiovascular stability. This may be of particular importance in patients who have a history of smoking and therefore a high incidence of coronary artery disease and systemic hypertension. 217, 218 Fifth, the halogenated drugs do not appear to decrease PaO2 any more than intravenous anesthetics during one-lung ventilation (see the following section). 177, 212

Intravenous Anesthetics.

The narcotics, especially fentanyl, have a number of desirable properties (Chs. 8– 11) that could be used to advantage for patients undergoing thoracic surgery. First, fentanyl has no significant adverse hemodynamic effects and therefore is a useful drug in patients who have coronary artery disease. Second, if significant blood levels exist at the end of surgery, the narcotics can allow an intubated patient to have a smooth transition from surgery into the postoperative period. Third, the narcotics, if used in moderate dosage, diminish the amount of volatile halogenated drug anesthesia required to achieve surgical levels of anesthesia. Fourth, high doses of narcotics or moderate doses in conjunction with halogenated drugs allow the use of a high FIO2 without loss of anesthesia. Fifth, the narcotics are thought not to diminish regional HPV and, therefore, should permit optimal oxygenation during one-lung ventilation.

Ketamine, in combination with nitrous oxide and a muscle relaxant, has also been used for anesthesia for thoracic surgery. 219 Although we do not ordinarily use ketamine for elective thoracic procedures, the drug is useful for induction of general anesthesia in critically ill patients undergoing emergency thoracic surgery for several reasons. First, ketamine has sympathomimetic properties 220 that are highly desirable because many emergency thoracic procedures are associated with hypovolemia (gunshot and stab wounds of the chest, blunt trauma, and massive hemoptysis). However, it should be remembered that ketamine depresses cardiovascular function (systemic blood pressure, cardiac contractility) if the degree of hypovolemia is severe and the patient is sympathetically exhausted. Second, ketamine has a rapid onset of action and can be used safely, along with cricoid pressure, to induce anesthesia in patients with full stomachs. Third,

ketamine may reduce bronchospasm in asthmatic patients 221 ; the clinical extrapolation of this effect to thoracic surgical patients is uncertain but seemingly reasonable at this time. Fourth, ketamine does not impair arterial oxygenation during one-lung ventilation (perhaps because of its lack of effect on HPV). 204

Recommended Anesthetic Drugs and Techniques

It should be obvious from the foregoing discussions that there are advantages and disadvantages to both inhaled and intravenous anesthetic drugs. One recent study made this point very well by examining the respiratory and cardiac differences between various anesthetic regimens (intravenous-based, inhalation-based, epidural-based) before and during one-lung ventilation. 222 The 36 patients were randomly allocated to one of the following groups: A—propofol 10 mg/kg/h, fentanyl; B—1 MAC enflurane, fentanyl; C—thoracic epidural anesthesia, 0.4 percent enflurane. Before induction of anesthesia significant differences were not found. During two-lung ventilation cardiac index was significantly decreased in the B in comparison with the C group (*P* ? .01). During one-lung ventilation significant differences were not found except for an increased shunt fraction (QS/QT) in group B (A versus B, *P* ? .05; B versus C, *P* ? .05; A versus C, not significant). Since QS/QT was significantly increased and hypoxemia occurred with regimen B, regimen A or C might be preferred in patients at high risk for hypoxia or in situations where the application of CPAP to the nondependent lung is not possible. Cardiac index was best maintained in group C. Regimen C might be of value in patients at high risk for poor perfusion, taking into account both the possible complications of the epidural block and the reduced need for postoperative analgesic agents.

The following recommended anesthetic technique takes advantage of the desirable properties and minimizes the undesirable properties of these drugs. Thus, the halogenated drugs are used for their effect on bronchomotor tone, to allow administration of 100 percent oxygen, and to allow early extubation without decreasing hemodynamic function and arterial oxygenation; fentanyl is used to ensure hemodynamic stability without jeopardizing early extubation if desired. If it is thought that the patient will not be extubated early or if greater hemodynamic stability is desired, anesthesia consisting of more fentanyl and less halogenated drug can be used.

Induction of Anesthesia.

The patient is preoxygenated by spontaneously breathing 100 percent oxygen through an anesthesia mask that is connected to an anesthesia circle system. Fentanyl is administered intravenously until the respiratory rate is approximately 8 to 10 breaths/min. This usually corresponds to a dose of 3 to 6 ?g/kg and is generally administered over several minutes. When the respiratory rate is relatively slow and deep and response to commands is becoming sluggish, a small dose of sodium thiopental (2–3 mg/kg) or ketamine (1.0–2.0 mg/kg) (if the patient is thought to have an especially reactive airway or to be minimally to moderately hypovolemic) is administered to render the patient unconscious and usually apneic. Control of the airway is then established and ventilation is begun with intermittent positive-pressure oxygen via the mask. While the patient is being ventilated with positive pressure, a concentration of 0.5 to 2.5 percent isoflurane is administered. The higher isoflurane concentration is used initially for a short period of time (overpressure, 1–2 minutes); as the patient demonstrates signs of deepening anesthesia, the inspired isoflurane concentration is decreased. In view of the fact that general anesthetics significantly decrease the ventilatory response to carbon dioxide (to a much greater degree in patients with mechanical ventilatory impairment than in normal patients), patients are not allowed to breathe spontaneously until the end of the procedure; alarming degrees of hypercapnia have been observed in similar circumstances when spontaneous ventilation was allowed. 223

Early during the period of positive-pressure ventilation with isoflurane, paralysis is induced with a nondepolarizing muscle relaxant. The development of full paralysis is noted with a neuromuscular blockade monitor. During the period of deepening isoflurane anesthesia and paralysis, blood pressure is supported with an infusion of approximately 10 mL/kg crystalloid followed by small doses of vasopressors if needed. When the patient has been judged to have been adequately (surgical stage) anesthetized (as ascertained by changes in blood pressure, heart rate, and eye signs [the eyes should be central, conjugate, fixed, staring, without tears, and with nondilated pupils]) and paralyzed in this manner, 1 mg/kg lidocaine is administered intravenously, laryngoscopy is performed, the tracheobronchial tree is sprayed with a laryngotracheobronchial topical anesthesia spray system, and the trachea is intubated with a doublelumen tube. The intravenous and intratracheal lidocaine should diminish both airway and cardiovascular response to endotracheal intubation. 224 The patient is then ventilated with maintenance doses of isoflurane and

given maintenance doses of narcotics and relaxants. Use of maintenance paralysis decreases isoflurane requirements, possibly allowing for a more rapid emergence from anesthesia.

Maintenance of Anesthesia.

Anesthesia is maintained with both isoflurane (concentration approximately 0.5–1.0 MAC) and narcotics. Isoflurane is used primarily if the patient is thought to have a reasonable chance of being extubated within the first couple of hours postoperatively. Narcotics (fentanyl) are primarily used if the patient is thought not to have a reasonable chance of being extubated in the immediate postoperative period and to require a significant period of postoperative ventilation. Relaxants are administered in small doses to keep the level of neuromuscular blockade, as judged by a neuromuscular blockade monitor, near the 90 percent paralysis level. If there is a reasonable chance of extubating the trachea in the first postoperative hour, the patient is turned supine, the double-lumen tube changed to a single-lumen tube, paralysis reversed, and spontaneous ventilation allowed to recur. Fentanyl is administered in extremely small increments (0.3 ?g/kg) while the patient is breathing spontaneously. The goal of the fentanyl administration is to have the patient breathing relatively slowly (approximately 10 to 12 breaths/min) and deeply when surgery is completed. The presence of a moderate narcotic base allows the patient to be returned to the recovery room for a short period of mechanical ventilatory support (if needed) and weaned and extubated in a relatively smooth manner.

THE INTRAOPERATIVE PERIOD - continued

Physiology of Spontaneous Ventilation with an Open Chest

Mediastinal Shift

An examination of the physiology of the open chest during spontaneous ventilation reveals why controlled positive-pressure ventilation is the only practical way to provide adequate gas exchange during thoracotomy. In the spontaneously breathing closed-chest patient in the LDP, gravity causes the pleural pressure in the dependent hemithorax to be less negative than in the nondependent hemithorax, but there is still negative pressure in each hemithorax on each side of the mediastinum. In addition, the weight of the mediastinum causes some compression of the lower lung, contributing to the pleural pressure gradient. With the nondependent hemithorax open, atmospheric pressure in that cavity exceeds the negative pleural pressure in the dependent hemithorax; this imbalance of pressure on the two sides of the mediastinum causes a further downward displacement of the mediastinum into the dependent thorax. During inspiration the caudad movement of the dependent-lung diaphragm increases the negative pressure in the dependent lung and causes a still further displacement of the mediastinum into the dependent hemithorax. During expiration, as the dependent-lung diaphragm moves cephalad, the pressure in the dependent hemithorax becomes relatively positive, and the mediastinum is pushed upward out of the dependent hemithorax (Fig. 48–8). Thus, the tidal volume in the dependent lung is decreased by an amount equal to the inspiratory displacement caused by mediastinal movement. This phenomenon is called *mediastinal shift* and is one mechanism that results in impaired ventilation in the open-chested, spontaneously breathing patient in the LDP. The mediastinal shift can also cause circulatory changes (decreased venous return) and reflexes (sympathetic activation) that result in a clinical picture similar to shock: the patient is hypotensive, pale, and cold, with dilated pupils. Local anesthetic infiltration of the pulmonary plexus at the hilum and the vagus nerve can diminish these reflexes. More practically, controlled positive-pressure ventilation abolishes these ventilatory and circulatory changes associated with mediastinal shift.

FIGURE 48–8 Schematic representation of mediastinal shift and paradoxical respiration in the spontaneously ventilating patient with an open chest who is placed in the lateral decubitus position. The open chest is always exposed to atmospheric pressure (+). During inspiration, negative pressure (–) in the intact hemithorax causes the mediastinum to move downward (mediastinal shift). In addition, during inspiration, movement of gas from the nondependent lung in the open hemithorax into the dependent lung in the closed hemithorax and movement of air from the environment into the open hemithorax causes the lung in the open hemithorax to collapse (paradoxic respiration). During expiration, relative positive (+) in the closed hemithorax causes the mediastinum to move upward (mediastinal shift). In addition, during expiration, the gas moves from the dependent lung to the nondependent lung and from the open hemithorax to the environment; consequently, the nondependent lung expands during expiration (paradoxic respiration). (From Benumof604)

Paradoxic Respiration

When a pleural cavity is exposed to atmospheric pressure, the lung is no longer held open by negative intrapleural pressure, and it tends to collapse because of unopposed elastic recoil. 225 Thus, the lung in an open chest is at least partially collapsed. It has long been observed during spontaneous ventilation with an open hemithorax that lung collapse is accentuated during inspiration, and, conversely, the lung expands during expiration. This reversal of lung movement during respiration with an open chest has been termed *paradoxic respiration.* The mechanism of paradoxic respiration is similar to that of mediastinal shift. During inspiration, the descent of the diaphragm on the side of the open hemithorax causes air from the environment to enter the pleural cavity on that side through the thoracotomy opening and to fill the space around the exposed lung. The descent of the hemidiaphragm on the closed-chest side causes gas to enter the closed-chest lung in the normal manner. However, gas also enters the closed-chest lung (which has a relatively negative pressure) from the open-chest lung (which remains at atmospheric pressure); this results in further reduction in the size of the open-chest lung during inspiration. During expiration the reverse occurs, with the collapsed, open-chest lung filling from the intact lung and air moving back out of the exposed hemithorax through the thoracotomy incision. The phenomenon of paradoxic respiration is illustrated in Figure 48–8. Paradoxic breathing is increased by a large thoracotomy and by increased airway resistance in the intact lung. Paradoxic respiration may be prevented either by manual collapse of the open-chest lung or, more commonly, by controlled positive-pressure ventilation.

Physiology of the Lateral Decubitus Position and the Open Chest During Controlled Two-Lung Ventilation: Distribution of Perfusion and Ventilation

Awake, Closed-Chest, Lateral Decubitus Position

Gravity causes a vertical gradient in the distribution of pulmonary blood flow in the LDP for the same reason that it does in the upright position (Ch. 15). Because the vertical hydrostatic gradient is less in the LDP than in the upright position, there is ordinarily less zone 1 blood flow (in the nondependent lung) in the former than in the latter position. Nevertheless, blood flow to the dependent lung is still significantly greater than that to the nondependent lung (Fig. 48–9). Thus, when the right lung is nondependent, it should receive approximately 45 percent of total blood flow as opposed to the 55 percent that it receives in the upright and supine positions. When the left lung is nondependent, it should receive approximately 35 percent of total blood flow, as opposed to the 45 percent that it receives in the upright and supine positions. 206, 207
FIGURE 48–9 Schematic representation of the effects of gravity on the distribution of pulmonary blood flow in the lateral decubitus position. The vertical gradient in the lateral decubitus position is less than in the upright position. Consequently, there is less zone 1 and more zone 2 and 3 blood flow in the lateral decubitus position than in the upright position. Nevertheless, pulmonary blood flow increases with lung dependency and is greater in the dependent lung than in the nondependent lung. PA, alveolar pressure; Ppa, pulmonary artery pressure; Ppv, pulmonary venous pressure. (From Benumof604)

Because gravity also causes a vertical gradient in pleural pressure (Ppl) in the LDP, ventilation is relatively increased in the dependent as compared with the nondependent lung (Fig. 48–10). In addition, in the LDP the dome of the lower diaphragm is pushed higher into the chest than the dome of the upper diaphragm; therefore, the lower diaphragm is more sharply curved than the upper diaphragm. As a result, the lower diaphragm is able to contract more efficiently during spontaneous respiration. Thus, in the awake patient in the LDP, the lower lung is normally better ventilated than the upper lung, regardless of the side on which the patient is lying, although there remains a tendency toward greater ventilation of the larger right lung. 226
Because there is greater perfusion to the lower lung, the preferential ventilation to the lower lung is matched by its increased perfusion, so that the distribution of the V/Q ratios of the two lungs is not greatly altered when the awake subject assumes the LDP. Because perfusion increases to a greater extent than ventilation with lung dependency, the V/Q ratio decreases from the nondependent to the dependent lung (just as it does in upright and supine lungs).
FIGURE 48–10 Awake, closed chest distribution of ventilation. (A) Pleural pressure (Ppl) in the awake upright patient is most positive in the dependent portion of the lung, and alveoli in this region are therefore most compressed and have the least volume. Pleural pressure is least positive (most negative) at the apex of the lung, and alveoli in this region are therefore least compressed and have the largest volume. When these regional differences in alveolar volume are translated over to a regional transpulmonary pressure–alveolar volume curve, the small dependent alveoli are on a steep (large-slope) portion of the curve, and the large nondependent alveoli are on a flat (small-slope) portion of the curve. In this diagram regional slope equals regional compliance. Thus, for a given and equal change in transpulmonary pressure, the dependent

part of the lung receives a much larger share of the tidal volume than the nondependent part of the lung. (B) In the lateral decubitus position gravity also causes pleural pressure gradients and therefore similarly affects the distribution of ventilation. The dependent lung lies on a relatively steep portion and the nondependent lung lies on a relatively flat portion of the pressure-volume curve. Thus, in the lateral decubitus position the dependent lung receives the majority of the tidal ventilation. V, alveolar volume; P, transpulmonary pressure. (From Benumof604)

Anesthetized, Closed-Chest, Lateral Decubitus Position

Comparison of the awake with the anesthetized patient in the LDP (Ch. 26) reveals no difference in the distribution of pulmonary blood flow between the dependent and nondependent lungs. Thus, in the anesthetized patient, the dependent lung continues to receive relatively more perfusion than the nondependent lung. The induction of general anesthesia, however, does cause significant changes in the distribution of ventilation between the two lungs.

In the LDP, more ventilation is switched from the dependent lung in the awake subject to the nondependent lung in the anesthetized patient 227, 228 (Fig. 48–11). There are several interrelated reasons for this change in the relative distribution of ventilation between the nondependent and dependent lungs. First, the induction of general anesthesia usually causes a decrease in FRC, and both lungs share in the loss of lung volume. Because each lung occupies a different initial position on the pulmonary pressure-volume curve while the subject is awake, a general anesthesia-induced reduction in the FRC of each lung causes each lung to move to a lower, but still different, portion of the pressure–volume curve (see Fig. 48–11). The dependent lung moves from an initially steep part of the curve (with the subject awake) to a lower and flatter part of the curve (after anesthesia is induced), while the nondependent lung moves from an initially flat portion of the pressure-volume curve (with the subject awake) to a lower and steeper part of the curve (after anesthesia is induced). In fact, in the LDP the ratio of nondependent to dependent lung FRC is approximately 1.5 (average values are 1,400 and 900 mL, respectively) in the adult patient. 229, 230, 231, 232 Similar findings may be expected in children. 230 Furthermore, in the LDP the compliances of the nondependent and dependent lung are 30 and 23 cm H_2O, respectively. 229 Thus, with the induction of general anesthesia, the lower lung moves to a less favorable (flat, noncompliant) portion and the upper lung to a more favorable (steep, compliant) portion of the pressure-volume curve. Second, if the anesthetized patient in the LDP is also paralyzed and mechanically ventilated, the high, curved diaphragm of the lower lung no longer confers any advantage in ventilation (as it does in the awake state), as it is no longer actively contracting. 231, 232 Third, the mediastinum rests on the lower lung and physically impedes lower lung expansion, as well as selectively decreasing lower lung FRC. Fourth, the weight of the abdominal contents pushing cephalad against the diaphragm is greatest in the dependent lung, which physically impedes lower lung expansion the most and disproportionately decreases lower lung FRC. Finally, suboptimal positioning, which fails to provide room for lower lung expansion, may considerably compress the dependent lung. Opening the nondependent hemithorax further disproportionately increases ventilation to the nondependent lung (see below).

FIGURE 48–11 The distribution of ventilation in the patient in the lateral decubitus position when awake (A) and when anesthetized (B). The induction of anesthesia has caused a loss of lung volume in both lungs, with the nondependent lung moving from a flat, noncompliant portion to a steep, compliant portion of the pressure-volume curve and the dependent lung moving from a steep, compliant part to a flat, noncompliant part of the pressure-volume curve. Thus, the anesthetized patient in a lateral decubitus position has more of the tidal ventilation in the nondependent lung (where there is the least perfusion) and less of the tidal ventilation in the dependent lung (where there is the most perfusion). V, alveolar volume; P, transpulmonary pressure. (From Benumof604)

In summary, the anesthetized patient, with or without paralysis, in the LDP and with a closed chest has a nondependent lung that is well ventilated but poorly perfused and a dependent lung that is well perfused but poorly ventilated, which results in an increased degree of V/Q mismatching. The application of PEEP to both lungs restores more of the ventilation to the lower lung. 207 Presumably, the lower lung returns to a steeper, more favorable part of the pressure-volume curve, and the upper lung resumes its original position on a flat, unfavorable portion of the curve.

Anesthetized, Open-Chest, Lateral Decubitus Position

As compared with the condition of the anesthetized, closed-chested patient in the LDP, opening the chest wall and pleural space alone does not ordinarily cause any significant alteration in the partitioning of pulmonary blood flow between the dependent and nondependent lungs; thus, the dependent lung continues to receive relatively more perfusion than the nondependent lung. Opening the chest wall and pleural space, however, does have a significant impact on the distribution of ventilation (which must now be delivered by positive pressure). The change in the distribution of ventilation may result in further V/Q mismatching 233 (Fig. 48–12).

FIGURE 48–12 Schematic depiction of a patient in the lateral decubitus position, comparing the closed-chested anesthetized condition with the open-chested anesthetized and paralyzed condition. Opening the chest increases nondependent lung compliance and reinforces or maintains the larger part of the tidal ventilation going to the nondependent lung. Paralysis also reinforces or maintains the larger part of tidal ventilation going to the nondependent lung because the pressure of the abdominal contents (PAB) pressing against the upper diaphragm is minimal (smaller arrow), and it is therefore easier for positive-pressure ventilation to displace this lesser resisting dome of the diaphragm. V, alveolar volume; P, transpulmonary pressure. (From Benumof604)

If the upper lung is no longer restricted by a chest wall and the total effective compliance of that lung is equal to that of the lung parenchyma alone, it will be relatively free to expand and will consequently be overventilated (and remain underperfused). Conversely, the dependent lung may continue to be relatively noncompliant and poorly ventilated and overperfused. 206 Surgical retraction and compression of the exposed upper lung can provide a partial, although nonphysiologic, solution to this problem in that if expansion of the exposed lung is mechanically or externally restricted ventilation will be diverted to the dependent, better-perfused lung. 208

Anesthetized, Open-Chest, Paralyzed Lateral Decubitus Position

In the open-chested anesthetized patient in the LDP, induction of paralysis alone does not cause any significant alteration in the partitioning of pulmonary blood flow between the dependent and nondependent lungs. Thus, the dependent lung continues to receive relatively more perfusion than the nondependent lung. There are, however, strong theoretical and experimental considerations indicating that paralysis might cause significant changes in the distribution of ventilation between the two lungs under these conditions.

In the supine position and the LDP, the weight of the abdominal contents pressing against the diaphragm is greatest on the dependent part of the diaphragm (posterior lung and lower lung, respectively) and least on the nondependent part of the diaphragm (anterior lung and upper lung, respectively) (see Fig. 48–12). In the awake, spontaneously breathing patient, the normally present active tension in the diaphragm overcomes the weight of the abdominal contents, and the diaphragm moves the most (largest excursion) in the dependent portion and least in the nondependent portion. This is a healthy circumstance because this is another factor that maintains the greatest amount of ventilation where there is the most perfusion (dependent lung) and the least amount of ventilation where there is the least perfusion (nondependent lung). During paralysis and positive-pressure breathing, the passive and flaccid diaphragm is displaced preferentially in the nondependent area, where the resistance to passive diaphragmatic movement by the abdominal contents is least; conversely, the diaphragm is displaced minimally in the dependent portion where the resistance to passive diaphragmatic movement by the abdominal contents is greatest. 234 This is an unhealthy circumstance because the greatest amount of ventilation may occur where there is the least perfusion (nondependent lung), and the least amount of ventilation may occur where there is the most perfusion (dependent lung). 234

Summary of Physiology of the Lateral Decubitus Position and the Open Chest

In summary (Fig. 48–13), the preceding section has developed the concept that the anesthetized, paralyzed patient in the LDP with an open chest may have a considerable V/Q mismatch, consisting of greater ventilation but less perfusion to the nondependent lung and less ventilation but more perfusion to the dependent lung. The blood flow distribution is mainly and simply determined by gravitational effects. The relatively good ventilation of the upper lung is caused in part by the open-chest and paralyzed conditions. The relatively poor ventilation of the dependent lung is caused in part by the loss of dependent lung volume with general anesthesia and by compression of the dependent lung by the mediastinum, abdominal contents, and suboptimal positioning effects. In addition, poor mucociliary clearance and absorption atelectasis with an increased FIO2 may cause further dependent-lung volume loss. Indeed, on rare occasion

the dependent lung may be massively atelectatic and edematous. 235 Consequently, two-lung ventilation under these circumstances may result in an increased alveolar-arterial oxygen tension difference [P(A-a)O2] and less than optimal oxygenation.
FIGURE 48–13 Schematic summary of ventilation-perfusion relationships in the anesthetized patient in the lateral decubitus position who has an open chest and is paralyzed and suboptimally positioned. The nondependent lung is well ventilated (as indicated by the large dashed lines) but poorly perfused (small perfusion vessel); the dependent lung is poorly ventilated (small dashed lines) but well perfused (large perfusion vessel). In addition, the dependent lung may also develop an atelectatic shunt compartment (indicated on the left side of the lower lung) because of the circumferential compression of this lung. (See text for detailed explanation.) PAB, pressure of the abdominal contents. (Modified from Benumof604)

A physiologic solution to the adverse effects of anesthesia and surgery in the LDP on the distribution of ventilation and perfusion during two-lung ventilation would be selective application of PEEP to the dependent lung (via a double-lumen endotracheal tube). 236 Selective PEEP to the lower lung should increase the ventilation to this lung by moving it up to a steeper, more favorable portion of the lung pressure-volume curve. Indeed, this has been done with reasonably good success. 236, 237 A series of 22 mechanically ventilated patients (both lungs) undergoing thoracotomy in the LDP was divided into two groups. 237 Group I patients had 10 cm H2 O of PEEP applied to the dependent lung while zero end-expiratory pressure (ZEEP) was applied to the nondependent lung. Group II (control) patients were intubated with a standard endotracheal tube, and both lungs were ventilated with ZEEP. Selective PEEP to the dependent lung in group I patients resulted in an adequate PaO2 with a lower FIO2 during surgery and a smaller P(A-a)O2 at the end of surgery than when both lungs were ventilated with ZEEP. Thus, even if the selective PEEP to the dependent lung increased dependent lung PVR and diverted some blood flow to the nondependent lung, the diverted blood flow could still participate in gas exchange with the ZEEP-ventilated nondependent lung. 238 However, it should be noted that this technique requires that the nondependent (and operative) lung be ventilated, and this may impede the performance of surgery. The physiology of one-lung ventilation and distribution of perfusion is discussed in the chapter on respiratory physiology.

One-Lung Anesthesia/Ventilation

Indications for Separation of the Two Lungs

There are several absolute and relative indications for separation of the two lungs during thoracic operations or procedures (Table 48–10).

TABLE 48–10. Indications for Separation of the Two Lungs (Double-Lumen Tube Intubation) and/or One-Lung Ventilation

Absolute Indications

Separation of the two lungs for any of the absolute indications discussed here should be considered a lifesaving maneuver because failure to separate the lungs under any of these conditions could result in a life-threatening complication or situation. There are three general absolute indications for separating the lungs (see Table 48–10). First, separation of one lung from the other is absolutely necessary to prevent spillage of pus or blood from an infected (abscessed) lung or bleeding lung, respectively, to a noninvolved lung. Acute contamination of a lung with either blood or pus from the other lung usually results in severe massive (bilateral) atelectasis, pneumonia, and sepsis. Second, there are a number of unilateral lung problems that can prevent adequate ventilation of the noninvolved side. A large bronchopleural or bronchopleural-cutaneous fistula or a surgically opened conducting airway has such a low resistance to gas flow that a tidal inspiration delivered by positive pressure will exit via the low-resistance pathway, and it may become impossible to ventilate the other, more normal, lung adequately. A giant unilateral bulla or cyst may rupture if exposed to positive-pressure ventilation and result in a tension pneumothorax or pneumomediastinum. Very severe or life-threatening hypoxemia due to unilateral lung disease may require differential lung ventilation and PEEP. 239 Finally, positive-pressure ventilation of a lung with a tracheobronchial tree disruption can result in dissection of gas into the pulmonary interstitial space or mediastinum, causing a tension pneumomediastinum. Third, separation of the lungs is absolutely necessary to perform unilateral bronchopulmonary lavage in patients with pulmonary alveolar proteinosis (and rarely, asthma or cystic fibrosis).

Relative Indications

There are a large number of relative indications for separation of the lungs, and they are all for the purpose of facilitating surgical exposure by collapsing the lung in the operative hemithorax. These relative indications can be divided into high-priority and low-priority categories (see Table 48–10). Of the relative indications, repair of a thoracic aortic aneurysm usually has the highest priority because it may require exposure of the thoracic aorta as it runs the entire length of the left hemithorax. A pneumonectomy, especially if performed through a median sternotomy, 240 is greatly aided by the wide exposure of the lung hilum that is afforded by collapse of the operative lung. Similarly, an upper lobectomy, which is technically the most difficult lobectomy, and many mediastinal exposures may be made much easier by eliminating ventilation to the lung on the side of the procedure. Examination of the pleural space (thoracoscopy) and pulmonary resections through a thoracoscope are considerably aided by collapse of the ipsilateral lung. The surgical items in the medium-priority category do not routinely require collapse of the lung on the operative side but still significantly aid surgical exposure and eliminate the need for the surgeon to handle (retract, compress, pack away) the operative lung. Severe intraoperative retraction of the lung on the operated side can traumatize the operative lung and impair gas exchange both intraoperatively 241, 242 and postoperatively. 243, 244 The lower-priority items consist of middle and lower lobectomies, less extensive pulmonary resections, thoracic spinal procedures that are approached anteriorly through the chest, and esophageal surgery. However, even relatively small operations such as wedge and segmental resections benefit by double-lumen tube insertion because of the ability to alternate easily and quickly between lung collapse and inflation, which is sometimes required to better visualize lung morphology and facilitate identification and separation of planes and fissures. Additionally, the separation of the lungs after removal of totally occluding and predominantly unilateral chronic pulmonary emboli (postcardiopulmonary bypass) can be very helpful because of the possibility of massive transudation of hemorrhagic fluid across the alveolar capillary membrane in the region of the lung supplied by the previously occluded vessel (reperfusion of a previously and chronically nonperfused vascular bed). Should significant and predominantly unilateral pulmonary edema occur following thromboembolectomy with cardiopulmonary bypass, the patient should be returned to cardiopulmonary bypass and a double-lumen endotracheal tube should be inserted so that differential lung ventilation may be used. Finally, significant hypoxemia due to unilateral lung disease may be more easily treated by differential lung ventilation and PEEP. 239

Techniques of Lung Separation

In general, three types of devices are available for providing one-lung ventilation during anesthesia: double-lumen endotracheal tubes (DLT), bronchial blockers, and endobronchial tubes. DLTs have come to be considered the lung separation technique of choice for the majority of thoracic surgery cases and are discussed later in detail. Bronchial blockade with the Univent tube and with independently passed bronchial blockers (Fogarty embolectomy catheter) in adults is greatly increasing in use and is also described at length below. Endobronchial tubes are not often used today and are only briefly described at the end of the chapter. The primary reason that DLTs are favored over bronchial blockers or endobronchial tubes for lung separation is that they are more versatile than the other two devices. The most important DLT function not available with a bronchial blocker is independent bilateral suctioning. In addition, it is easier to apply CPAP to the nonventilated operative lung with a DLT than with a bronchial blocker, and it is also easier to rapidly convert from two-lung to one-lung ventilation and vice versa with a DLT than with a bronchial blocker. Endobronchial tubes are very limited in function and only allow one-lung ventilation.

There are two firm disadvantages (contraindications) to the use of a DLT as compared with a bronchial blocker. First, very distorted tracheobronchial tree anatomy, including exophytic and stenotic lesions, as well as tortuosity, may preclude successful correct placement or positioning of a DLT. Second, changing from a DLT to a single-lumen tube during or at the end of an operation can be expected to be a difficult and/or risky procedure on occasion. Such a situation might occur in a patient with a relatively difficult airway before operation who undergoes a long operation requiring considerable intravenous fluids; one would expect the airway to be edematous and thus a postoperative tube change more hazardous in that setting.

There are two relatively minor disadvantages to DLTs, both related to the fact that the lumina of a DLT may be narrow. First, suctioning may be more difficult down a narrow lumen, but this is usually not a problem with the new disposable Robertshaw type of DLTs which have nonadhering suction catheters that slide easily down the lumina. Second, although airway resistance may be increased with a narrow lumen, the increased resistance can be easily overcome by positive-pressure ventilation. 245

Double-Lumen Endotracheal Tubes

Commonly Used Double-Lumen Endotracheal Tubes
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A DLT is essentially two catheters bonded together side by side, with each lumen intended to ventilate one of the two lungs. DLTs are made as left- and right-sided tubes. With a left-sided tube, the left lung catheter is placed into the left mainstem bronchus, whereas the right lung catheter ends in the trachea; therefore, for a left-sided tube, the left lung catheter is longer than the right lung catheter (Fig. 48–14). With a right-sided tube the right lung catheter is placed into the right mainstem bronchus, whereas the left lung catheter ends in the trachea; therefore, for a right-sided tube, the right lung catheter is longer than the left lung catheter (see Fig. 48–14). All DLTs have a proximal cuff for the trachea and a distal cuff for a mainstem bronchus; the endobronchial cuff causes separation and sealing off of the lungs from each other, and the tracheal cuff causes separation and sealing off of the lungs from the environment. The part of the right lung catheter of the right-sided DLT that is in the right mainstem bronchus must be slotted to allow ventilation of the right upper lobe (see Fig. 48–14) because the right mainstem bronchus is too short to accommodate both the right lumen tip and the right endobronchial cuff. All double-lumen endotracheal tubes have two curves that lie in planes approximately 90 degrees apart from one another. The distal curve is designed to facilitate placement of the distal catheter tip into the appropriate mainstem bronchus, and the proximal curve is designed to approximate the oropharyngolaryngeal curve.
FIGURE 48–14 Schematic diagram depicting the essential features and parts of left-sided and right-sided double-lumen endotracheal tubes. RUL, right upper lobe; LUL, left upper lobe. (From Benumof604)

The DLTs that are now used for lung separation and one-lung ventilation are the Carlens and the Robertshaw. The Robertshaw type of tube is by far the more commonly used, and the disposable polyvinylchloride (PVC) Robertshaw tube has significantly replaced the red rubber Robertshaw tube (the former is easier to pass, is positioned more quickly, and causes less mucosal damage). 246 Consequently, the modern PVC tube will be described in great detail.

The left-sided Carlens tube (Fig. 48–15) was the first DLT used for one-lung ventilation. 247 The tube has a carinal hook to aid in its proper placement and minimize tube movement after placement. Potential problems with carinal hooks include increased difficulty (more rotations) and laryngeal trauma during intubation, amputation of the hook during or after passage, malpositioning of the tube caused by the hook, and physical interference during pneumonectomy. 248 Therefore, some anesthesiologists prefer to use the tube with the hook removed. The tube is available in four sizes: 41, 39, 37, and 35 French (which correspond to an internal diameter of each lumen of approximately 6.5, 6.0, 5.5, and 5.0 mm, respectively). The cross-sectional shape of each lumen is oval, and this accounts for the occasional difficulty in passing a suction catheter down the lumen.
FIGURE 48–15 (A) Sketch of the red rubber (nondisposable) Carlens double-lumen endotracheal tube. (B) Close-up of the placement of the red rubber Carlens double-lumen endotracheal tube at the carina. Note that the left endobronchial lumen and carinal hook straddle the carina. (From Benumof604)

The original Robertshaw DLT, introduced in 1962, was made as a reusable red rubber tube (Fig. 48–16). 249 This tube was designed to provide the largest possible lumen to decrease airway resistance and facilitate removal of secretions. The lumina are D-shaped and lie side by side, like those of the Carlens tube, but are larger in size. As with the other DLTs, it has two curves (in planes approximately 90 degrees apart), which facilitate intubation and proper endobronchial placement. Both a right- and a left-sided tube are available, and the absence of a carinal hook allows for easier tracheal intubation and perhaps correct positioning. The right-sided tube has a slotted endobronchial cuff to effect ventilation of the right upper lobe. The right upper lobe ventilation slot is relatively long, which facilitates ventilation of this lobe. However, the endobronchial cuff has an additional area of inflation on the nonslotted side above the slot to effect a more reliable seal (see Fig. 48–16A). On the slotted side, inflation of the endobronchial cuff is restricted. However, the right endobronchial cuff design forces the right upper lobe slot to lie flat against the right upper lobe orifice, and if this slot is not perfectly aligned with the right upper lobe orifice, the ventilation slot will be

blocked (obstructed) by the right mainstem bronchial wall (and vice versa). Nevertheless, because of its many good features, the original Robertshaw DLT rapidly gained wide popularity. 250
FIGURE 48–16 (A) Sketch of the left-sided red rubber Robertshaw double-lumen endotracheal tube. (B) Close-up of the placement of the left-sided Robertshaw double-lumen endotracheal tube at the carina. (C) Sketch of the right-sided Robertshaw double-lumen endotracheal tube. (D) Close-up of the placement of the right-sided Robertshaw double-lumen endotracheal tube at the carina. (From Benumof604)

The Robertshaw type of tube is now made of a clear nontoxic tissue-implantable plastic (denoted by the marking Z-79) and is disposable (see Fig. 48–14). The tubes are made in sizes 41, 39, 37, 35, 28, and 26 French (internal diameter of each lumen is approximately 6.5, 6.0, 5.5, 5.0, 4.5, or 4.0 mm, respectively). The 26 and 28 French tubes are available only as left-sided models. These tubes are relatively easy to insert and have appropriate end-of-lumen and cuff arrangements that minimize lobar obstruction. The endobronchial cuff is colored brilliant blue, which is an important recognition feature when using a fiberoptic bronchoscope. The ends of both lumina have a black radiopaque line, which is an essential recognition marker when viewing a chest radiograph. The tubes have high-volume, low-pressure tracheal and endobronchial cuffs. The slanted doughnut-shaped endobronchial cuff on the Bronchocath model right-sided DLT allows the right upper lobe ventilation slot to ride off (away from) the right upper lobe orifice, which minimizes the chance of right upper lobe obstruction by the tube. The clear tubing is helpful because it permits continuous observation of the tidal movement of respiratory moisture as well as observation of secretions from each lung. The tubes are packaged with malleable stylets and are relatively easy to insert and position. These tubes have large internal to external diameter ratios and therefore are relatively easy to suction through, and they are packaged with their own nonadhering suction catheters. The large internal to external diameter ratio also provides a relatively low resistance to ventilation. For all these reasons, these disposable tubes are now considered the DLTs of choice by most anesthesiologists. Several companies are currently manufacturing them. Reviews of other DLTs are available. 251

A left-sided DLT should be used for right thoracotomies requiring collapse of the right lung and ventilation of the left lung (Fig. 48–17). A left- or right-sided tube may be used for left thoracotomies requiring collapse of the left lung and ventilation of the right lung (see Fig. 48–17). However, because the right upper lobe ventilation slot of a right-sided tube has to be closely apposed to the right upper lobe orifice to allow unobstructed right upper lobe ventilation and since there is considerable anatomic variation in the exact position of the right upper lobe orifice and therefore in the length of the right mainstem bronchus (in fact, it is well known that an anomalous right upper lobe can take off from the trachea), use of a right-sided tube for left lung collapse introduces the risk of inadequate right upper lobe ventilation. For this reason, a left-sided tube is preferable for most cases requiring one-lung ventilation. If clamping of the left mainstem bronchus is necessary, the tube can be withdrawn at that time into the trachea and then used in the same manner as a single-lumen endotracheal tube (ventilation of the right lung with both lumina) (see Fig. 48–17). Contraindications to use of a left-sided DLT are carinal and proximal left mainstem bronchial lesions that could be traumatized by the passage of a left-sided tube. These lesions include strictures, endoluminal tumors, tracheobronchial disruptions, compression of the airway by an external mass, and tenting of the left mainstem bronchus so that the angle of the take-off from the trachea is approximately 90 degrees. The largest size of tube that can comfortably pass the glottis should be used, as a relatively small DLT may require excessive cuff volume for endobronchial cuff seal and may cause difficulty with suctioning secretions or ventilating the patient. In general, as height and weight increase, the appropriate DLT size (as defined above) increases, although height is much more important than weight. 252
FIGURE 48–17 Use of left-sided and right-sided double-lumen endotracheal tubes for left and right lung surgery (as indicated by the clamp). (A) When surgery is performed on the right lung, a left-sided double-lumen endotracheal tube should be used. (B) When surgery is performed on the left lung, a right-sided double-lumen endotracheal tube can be used. However, because of uncertainty about the alignment of the right upper lobe ventilation slot with the right upper lobe orifice, a left-sided double-lumen endotracheal tube can also be used for left lung surgery. (C) If the left lung surgery requires a clamp to be placed high on the left main stem bronchus, the left endobronchial cuff should be deflated, the left-sided double-lumen endotracheal tube pulled back into the trachea, and the right lung ventilated through both the lumina (use the double-lumen endotracheal tube as a singlelumen tube). (From Benumof604)

In summary, the plastic disposable Robertshaw-type tubes are by far the most commonly used DLTs. Because a right-sided tube incurs the risk of inadequate right upper lobe ventilation, left-sided tubes are used far more often. Consequently, the rest of this chapter emphasizes the insertion and precise positioning of the left-sided Robertshaw-type DLT.

Conventional Double-Lumen Tube Intubation Procedure
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Before intubation with a double-lumen endotracheal tube, both cuffs and the lumen connections are checked. A 3-mL syringe with stopcock should be placed on the end of the bronchial cuff pilot tube, because proper bronchial cuff inflation rarely requires more than 1 to 2 mL of air; a 5- or 10-mL syringe with stopcock should also be placed on the tracheal cuff pilot tube. Because the high-volume, low-pressure cuffs can be easily torn by teeth, the distal tube is coated with a lubricating ointment (preferably containing a local anesthetic) to minimize this possibility. If a less than optimal view of the larynx is anticipated, the stylet that is packaged with the tube is lubricated, inserted into the left lumen, and appropriately curved. The patient is then anesthetized and paralyzed as described previously. A curved open-phalanged blade (MacIntosh) is usually preferred for laryngoscopy, because it approximates the curvature of the tube and therefore provides the largest possible area through which to pass the tube. However, a straight (Miller) blade may be a better choice in patients with overriding upper teeth or an excessively anterior larynx.

Double-lumen endotracheal tubes with carinal hooks are first inserted through the vocal cords with the hook facing posteriorly. When the tip of the tube has passed the vocal cords, the tube is rotated 180 degrees so that the hook passes anteriorly through the glottis. If this maneuver is not done, then one can damage the vocal apparatus by dislocating an arytenoid cartilage should the hook snare or hang up on the arytenoid cartilage. After the tube tip and hook pass the larynx, the tube is rotated 90 degrees so that the tube tip enters the appropriate bronchus.

The Robertshaw-type DLT is passed with the distal curvature initially concave anteriorly (Fig. 48–18A). After the tube tip passes the larynx and while anterior force on the laryngoscope is continued, the stylet (if used) is removed, and the tube is carefully rotated 90 degrees (so that the distal curve is now concave toward the appropriate side and the proximal curve is concave anteriorly) to allow endobronchial intubation on the appropriate side (Fig. 48–18B). Continued anterior force by the laryngoscope during tube rotation prevents hypopharyngeal structures from falling in around the tube and interfering with a free 90-degree distal tube tip rotation. Failure to obtain a near 90-degree rotation of the distal tube tip while the proximal end rotates 90 degrees will cause either a kink or a twist in the shaft of the tube and/or prevent the distal end of the lumen from lying free in the mainstem bronchus (i.e., not up against the bronchial wall). After rotation, the tube is advanced until most of it is inserted 252 (Fig. 48–18C). When the proper depth of insertion has been achieved (defined as when the cephalad surface of the bronchial cuff is immediately below the carinal bifurcation), the average depth of insertion for both male and female patients 170 cm tall is 29 cm, and for each 10-cm increase or decrease in height, average placement depth is increased or decreased by 1 cm. 252 The correlation between depth of insertion and height is highly significant ($P<.0001$) for both male and female patients. Nevertheless, it should be understood that the depth of DLT insertion at any given height is still normally distributed, and correct DLT position should always be confirmed fiberoptically after initial placement. DLTs may also be passed successfully via tracheostomy, although it should be remembered that the tracheal cuff may be at the tracheal stoma or lie partly outside the trachea in this situation. 253, 254 Because of this, one may prefer to use a specially manufactured (i.e., short) nondisposable double-lumen endotracheal tube for these particular patients. 255

FIGURE 48–18 Schematic diagram depicting the passage of the left-sided double-lumen endotracheal tube in a supine patient. (A) The tube is held with the distal curvature concave anteriorly and the proximal curve concave to the right and in a plane parallel to the floor. The tube is then inserted through the vocal cords until the bronchial cuff passes the vocal cords. The stylet is then removed. (B) The tube is rotated 90 degrees counterclockwise so that the distal curvature is concave anteriorly and the proximal curvature is concave to the left and in a plane parallel to the floor. (C) The tube is inserted until either a mild resistance to further passage is encountered or the end of the common molding of the two lumina is at the teeth. Both cuffs are then inflated, and both lungs are ventilated. Finally, one side is clamped while the other side is ventilated and vice versa. (See text for further explanation.) (From Benumof604)

Once the tube tip is thought to be in an endobronchial position, the following checklist is used to ensure proper functioning of the tube. Inflate the tracheal and endobronchial cuffs until moderate tension is palpated in the external pilot balloons (the endobronchial cuff should not require more than 1–2 mL of air); deliver

several positive-pressure ventilations, auscultate, and observe the chest bilaterally to determine that the trachea rather than the esophagus has been intubated and that both lungs are being ventilated (see Fig. 48–18C). In addition to seeing the tube go through the vocal cords, check correct intubation position by feeling and observing the anesthesia reservoir bag to make sure it has the appropriate compliance and movement while maintaining normal pulse oximetry and ETCO2 values, and perhaps palpating the tracheal cuff in the neck. If only unilateral breath sounds or chest movement is present, it is likely that both lumina of the tube have entered a mainstem bronchus (if both lumina enter the left main stem bronchus, the findings may mimic an esophageal intubation, and vice versa). In this situation, quickly deflate the cuffs, withdraw the tube 1 to 2 cm at a time, inflate the cuffs, and reassess ventilation until bilateral breath sounds are heard. If bilateral breath sounds are not heard and the tube has been withdrawn a significant amount, the entire procedure must be repeated, beginning with establishing the airway and oxygen ventilation via mask, laryngoscopy, and reinsertion of the DLT through the vocal cords. If bilateral breath sounds are present, then one side is clamped and breath sounds and chest movement should disappear on the ipsilateral side and remain on the contralateral side. Breath sounds should be at least as audible anteriorly, at the apex of the lung, as they are along the lateral chest. If they are not heard clearly at the lung apex, it is likely that the tube has been advanced too far distally and that the upper lobe on that side is not being ventilated adequately. In that case the tube must be pulled back 1 cm at a time until apical breath sounds are clearly heard. In addition, when the lumen cap is opened proximal to the clamp, there should be no leakage of air out of that lumen, indicating that the bronchial cuff has effected a proper seal. Next, the clamped side should be unclamped and the lumen cap replaced, and the breath sounds and chest movement should reappear on that side. During unilateral clamping, the breath sounds on the ventilated side should be compared with and calibrated against unilateral chest wall movements and the inspiratory disappearance and expiratory appearance of respiratory gas moisture in the clear tubing of the ventilated side. In addition, the compliance of the lung should be gauged by using hand ventilation. The unilateral clamping and unclamping and removal of the lumen cap proximal to the clamp should then be repeated on the opposite side to ensure adequate lung separation and cuff seal.

In summary, when DLT position is correct, the breath sounds are normal and follow the expected unilateral pattern with unilateral clamping, the chest rises and falls in accordance with the breath sounds, the ventilated lung feels reasonably compliant, no leaks are present, and respiratory gas moisture appears and disappears with each tidal ventilation. Conversely, when the DLT is malpositioned, any or all of the following may occur: the breath sounds may be poor and correlate poorly with unilateral clamping, the chest movements may not follow the expected pattern, the ventilated lung may feel noncompliant, leaks may be present, or the respiratory gas moisture in the clear tubing may be relatively stationary. It is very important to realize, however, that even if the DLT is thought to be properly positioned by clinical signs, subsequent fiberoptic bronchoscopy may reveal an incidence of malpositioning that ranges from 38 to 78 percent. 256, 257

When it is believed, on the basis of clinical signs, that the DLT is malpositioned, it is theoretically possible to diagnose the malposition of the tube more precisely by a combination of several unilateral clamping, chest auscultation, and left endobronchial cuff inflation-deflation maneuvers (Fig. 48–19). With reference to a left-sided DLT, there are three possible gross malpositions: in too far on the left (both lumina in left mainstem bronchus), out too far (both lumina in the trachea), and in or down the right mainstem bronchus (at least the left lumen in the right mainstem bronchus). When the right (tracheal) side is clamped and the tube is in too far on the left side, breath sounds are heard only on the left side. When the tube is out too far and the right side is clamped, breath sounds are heard bilaterally. When the tube is in or down the right side and the right side is clamped, breath sounds are heard only on the right side. When the left side is clamped and the left endobronchial cuff is inflated, the right lumen is blocked by the left cuff in all three malpositions. Consequently, with the left side clamped and the left cuff inflated, no or very diminished breath sounds are heard bilaterally in all three of the malpositions. When the left side is clamped and the left cuff is deflated, so that the right lumen is no longer blocked by the left cuff, breath sounds are heard only on the left side when the tube is in too far on the left, bilaterally when the tube is out too far, and only on the right side when the tube is in the right side. The left cuff inflation and deflation findings provide the key diagnostic data because they essentially define the position of the right tracheal lumen by blocking and unblocking it with the left cuff. FIGURE 48–19 There are three major malpositions (involving a whole lung) of a left-sided double-lumen endotracheal tube. The tube can be in too far on the left (both lumina are in the left main stem bronchus), out too far (both lumina are in the trachea), or down the right main stem bronchus (at least the left lumen is in the right main stem bronchus). In each of these three malpositions the left cuff, when fully inflated, can completely block the right lumen. Inflation and deflation of the left cuff while the left lumen is clamped create

a breath sound differential diagnosis of tube malposition. (See text for full explanation.) L, left; R, right; ?, decreased. (From Benumof604)

There are, however, several situations in which these unilateral clamping, auscultation, and cuff inflation and deflation maneuvers for determining the integrity of lung separation are either unreliable or impossible. First, and most importantly, when the patient is in the LDP, has had a skin preparation, and is draped, access to the chest wall is impossible, and the anesthesiologist cannot listen to the chest. Second, the presence of unilateral or bilateral lung disease, either preexisting before anesthesia and surgery or anesthesia-induced, may markedly obscure the crispness of the chest auscultation end points. Third, the diagnosis of exactly where the DLT is located may be confused when the tube is just slightly malpositioned. Fourth, the tube may have moved as a result of some event, such as coughing, head flexion or extension while turning into the LDP, or tracheal manipulation and hilar retraction by the surgeon. Finally, some combination of these factors may culminate in uncertainty about where the DLT has located. The solution to any uncertainty about the exact position of the DLT is to determine the position by fiberoptic bronchoscopy.

Use of Fiberoptic Bronchoscope to Determine Precise Double-Lumen Tube Position
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As noted, even when a DLT is thought to be in proper position on the basis of clinical signs, subsequent fiberoptic bronchoscopy will reveal an incidence of malpositioning as high as 78 percent. 256 Indeed, when the position of the DLT is checked only by clinical signs, in up to 25 percent of cases there may be intraoperative problems with either deflating the nondependent lung, ventilating the dependent lung, or completely separating the two lungs. 256, 258 Given the high incidence of malpositioned DLTs when DLT position is determined by only auscultation (i.e., “blindly”) and the potentially serious consequences associated with a malpositioned DLT, it is only a matter of simple common sense to routinely use a fiberoptic bronchoscope to easily, quickly, and precisely determine the position of the DLT.

The exact position of a left-sided DLT can be ascertained at any time, in less than a minute, by simply passing a pediatric-size fiberoptic bronchoscope through the tracheal lumen of the DLT. It is rarely necessary also to have to pass the fiberoptic bronchoscope down the left endobronchial lumen. With reference to a left-sided DLT, looking down the right (tracheal) lumen the endoscopist should have a clear straight-ahead view of the tracheal carina, the left lumen going off to the left, and the upper surface of the blue left endobronchial balloon just below the tracheal carina (Fig. 48–20). Because the distance between the right and left lumen tips for the clear plastic tube (69 mm) is longer than the length of the left mainstem bronchus (average 50 mm) in a typical patient and if the blue upper surface of the endobronchial balloon is not visible, it is possible for the right lumen to be above the tracheal carina while the left lumen tip obstructs the left upper lobe. 259 However, no matter which manufactured tube or size of tube is used and no matter how long or short the left mainstem bronchus is (within the range of extremes observed in extensive studies), 259 when the upper surface of the left endobronchial balloon is just below the tracheal carina, it is not possible for the left lumen tip to obstruct the left upper lobe or for the right (tracheal) lumen to be near a mainstem bronchus. It is important that the volume of air used to fill the left endobronchial cuff not cause the endobronchial cuff to herniate over the tracheal carina or cause the tracheal carina to deviate to the right (see Fig. 48–20); both cuff herniation and carinal deviation can be readily appreciated by looking down the tracheal lumen. Looking down the left lumen (as is sometimes done when inserting a left-sided DLT with a fiberoptic bronchoscope [see the section immediately below] and in all cases of bronchopulmonary lavage where perfect tube position and tight cuff seal are extremely critical), the endoscopist should see a very slight narrowing of the left lumen (due to endobronchial cuff pressure) as well as the bronchial carina distal to the end of the tube (see Fig. 48–20). The endoscopist should not see excessive left luminal narrowing (due to excessive left cuff pressure) (see Fig. 48–20). Thus, aside from gross malposition, important undesirable findings on endoscopy are related to excessive left cuff inflation and pressure and consist of cuff herniation over the tracheal carina, carinal deviation to the right (both of which may block the right mainstem bronchial orifice and impair right lung ventilation), and excessive left lumen constriction (invagination), which may impair left lung ventilation 260 (see Fig. 48–20). In addition, when an inappropriately undersized tube is used, the large endobronchial cuff volume required for endobronchial cuff seal tends to force the entire DLT cephalad, making a functional bronchial seal more difficult. 261

FIGURE 48–20 This schematic diagram depicts the complete fiberoptic bronchoscopy picture of left-sided double-lumen endotracheal tubes (both the desired view and the view to be avoided from both of the lumina). (A) When the bronchoscope is passed down the left lumen of the left-sided tube, the endoscopist should see a very slight left luminal narrowing and a clear straight-ahead view of the bronchial carina off in the distance. Excessive left luminal narrowing should be avoided. (B) When the bronchoscope is passed down the right lumen of the left-sided tube, the endoscopist should see a clear straight-ahead view of the tracheal carina and the upper surface of the blue left endobronchial cuff just below the tracheal carina. Excessive pressure in the endobronchial cuff, as manifested by tracheal carinal deviation to the right and herniation of the endobronchial cuff over the carina, should be avoided. (From Benumof604)

With reference to a right-sided DLT, looking down the left (tracheal) lumen, the endoscopist should see a clear straight-ahead view of the tracheal carina and the right lumen going off to the right (Fig. 48–21A). The upper surface of the right endobronchial balloon may not be visible below the tracheal carina. Looking down the right lumen, the endoscopist should see a very slight narrowing of the right lumen as well as the right middle-lower lobe bronchial carina distal to the end of the tube. Most importantly, the endoscopist should locate the right upper lobe ventilation slot and be able to look directly into the right upper lobe orifice through the right upper lobe ventilation slot by simply flexing the tip of the fiberoptic bronchoscope superiorly and laterally (Fig. 48–21B). There should be no overriding of the right upper lobe ventilation slot on the bronchial mucosa, and the bronchial mucosa should not be covering any of the right upper lobe ventilation slot.
FIGURE 48–21 This schematic diagram portrays use of a fiberoptic bronchoscope to determine precise right-sided double-lumen tube position. (A) When the fiberoptic bronchoscope is passed down the left (tracheal) lumen, the endoscopist should see a clear straight-ahead view of the tracheal carina and the right lumen going off into the right main stem bronchus. (B) When the fiberoptic bronchoscope is passed down the right (bronchial) lumen, the endoscopist should see the bronchial carina off in the distance; when the fiberoptic bronchoscope is flexed cephalad and passed through the right upper lobe ventilation slot, the right upper lobe bronchial orifice should be visualized. (From Benumof604)

In our experience, in 8 to 9 out of 10 cases, the clinical signs (breath sounds, chest movements, compliance of the lung[s], movement of respiratory gas moisture) indicate that the lungs are apparently clearly and without doubt completely separated when the DLT is first inserted with the patient in the supine position. However, in view of the finding that up to 78 percent of DLTs are malpositioned to some extent in the supine position, even though clinical signs indicate no problem, 256 it is strongly advisable to check the position of the tube with a fiberoptic bronchoscope in the supine position (especially in view of the fact that the procedure takes less than 1 minute). Even if no problem is identified, the procedure still allows the endoscopist to become familiar with the patient's anatomy and facilitates the more important endoscopy after turning the patient into the LDP. In approximately 1 to 2 out of 10 cases, there is a definite doubt about tube location in the supine position, and in these patients the fiberoptic bronchoscope is always used to correct the DLT malposition. The fiberoptic bronchoscope is again used to confirm DLT position after the patient has been turned into the LDP. Of course, a determined effort is made to prevent dislodgement of the tube during turning by holding on to the tube at the level of the incisors and by keeping the head absolutely immobile in a neutral or slightly flexed position. Head extension can cause movement of the tube in a cephalad direction, which may result in bronchial decannulation; head flexion can cause movement of the tube in a caudad direction, which may result in an upper lobe obstruction or in both luminas being in a mainstem bronchus (see next section). 262, 263 Finally, the fiberoptic bronchoscope is used whenever during the case there is a question about DLT position. This is not an infrequent occurrence and is usually caused by surgical manipulation and traction on the hilum, carina, or trachea.

Use of Fiberoptic Bronchoscope to Insert the Double-Lumen Tube
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The insertion of the bronchial lumen of a DLT into the appropriate mainstem bronchus may be aided by the use of a fiberoptic bronchoscope. This may be especially helpful if anatomic variation or a pathologic condition has caused carinal distortion. The DLT is first placed in the trachea in a conventional manner (laryngoscopy, manual tube insertion) until the tracheal cuff just passes the vocal cords, the tracheal cuff is inflated, and both lungs are ventilated through both lumina (the DLT should be used as if it were a single-lumen tube). A pediatric-sized fiberoptic bronchoscope can then be inserted into the bronchial lumen through

a self-sealing diaphragm in the elbow connector to the bronchial lumen (which permits continued positive-pressure ventilation through that lumen around the fiberoptic bronchoscope) and passed into the appropriate mainstem bronchus. The tracheal cuff is then deflated and the bronchial lumen is passed over the fiberoptic bronchoscope stylet into the appropriate mainstem bronchus. The fiberoptic bronchoscope is then withdrawn from the bronchial lumen and passed down the tracheal lumen to determine the precise DLT position (see the preceding section).

Alternatively, once the DLT is in the trachea, the fiberoptic bronchoscope can be inserted into the tracheal lumen and passed just proximal to the tracheal carina. While the carina and the two mainstem bronchial orifices are in view, the DLT can be advanced and the degree of lateral rotation adjusted so that the appropriate lumen enters the appropriate mainstem bronchus. Final precise positioning (see the preceding section) can be done with the fiberoptic bronchoscope remaining in the tracheal lumen if a left-sided tube is used. If a right-sided tube is used, precise positioning must be confirmed with the bronchoscope passed through the bronchial lumen.

Relationship of Fiberoptic Bronchoscope Size to Double-Lumen Tube Size

The clear plastic disposable right and left double-lumen endotracheal tubes are manufactured in four sizes: 35, 37, 39, and 41 French. In addition, 26 and 28 French–sized tubes are available only as left-sided models. A 5.6-mm outside diameter diagnostic fiberoptic bronchoscope will not pass down the lumina of any size DLT. A 4.9-mm outside diameter fiberoptic bronchoscope passes easily through the lumina of the 41 French tube and moderately easily with lubrication through the 39 French tube; it causes a tight fit that needs a liberal amount of lubrication and a strong pushing force to pass through the lumen of the 37 French tube and does not pass through the lumen of the 35 French tube. A silicon-based fluid (such as that made by the American Cystoscope Co.) is the best lubricant for a fiberoptic bronchoscope because it does not dry out or crust and does not interfere with the view even if it coats the tip of the bronchoscope. Fortunately, from the point of view of using a 4.9-mm outside diameter fiberoptic bronchoscope, a 37 French tube or larger can be used in almost all adult females and 39 French tube or larger in almost all adult males. A 3.6- to 4.2-mm outside diameter (pediatric-sized) fiberoptic bronchoscope passes easily through the lumina of all adult-sized DLTs and because the bronchoscope has an increased amount of space, the maneuverability of the tip of the bronchoscope is greatly increased. Therefore, the 3.6- to 4.2-mm outside diameter bronchoscope is obviously the bronchoscope of choice for DLTs. Table 48–11 summarizes these fiberoptic bronchoscope–DLT relationships. Several companies (Olympus, Machida, Pentax) presently manufacture 4.9- and 3.6- to 4.2-mm outside diameter fiberoptic bronchoscopes that are of adequate length and have a suction channel.

TABLE 48–11. Relationship of Fiberoptic Bronchoscope Size to Adult Double-Lumen Endotracheal Tube Size

Use of Chest Radiograph to Determine Double-Lumen Tube Position

The chest radiograph can be used to determine DLT position. The chest radiograph may be more useful than conventional unilateral auscultation and clamping in some patients, but it is always less precise than fiberoptic bronchoscopy. To use the chest radiograph, the DLT must have radiopaque markers at the end of the right and left lumina. The key to discerning DLT position on the chest radiograph is seeing where the marker at the end of the tracheal lumen is in relation to the tracheal carina and whether the endobronchial lumen is located in the correct main stem bronchus. The end of the tracheal lumen marker must be above the tracheal carina; however, this does not guarantee correct position because this technique may not reveal a subtle obstruction of an upper lobe. If the tracheal carina cannot be seen (as sometimes happens with portable anteroposterior film), the chest radiograph method of determining DLT position is not usable. Furthermore, the chest radiograph method is time-consuming (for film transport and film development), costly, and awkward to perform and may dislodge the tube (the cassettes are often difficult to place under the operating room table and require moving the patient).

Other Methods to Determine Double-Lumen Tube Position

Three other methods may help to determine the position of a DLT. First, comparison of capnography (waveform and end-tidal CO_2 pressure [$PETCO_2$] value) from each lumen may reveal a marked discrepancy. For example, with all other conditions equal, one lung may be very poorly ventilated in relation

to the other lung (high PETCO2), indicating obstruction to that lung; one lung may be very overventilated in relation to the other lung (low PETCO2), perhaps indicating ventilation of just a lobe of that lung; or the capnogram from one lung may have a much steeper slope to the alveolar plateau, indicating expiratory obstruction. 264, 265 Second, continuous spirometric data (Datex Capnomac Ultima) from both lungs and from each lung separately, such as pressure-volume or flow-volume loops, may be displayed and compared with a control loop that is stored in memory. 266 Third, the surgeon may be able to palpate the position of the DLT from within the chest and may be able to redirect or assist in changing its position (by deflecting the DLT away from the wrong lung, etc.). 267

Quantitative Determination of Cuff Seal Pressure Hold

The use of fiberoptic bronchoscopy to determine DLT position does not provide evidence or a guarantee that the lungs are functionally separated (i.e., against a fluid and/or air pressure gradient). There are times, such as during the performance of unilateral pulmonary lavage, when the anesthesiologist must be absolutely certain that functional separation has been achieved. Complete separation of the lungs by the left endobronchial cuff can be demonstrated in a left-sided tube by clamping the connecting tube to the right lung proximal to the right suction port and attaching a small tube (i.e., intravenous extension tubing) to the open right suction port (by appropriate adaptors) (Fig. 48–22). The free end of this tube is submerged in a beaker of water. When the left lung is statically inflated to any pressure considered necessary and the left endobronchial cuff is not sealed, air will enter the left lung and will also escape from around the unsealed left cuff, move up the right lumen to the small connecting tube, and bubble through the beaker of water (Fig. 48–22B). If the left endobronchial cuff is sealed, no bubbles should be observed passing through the beaker of water (Fig. 48–22A). After demonstration of functional lung separation, the right connecting tube is unclamped, the right suction port closed, and ventilation to both lungs resumed. To test for lung separation with the pressure gradient across the endobronchial balloon reversed, the left airway connecting tube is clamped proximal to the left suction port, the left suction port opened to the beaker of water via the small tube, the right lung statically inflated to any desired pressure, and the absence or presence of air bubbles in the beaker of water noted. It should be remembered that even though the left endobronchial cuff may be adequately sealed, it is possible that during these maneuvers, compression of the nonventilated lung by the ventilated lung may initially cause some small amount of bubbling in the beaker, which will cease with repetitive inflation of the ventilated lung (no bubbles should be seen after several inflations). 251, 260 The absence of airflow from the nonventilated lung suction port is a very simple but sensitive indicator of functional separation of the two lungs.

FIGURE 48–22 Schematic diagram showing the air bubble detection method for checking adequacy of the seal of the left endobronchial cuff of a left-sided double-lumen tube. (A) When the left lung is selectively ventilated or exposed to any desired distending pressure and the left cuff is adequately sealed, no air will escape around the left cuff and out the open right suction port, and thus no bubbles will be observed passing through the beaker of water. (B) When the left lung is ventilated or exposed to any desired distending pressure and the left endobronchial cuff is not adequately sealed, air will escape around the left cuff and out the open right suction port, and thus air bubbles will be observed passing through the beaker of water. (From Benumof604)

Complications of Double-Lumen Endotracheal Tubes

In addition to the impediment to arterial oxygenation that is inherent in the use of DLTs for one-lung anesthesia, the tubes themselves occasionally cause other serious complications. These complications include tracheobronchial tree disruption (with the Carlens tube, 268 the red rubber Robertshaw tube, 269 the red rubber White tube, 270 and the disposable low-pressure cuff plastic tubes 271, 272) , traumatic laryngitis, 248 and suturing of a pulmonary vessel to the DLT. 273 With regard to tracheobronchial tree disruptions, a common thought in the reports cited is that excessive air volume and pressure in the bronchial balloon may be major factors in the genesis of these tears after DLT insertion. Recommendations to minimize tracheobronchial wall damage due to the cuffs include being particularly cautious in the use of DLTs in patients with bronchial wall abnormalities, choosing an appropriately sized clear plastic tube, 246 being certain the tube is not malpositioned, preventing overinflation of the endobronchial cuff, deflating the endobronchial cuff during turning, inflating the endobronchial cuff slowly, inflating the endobronchial cuff with inspired gases if nitrous oxide is used, and preventing the tube from moving during turning (Table 48–12).

TABLE 48–12. Endobronchial Cuff Considerations to Minimize Tracheobronchial Wall Damage (Disruption)

Relative Contraindications to Use of Double-Lumen Endotracheal Tubes

Lung separation by a DLT may be relatively contraindicated in several situations because insertion of the tube is either difficult or dangerous. These situations involve patients who have a full stomach (risk of aspiration); patients who have a lesion (airway stricture, 274 endoluminal tumor) that is present somewhere along the pathway of the DLT and thus could be traumatized; small patients for whom a 35 French tube is too large to fit comfortably through the larynx and for whom a 28 French tube is considered too small; patients whose upper airway anatomy precludes safe insertion of the tube (recessed jaw, prominent teeth, bull neck, anterior larynx); extremely critically ill patients who have a single-lumen tube already in place and who will not tolerate being taken off mechanical ventilation and PEEP even for a short time; and patients having some combination of these problems. Under these circumstances, it is still possible to separate the lungs safely and adequately by using a single-lumen tube and fiberoptic bronchoscopic placement of a bronchial blocker or by fiberoptic bronchoscopic placement of a single-lumen tube in a main stem bronchus.

Bronchial Blockers (With Single-Lumen Endotracheal Tubes)
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Lung separation can be effectively achieved with the use of a single-lumen endotracheal tube and a fiberoptically placed bronchial blocker. This is often necessary in children because DLTs are too large to be used in them. The smallest DLT available is a left-sided 26 French tube, which may be used in patients 8 to 12 years old and weighing 25 to 35 kg. Bronchial blockers that are balloon-tipped luminal catheters have the advantage of allowing suctioning and injection of oxygen down the central lumen. The bronchial blocker most widely used for adults is the movable bronchial blocker that is contained in and is an integral part of the Univent single-lumen tube system (made by Fuji Systems Corp., Tokyo) 275, 276, 277, 278, 279, 280, 281 (Fig. 48–23). The physiology of one-lung ventilation produced by bronchial blockade is identical to that produced by clamping one lumen of a DLT.
FIGURE 48–23 Single-lumen tube of Univent bronchial blocker system.

Insertion of the Univent Tube and Positioning of the Bronchial Blocker

The tube is inserted in the following manner. First, the single-lumen tube along with the bronchial blocker (in the fully retracted position) is inserted as a unit into the trachea (Fig. 48–24A). The cuff on the main endotracheal tube lumen is inflated, and the patient is ventilated and oxygenated (see Fig. 48–24A). A fiberoptic bronchoscope is inserted through a self-sealing diaphragm in the elbow connector to a single-lumen tube while ventilation is maintained around the bronchoscope (but within the single-lumen tube) (Fig. 48–24C). The right and left mainstem bronchi are identified (by noting the relationship of the main stem bronchi to the posterior membrane and the anterior cartilaginous rings [Fig. 48–24B.]), and the tube of the bronchial blocker is located (by moving the bronchial blocker in and out just beyond the end of its own lumen and the main lumina of the Univent tube [Fig. 48–24.]). The bronchial blocker cuff is colored blue and is easy to see. It will be seen that the bronchial blocker will usually (almost always) enter the right mainstem bronchus if it is simply pushed in (and the main single-lumen tube is not turned). If the left mainstem bronchus is to be blocked, the main single-lumen tube is turned 90 degrees to the left (counterclockwise) so that the concavity of the tube is facing toward the left side (Fig. 48–24C) (and vice versa for the right side, if necessary). The bronchial blocker can also be rotated a slight amount at its distal end (obtain 1–3 mm of laterality) by twirling the proximal end in the fingers. The bronchial blocker is then advanced into the mainstem bronchus under direct vision (Fig. 48–24D). Attempting to advance the bronchial blocker blindly into the appropriate mainstem bronchus (particularly the left) will be unsuccessful 87 percent of the time, and repeated attempts may cause excoriation of the tracheal mucosa. 227 In fact, blindly pushing the somewhat stiff bronchial blocker may result in perforation of the tracheobronchial tree and consequent tension pneumothorax. 278 The balloon is inflated until the cephalad surface of the balloon is just below the tracheal carina (Fig. 48–24E) (so that the upper lobe of the blocked lung may also distend if CPAP is applied to the blocked lung [see below], and the fiberoptic bronchoscope is then withdrawn (Fig. 48–24F).
FIGURE 48–24 The sequential steps of the fiberoptic-aided method of inserting and positioning the Univent bronchial blocker (BB) in the left mainstem bronchus are illustrated. One- or two-lung ventilation is achieved by simply inflating or deflating, respectively, the bronchial blocker balloon.

Advantages and Noteworthy Positive Attributes of the Univent Bronchial Blocker Tube System

The Univent bronchial blocker tube has six important attributes that require special mention (Table 48–13). First and foremost, the degree of difficulty in inserting the Univent tube is equivalent to a standard single-lumen tube and therefore in many instances will be an easier and quicker way to separate the lungs to obtain simple one-lung ventilation (as compared with a DLT). 91, 93 Thus and as an example, the Univent tube may be preferable when difficult intubation is anticipated and when the patient has been treated with anticoagulants. Second, the patient can be continuously ventilated while the bronchial blocker is being placed into a mainstem bronchus, and the bronchial blocker can be placed into a mainstem bronchus just as easily in the LDP as in the supine position. Third, and provided that the postanesthesia care unit and intensive care unit personnel are instructed in the design and function of the Univent tube (particularly the ventilatory consequence of inflating the bronchial blocker cuff just distal to the main lumen [i.e., the main lumen will be obstructed]), the Univent tube may be left *in situ* for postoperative mechanical ventilation and the risk of a potentially difficult tube change (e.g., from a DLT to a single-lumen tube) thereby avoided. Fourth and similarly, the Univent tube may be left *in situ* if a patient is turned from the supine to the prone position midway through a surgical procedure (a common occurrence with surgery on the thoracic spine). Fifth, the unique characteristic of a movable endobronchial blocker permits the Univent endotracheal tube to create selective partial collapse (e.g., of a lobe) or total collapse of the targeted lung. 282 The capability of selectively blocking lung segments is extremely important in cases of isolated pulmonary hemorrhage. Partial versus total one-lung ventilation may allow for an improvement in PaO2 in cases of intraoperative hypoxemia during thoracic operations. Finally, although this is not a distinct advantage over a DLT, it should be noted that it is possible to apply CPAP to the nonventilated operative lung through the lumen of the bronchial blocker, 283 and therefore the Univent tube provides the same best solution to hypoxemia during one-lung ventilation as does a DLT. In fact, except for independent unilateral intermittent positive-pressure ventilation and suctioning, all selective differential lung functions possible with a DLT are possible with a Univent bronchial blocker tube.

TABLE 48–13. Advantage/Noteworthy Positive Attributes of the Univent Bronchial Blocker Tube (Relative to a Double-Lumen Tube and Other Bronchial Blockers)

Potential Limitations of the Univent Bronchial Blocker Tube System and Solutions to the Limitations

There are several distinct limitations to the Univent bronchial blocker tube system, but fortunately all have a relatively simple remedy (Table 48–14). First, the small lumen of the bronchial blocker results in slow inflation of the lung if gases are just insufflated (e.g., at a flow rate of 10 L/min, a lung will require at least 20 seconds to reach total lung capacity) or pushed in by conventional positive pressure. The operative lung may be made to expand rapidly if the bronchial blocker cuff is deflated (the operative lung will expand with one positive-pressure breath from the main single lumen) or if one very short (e.g., <0.5-sec) burst of wall oxygen-powered 20- to 30-psi jet ventilation (reduced from 50 psi) is administered. However, connection of the bronchial blocker lumen to a jet ventilator is potentially dangerous (i.e., it can cause barotrauma) because the lung can expand extremely rapidly, and it is of paramount importance that the anesthesiologist directly observe the lung and that the ventilation be very short or the pressure limited to 20 to 30 psi by an additional in-line regulator. Second, and also because the bronchial blocker lumen is small, the lung will deflate very slowly when blocked. This is easily remedied by deflating the bronchial blocker cuff (which reestablishes continuity between the operative lung and the main single lumen), disconnecting the patient from the ventilator, and leaving the endotracheal tube open to air while the surgeon gently compresses the lung to evacuate air from the operative lung through the main single lumen. After the lung is thus fully collapsed, the blocker balloon is inflated and ventilation resumed. 91, 92 Alternatively, the lumen of the bronchial blocker may be connected to the suction apparatus while the cuff is inflated; a normal amount of wall suction greatly facilitates lung collapse. Third, and also because the bronchial blocker lumen is small, the lumen is relatively easily blocked by blood and/or pus. High suction will occasionally clear the lumen of these materials and total blockage by inspissated secretions can be broken up by a wire stylet. Fourth, the Univent bronchial blocker behaves as a high-pressure cuff when intracuff volume is greater than 2 mL (the resting volume of the cuff) and may be expected to have an intracuff pressure between 150 and 250 mm Hg and a transmural pressure (intracuff pressure within the airway minus intracuff pressure outside the airway [free in the room]) between 50 and 60 mm Hg when intracuff volumes of 4 to 6 mL are used to seal 12- to 18-mm airways against the usual proximal airway pressure. 284, 285 Thus, the order of usual bronchial cuff pressures is left-sided PVC DLT < right-sided polyvinylchloride DLT < Univent Bronchial Blocker cuff < red rubber double-lumen tube. 286 These findings underscore the need to inflate the bronchial blocker cuff with

a just-seal volume of air. Fifth, the Univent bronchial blocker has on occasion been reported to have a minor leak during surgery (25% in one series), 277 but this is not understandable in view of experiments showing that the Univent bronchial blocker cuff seals within normal-sized mainstem bronchi against proximal airway pressures as great as 100 cm H2 O with inflation volumes that are within the manufacturer's recommendation. 285 Consequently, if an intraoperative leak occurs when less than a 6- to 7-mL intracuff volume has been used and the bronchial blocker cuff is completely subcarinal (as determined by fiberoptic bronchoscopy) and intact, the intracuff volume should be increased. If an intraoperative leak develops even though an adequate cuff inflation volume has been used (the bronchial blocker can be seen [fiberoptically] to fill the mainstem bronchus in question) and the bronchial blocker cuff is completely subcarinal (as determined fiberoptically) and intact, the relationship between the mainstem bronchus and the bronchial blocker cuff may no longer be a simple matter of a sphere or ellipsoid being inflated within a cylinder. Under these circumstances the surgeon may need to rearrange the surgical field so that the mainstem bronchus and bronchial blocker cuff are less distorted. Finally, the addition of the lumen for the bronchial blocker results in an endotracheal tube that has a large outside anteroposterior diameter relative to its inside diameter.

TABLE 48–14. Limitations to the Use of the Univent Bronchial Blocker Tube and Solutions to the Limitations

Methods to Obtain a Just-Seal Volume in the Bronchial Blocker Cuff

There are two methods to obtain a just-seal volume of air in the bronchial blocker cuff. The first method is the same as that already described for obtaining a just-seal volume of air in the endobronchial cuff of a DLT. It consists of pressurizing the main single lumen until air ceases to escape from the bronchial blocker lumen (detected by connecting the bronchial blocker lumen to a catheter that is submerged beneath the surface of a beaker of water; when air bubbles cease to come out, the bronchial blocker cuff has sealed) (see Fig. 48–22).

The second method appears promising and uses capnography. End-tidal CO2 analyzers draw gas samples from the anesthesia breathing circuit via tubing that terminates, at the patient end of the tubing, in a standard Luer lock male connector that inserts into a female port in the breathing circuit. The male connector also inserts into/attaches to the female port at the proximal end of the Univent's bronchial blocker. The tracing from a gas analyzer, connected to the blocker with its cuff deflated, shows a typical respiratory waveform. As the cuff of the bronchial blocker is steadily inflated, a point is reached at which the respiratory waveform abruptly ceases, and a straight line is seen, indicating that lung isolation has occurred (Fig. 48–25). The CO2 concentration remains near its end-tidal value until the blocked lung has collapsed and then rapidly decreases. The strengths of this method include simplicity, repeatability, and ability to ventilate the unblocked lung continuously throughout the procedure.
FIGURE 48–25 Capnogram tracing showing normal respiratory waveform changing to a straight line as bronchial seal occurs. (From Essig K and Freeman JA605)

Clinical Indications for Use of the Univent Bronchial Blocker System

There are several clinical situations in which use of the Univent bronchial blocker tube is relatively indicated. First, whenever it is anticipated that postoperative ventilation will be necessary (e.g., poor pulmonary function preoperatively, anticipated lung damage or massive fluid and/or blood infusion intraoperatively, anticipated very long case), use of the Univent bronchial blocker tube for lung separation may avoid a risky postoperative DLT to single-lumen tube change. Second and similarly, use of the Univent bronchial blocker tube will avoid a potentially dangerous DLT to single-lumen tube change in cases of surgery on the thoracic spine in which a thoracotomy in the supine or lateral decubitus position is followed by surgery in the prone position. Third, a very severely distorted airway may prevent successful placement of a DLT, whereas such distortion may have much less of an effect on the proper placement of the Univent tube. Finally, but least predictable or compelling, are situations in which both lungs may need to be blocked (e.g., bilateral operations, indecisive surgeons).

Bronchial Blockers That Are Independent of a Single-Lumen Tube
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The bronchial blocker that is independent of the single-lumen tube most often used for adults is a Fogarty occlusion (embolectomy) catheter with a 3-mL balloon. 287 The Fogarty catheter includes a stylet so that it is possible to place a curvature at the distal tip to facilitate entry into the larynx and either mainstem bronchus (by twirling the proximal end). If no endotracheal tube is in place, the operator exposes the larynx and places a single-lumen tube with a high-volume cuff in the trachea. The Fogarty catheter is then placed either inside 288, 289 or alongside the single-lumen tube (Fig. 48–26). In either case (bronchial blocker inside or outside the single-lumen tube), a fiberoptic bronchoscope is passed down to the end of the single-lumen tube through a self-sealing diaphragm in the elbow connector (which permits continued positive-pressure ventilation around the fiberoptic bronchoscope), and the Fogarty catheter is visualized below the tip of the single-lumen tube. The proximal end of the bronchial blocker is then twirled in the fingertips and advanced until the distal tip locates in the desired mainstem bronchus. The catheter balloon is then inflated under direct visualization and the fiberoptic bronchoscope withdrawn through the self-sealing diaphragm.
FIGURE 48–26 Lung separation with a single-lumen tube, fiberoptic bronchoscope, and right lung bronchial blocker. The sequence of events is as follows: (A) A single-lumen tube is inserted and the patient is ventilated. (B) A bronchial blocker is passed alongside the indwelling endotracheal tube. (C) A fiberoptic bronchoscope is passed through a self-sealing diaphragm in the elbow connecter to the endotracheal tube and is used to place the bronchial blocker into the right mainstem bronchus under direct vision. (D) The balloon on the bronchial blocker is also inflated under direct vision and is positioned just below the tracheal carina. (E) The fiberoptic bronchoscope is then removed (right lower diagram). During the lower panel sequence (insertion and use of fiberoptic bronchoscope, Figs. C to E) the self-sealing diaphragm allows the patient to continue to be ventilated with positive-pressure ventilation (around the fiberoptic bronchoscope, but within the lumina of the endotracheal tube). LL, left lung; RL, right lung. (From Benumof604)

Finally, other balloon-tipped luminal catheters (such as the Magill or Foley type) may be used as bronchial blockers.

For bronchial blockage in very small children (10 kg or less) a Fogarty embolectomy catheter with a balloon capacity of 0.5 mL or a Swan-Ganz catheter (1-mL balloon) should be used. 290 Of course, these catheters have to be positioned under direct vision; a fiberoptic bronchoscope method, as depicted in Figure 48–26, is perfectly acceptable, except the fiberoptic bronchoscope outside diameter must be approximately 2 mm to fit inside the endotracheal tube (3-mm internal diameter or greater). Otherwise, the bronchial blocker must be situated with a rigid bronchoscope. Pediatric patients of intermediate size require intermediate size occlusion catheters and judgment on the mode of placement (i.e., via rigid versus fiberoptic bronchoscope).

Disadvantages of bronchial blockage with a blocker that is independent of the single-lumen tube as compared with DLT lung separation include inability to suction and/or to ventilate the lung distal to the blocker, increased placement time, and the definite need for a fiberoptic or rigid bronchoscope. In addition, if a mainstem bronchial blocker backs out into the trachea, the seal between the two lungs will be lost, and two catastrophic complications may occur. First, if the bronchial blocker was being used to seal off fluid (blood or pus) in one lung, then both lungs may become contaminated with the fluid. Second, the trachea will be at least partially obstructed by the blocker, and ventilation will be greatly impaired. Therefore, bronchial blockage requires that the anesthetist continuously and intensively monitor the compliance and breath sounds of the ventilated lung.

Endobronchial Intubation With Single-Lumen Tubes

In adults presenting with hemoptysis, endobronchial intubation with a single-lumen tube is often the easiest, quickest way of effectively separating the two lungs, especially if the left lung is bleeding, in which case one can simply take an uncut single-lumen endotracheal tube and advance it inward until moderate resistance is felt. In the vast majority of patients, the single-lumen tube will locate in the right mainstem bronchus, thereby blocking off the bleeding left lung and allowing selective ventilation of only the right lung. Under these circumstances it is highly possible that the right upper lobe bronchus will be blocked off as well, resulting in ventilation of only the right middle and lower lobes. Ventilation of only a soiled right lung or ventilation of only the right middle and lower lobes (even if they are unsoiled) incurs the risk of serious hypoxemia due to the very large transpulmonary shunt that is necessarily created by single-lung endobronchial intubation.

If the right lung is bleeding, there are two ways of selectively intubating the left mainstem bronchus. First, this may be done blindly with approximately a 92 percent success rate by turning the patient‘s head to the

right and passing the single-lumen tube with the concavity of the tube facing posterior (rotated 180 degrees from its normal position and relationship to the trachea). 291 The single-lumen tube enters the right or left mainstem bronchus when the concavity of the tube faces anteriorly or posteriorly, respectively, because the bevel is left- or right-facing, respectively (i.e., a left-facing bevel enters the right mainstem bronchus and a right-facing bevel enters the left mainstem bronchus). 292 Second, a fiberoptic bronchoscope can be passed through a self-sealing diaphragm in the single-lumen tube elbow connector and directed into the left mainstem bronchus. Persistent large, soft catheter suctioning of the carinal area through the single-lumen tube before use of the fiberoptic bronchoscope and suctioning through the fiberoptic bronchoscope (through the single-lumen tube) may be required to visualize the tracheal carina. The single-lumen tube can then be passed over the fiberoptic bronchoscope into the left mainstem bronchus, thereby isolating the bleeding right lung and allowing selective ventilation of the left lung. Passing the fiberoptic bronchoscope through a self-sealing diaphragm allows the continuance of positive-pressure ventilation and PEEP around the bronchoscope. However, it should be realized that visualization of the carina may not be possible when the bleeding is copious and that the only hope for the patient may lie in rapid thoracotomy and control of bleeding from within the chest. In addition, under these adverse conditions, conventional passage of a DLT tube may more rapidly and effectively separate the two lungs than visualization of the anatomy with a fiberoptic bronchoscope.

In summary, use of DLTs is the method of choice for separating the lungs in most adult patients. If there is any question, the precise location of a DLT can be determined by fiberoptic bronchoscopy at any time. There are a number of situations in which insertion of a DLT may be difficult and/or dangerous, and under these circumstances consideration should be given to separating the lungs with a single-lumen tube alone or in combination with a bronchial blocker (e.g., the Univent tube). However, when using a single-lumen tube in a mainstem bronchus or when using a bronchial blocker, ability to suction the operative site and control oxygen uptake (the blocked lung cannot be ventilated with oxygen at any time) is limited. In addition, placement of the single-lumen tube into one or the other mainstem bronchus and proper placement of a bronchial blocker usually require fiberoptic bronchoscopy. Therefore, no matter what method of separating the lungs is chosen, there is a real need for the immediate availability of a small-diameter fiberoptic bronchoscope (for checking the position of the DLT, placing a single-lumen tube in the left mainstem bronchus, and placing a bronchial blocker) that has a suction port to clear secretions and blood from the airway.

Physiology of One-Lung Ventilation

Comparison of Arterial Oxygenation and Carbon Dioxide Elimination During Two-Lung Versus One-Lung Ventilation

As discussed previously, the matching of ventilation and perfusion is impaired during two-lung ventilation in an anesthetized, paralyzed, open-chested patient in the LDP. The reason for the mismatching is relatively good ventilation but poor perfusion of the nondependent lung and poor ventilation and good perfusion of the dependent lung (see Figs. 48–13 and 48–26A). The blood flow distribution has been seen to be mainly and simply determined by gravitational effects. The relatively good ventilation of the nondependent lung has been seen to be caused, in part, by the open chest and paralysis. The relatively poor ventilation of the dependent lung has been seen to be caused, in part, by the loss of dependent lung volume with general anesthesia and by circumferential compression of the dependent lung by the mediastinum, abdominal contents, and suboptimal positioning effect. The compression of the dependent lung may cause the development of a shunt compartment in this lung (see Fig. 48–13; Fig. 48–27A). Consequently, two-lung ventilation under these circumstances may result in an increased P(A-a)O2 and impaired oxygenation.
FIGURE 48–13 Schematic summary of ventilation-perfusion relationships in the anesthetized patient in the lateral decubitus position who has an open chest and is paralyzed and suboptimally positioned. The nondependent lung is well ventilated (as indicated by the large dashed lines) but poorly perfused (small perfusion vessel); the dependent lung is poorly ventilated (small dashed lines) but well perfused (large perfusion vessel). In addition, the dependent lung may also develop an atelectatic shunt compartment (indicated on the left side of the lower lung) because of the circumferential compression of this lung. (See text for detailed explanation.) PAB, pressure of the abdominal contents. (Modified from Benumof604)
FIGURE 48–27 Schematic representation of two-lung ventilation versus one-lung ventilation. Typical values for fractional blood flow to the nondependent and dependent lungs as well as arterial oxygen tension (PaO2) and shunt (QS/QT) for the two conditions are shown. The QS/QT during two-lung ventilation is assumed to be distributed equally between the two lungs (5 percent to each lung). The essential difference between two-

lung and one-lung ventilation is that during one-lung ventilation the nonventilated lung has some blood flow and therefore has an obligatory shunt, which is not present during two-lung ventilation. The 35 percent of total flow perfusing the nondependent lung, which was not shunt flow, was assumed to be able to reduce its blood flow by 50 percent by hypoxic pulmonary vasoconstriction. The increase in QS/QT from two-lung to one-lung ventilation is assumed to be solely due to the increase in shunt through the nonventilated, nondependent lung during one-lung ventilation. (From Benumof604)

If the nondependent lung is nonventilated, as during one-lung ventilation, then any blood flow to the nonventilated lung becomes shunt flow, in addition to whatever shunt flow might exist in the dependent lung (Fig. 48–27B). Thus, one-lung ventilation creates an obligatory right-to-left transpulmonary shunt through the nonventilated nondependent lung, which is not present during two-lung ventilation. Consequently, it is not surprising to find that, given the same inspired oxygen concentration (FIO2) and hemodynamic and metabolic status, one-lung ventilation results in a much larger P(A-a)O2 and lower PaO2 than two-lung ventilation. This contention is best supported by one study that compared arterial oxygenation during two-lung and one-lung ventilation, wherein each patient served as his or her own control. 293

One-lung ventilation has much less of a steady-state effect on PaCO2 than on PaO2. Blood passing through underventilated alveoli retains more than a normal amount of carbon dioxide and does not take up a normal amount of oxygen; blood traversing overventilated alveoli gives off more than a normal amount of carbon dioxide but cannot take up a proportionately increased amount of oxygen because of the flatness of the top end of the oxyhemoglobin dissociation curve. Thus, during one-lung ventilation (the one-lung minute ventilation equals the two-lung minute ventilation) the ventilated lung can eliminate enough carbon dioxide to compensate for the nonventilated lung, and PACO2 to PaCO2 gradients are small; however, the ventilated lung cannot take up enough oxygen to compensate for the nonventilated lung, and PAO2 to PaO2 gradients are usually large. With a constant minute ventilation (two-lung ventilation as compared with one-lung ventilation), the retention of carbon dioxide by blood traversing the nonventilated lung usually slightly exceeds the increased elimination of carbon dioxide from blood traversing the ventilated lung, and the PaCO2 will usually slowly increase (along with the ETCO2).

The initiation of one-lung ventilation has much more of an acute effect (first 5 minutes) on PETCO2. When one-lung ventilation is begun (keeping total tidal volume and respiratory rate constant), the ventilated lung is immediately hyperventilated in relation to its perfusion (i.e., has an increased V/Q ratio) and PETCO2 from this lung decreases in the first minute (e.g., by 5 mm Hg). 294

Over the next 5 minutes, HPV in the nonventilated lung shifts blood flow over to the ventilated lung, increases ventilated lung perfusion, decreases ventilated lung V/Q ratio and increases the PETCO2 back to the baseline two-lung ventilation value. 294 Thereafter, and as discussed previously, PETCO2 will slowly increase (along with PaCO2) because the same total minute ventilation to one lung is not as effective as when it is delivered to both lungs (i.e., there is an increased alveolar dead space within the one ventilated lung). 294

Blood Flow Distribution During One-Lung Ventilation

Blood Flow to the Nondependent, Nonventilated Lung

Fortunately, both passive mechanical and active vasoconstrictor mechanisms are usually operant during one-lung ventilation that minimize the blood flow to the nondependent, nonventilated lung and thereby prevent PaO2 from decreasing as much as might be expected on the basis of the distribution of blood flow during two-lung ventilation. The passive mechanical mechanisms that decrease blood flow to the nondependent lung are gravity, surgical interference with blood flow, and perhaps the extent of preexisting disease in the nondependent lung (Fig. 48–28). Gravity causes a vertical gradient in the distribution of pulmonary blood flow in the LDP for the same reason that it does in the upright position (see Fig. 48–9). Consequently, blood flow to the nondependent lung is less than that to the dependent lung. The gravity component of blood flow reduction to the nondependent lung should be constant with respect to both time and magnitude.

FIGURE 48–28 Schematic diagram of the determinants of blood flow distribution during one-lung ventilation. The major determinants of blood flow to the nondependent lung are gravity, surgical interference with blood flow, amount of nondependent lung disease, and magnitude of nondependent lung hypoxic pulmonary

vasoconstriction. The determinants of dependent lung blood flow are gravity, amount of dependent lung disease, and dependent lung hypoxic pulmonary vasoconstriction. (From Benumof604)
FIGURE 48–9 Schematic representation of the effects of gravity on the distribution of pulmonary blood flow in the lateral decubitus position. The vertical gradient in the lateral decubitus position is less than in the upright position. Consequently, there is less zone 1 and more zone 2 and 3 blood flow in the lateral decubitus position than in the upright position. Nevertheless, pulmonary blood flow increases with lung dependency and is greater in the dependent lung than in the nondependent lung. PA, alveolar pressure; Ppa, pulmonary artery pressure; Ppv, pulmonary venous pressure. (From Benumof604)

Surgical compression (directly compressing lung vessels) and retraction (causing kinking and tortuosity of lung vessels) of the nondependent lung may further passively reduce nondependent lung blood flow. In addition, ligation of pulmonary vessels during pulmonary resection greatly decreases nondependent lung blood flow. The surgical interference component of blood flow reduction to the nondependent lung should be variable with respect to both time and magnitude.

The amount of disease in the nondependent lung is also a significant determinant of the amount of blood flow to the nondependent lung. If the nondependent lung is severely diseased, there may be a fixed reduction in blood flow to this lung preoperatively and collapse of such a diseased lung may not cause much of an increase in shunt. The notion that a diseased pulmonary vasculature might be incapable of HPV is supported by the observations that administration of sodium nitroprusside and nitroglycerin (which should abolish any preexisting HPV) to COPD patients (who have a fixed reduction in the cross-sectional area of their pulmonary vascular bed) does not cause an increase in shunt, 295 whereas these drugs do increase shunt in patients with acute regional lung disease who have an otherwise normal pulmonary vascular bed. 296 If the nondependent lung is normal and has a normal amount of blood flow preoperatively, collapse of such a normal lung may be associated with a higher nonventilated nondependent lung blood flow and shunt. A higher one-lung ventilation shunt through the nondependent lung may be more likely to occur in patients who require thoracotomy for nonpulmonary disease. 297 Two studies have systematically validated the inverse correlation between the amounts of nondependent lung disease and shunt during one-lung ventilation. 298, 299

The most significant reduction in blood flow to the nondependent lung is caused by an active vasoconstrictor mechanism. The normal response of the pulmonary vasculature to atelectasis is an increase in PVR (in just the atelectatic lung); this increase, thought to be due almost entirely to HPV, 153, 154, 155 diverts blood flow from the atelectatic lung toward the remaining normoxic or hyperoxic ventilated lung. The diversion of blood flow minimizes the amount of shunt flow that occurs through the hypoxic lung. Figure 48–6 shows the theoretically expected effect of HPV on arterial oxygen tension (PaO2) as the amount of lung that is made hypoxic increases. 209 When very little of the lung is hypoxic (near 0%) it does not matter, in terms of PaO2, whether the small amount of lung has HPV operating or not because in either case the shunt will be small. When most of the lung is hypoxic (near 100%) there is no significant normoxic region to which the hypoxic region can divert flow, and again it does not matter, in terms of PaO2, whether or not the hypoxic region has HPV operating. When the percentage of lung that is hypoxic is intermediate (between 30 and 70%), which is the amount of lung that is typically hypoxic during the one-lung ventilation-anesthesia condition, there is a large difference between the PaO2 expected with a normal amount of HPV (which is a 50% blood flow reduction for a single lung) 209 as compared with no HPV. In fact, in this range of hypoxic lung, HPV can increase PaO2 from hypoxemic levels to much higher and safer values. It is not surprising, then, that numerous clinical studies on one-lung ventilation have found that the shunt through the nonventilated lung is usually 20 to 30 percent of the cardiac output, as opposed to the 40 to 50 percent shunt that might be expected if there were no HPV operating in the nonventilated lung. 297, 300, 301, 302, 303, 304, 305 Thus, HPV is an autoregulatory mechanism that protects the PaO2 by decreasing the amount of shunt flow that can occur through hypoxic lung.
FIGURE 48–6 Effect of hypoxic pulmonary vesoconstriction (HPV) on PaO2. As the amount of lung that is made hypoxic is increased (x axis), the arterial oxygen tension (PaO2) decreases (y axis). In the range of 30 to 70 percent of hypoxic lung, the normal expected amount of HPV increases PaO2 from arrhythmogenic levels to much higher and safer levels. Normal cardiac output, hemoglobin concentration, and mixed venous oxygen tension ($\bar{P}v O_2$) are assumed. (Data from Marshall and Marshall209)

Figure 48–29 outlines the major determinants of the amount of atelectatic lung HPV that might occur during anesthesia. In the following discussion, the HPV issues or considerations are numbered as they are in Figure 48–29.

FIGURE 48–29 Listing of many of the components of the anesthetic experience that might determine the amount of regional hypoxic pulmonary vasoconstriction (HPV). The clockwise numbering of considerations corresponds to the order in which these considerations are discussed in the text. V/Q, ventilation/perfusion ratio; PVP, pulmonary vascular pressure; $P\bar{v}O_2$, mixed venous oxygen tension; FIO_2, inspired oxygen fraction; $PACO_2$, alveolar carbon dioxide tension; PEEP, positive end-expiratory pressure. (From Benumof604)

1.
The distribution of the alveolar hypoxia is probably not a determinant of the amount of HPV; all regions of the lung (either the basilar or dependent parts of the lungs [supine or upright] or discrete anatomic units such as a lobe or single lung) respond to alveolar hypoxia with vasoconstriction. 306 However, recent evidence suggests that on a sublobar level, collateral ventilation may be the first line and HPV the second line of defense against the development of arterial hypoxemia. 307
2.
As with low V/Q ratios and nitrogen-ventilated lungs, it appears that the preponderance of blood flow reduction in acutely atelectatic lung is due to HPV and none of it to passive mechanical factors (such as vessel tortuosity). 153, 154, 155 This conclusion is based on the observation that reexpansion and ventilation of a collapsed lung with nitrogen (removing any mechanical factor) do not increase the blood flow to the lung, whereas ventilation with oxygen restores all the blood flow to precollapse values. This conclusion applies whether ventilation is spontaneous or due to positive pressure and whether the chest is open or closed. 154 In canines, a slight amount of further subacute (>30 min) decrease in blood flow to atelectatic lung may have been due to some mechanical effect of the atelectasis on lung blood vessels. 308 However, in humans a prolonged unilateral hypoxic challenge during anesthesia results in an immediate vasoconstrictor response with no further potentiation or diminution of the HPV response. 309
3.
Most systemic vasodilator drugs either inhibit regional HPV directly or have an effect in a clinical situation that is consistent with inhibition of regional HPV (i.e., decreasing PaO_2 and increasing shunt in patients with acute respiratory disease). The vasodilator drugs that have been shown to inhibit HPV or to have a clinical effect con-sistent with inhibition of HPV are nitroglycerin, 296, 310, 311, 312, 313, 314, 315, 316 nitroprusside, 296, 317, 318, 319, 320, 321, 322, 323 dobutamine, 324, 325 several calcium antagonists, 326, 327, 328, 329, 330, 331 and many ?2 -agonists (isoproterenol,ritodrine, orciprenaline, salbutamol, ATP, and glucagon). 325, 332, 333, 334, 335, 336, 337, 338 Nitric oxide, a potent inhaled pulmonaryvasodilator, has been studied extensively and is considered to inhibit HPV. 339, 340, 341, 342, 343, 344 Aminophylline and hydralazine may not decrease HPV. 345
4.
The effect of anesthetic drugs on regional HPV was previously discussed in the section *Effect of Anesthetics on Hypoxic Pulmonary Vasoconstriction.*
5.
The HPV response is maximal at normal and decreased at either high or low pulmonary vascular pressure. The mechanism for high pulmonary vascular pressure inhibition of HPV whether the cardiac output is high or low 346, 347 is simple; the pulmonary circulation is poorly endowed with smooth muscle and cannot constrict against an increased vascular pressure. Furthermore, in the one-lung ventilation situation in the LDP, it is obvious, with all other factors remaining constant, that the fraction of the cardiac output perfusing the collapsed nondependent lung will increase with increasing pulmonary arterial pressure (i.e., the effect of gravity will be overcome). 348 The mechanism for low pulmonary vascular pressure inhibition of HPV is more complex. For this to occur, the hypoxic compartment must be atelectatic. Under these circumstances, when pulmonary vascular pressure decreases, it is possible for part of the ventilated lung (but not the atelectatic lung) to be in a zone 1 condition (alveolar pressure increases in relation to pulmonary artery pressure) and to experience a disproportionate increase in PVR, which would divert blood flow back over to the atelectatic lung, thereby inhibiting atelectatic lung HPV. 349
6.
The HPV response also is maximal when $P\bar{v}O_2$ is normal and is decreased by either high or low $P\bar{v}O_2$. The mechanism for high $P\bar{v}O_2$ inhibition of HPV is presumably due to reverse diffusion of oxygen, causing the oxygen tension of either the vessels, the interstitial or alveolar spaces, or all of these to be increased above the HPV threshold. 350 That is, if enough oxygen can reach some receptor in the small arteriole-capillary-alveolar area, then the vessels will not vasoconstrict. The mechanism for low $P\bar{v}O_2$ inhibition of HPV is a result of the low $P\bar{v}O_2$ decreasing alveolar oxygen tension in the normoxic compartment down to a level sufficient to induce HPV in the supposedly normoxic lung. 351 The HPV in the presumably normoxic

lung competes against and offsets the HPV in the originally hypoxic lung and results in no blood flow diversion from the more obviously hypoxic lung.

7.

Selectively decreasing the FIO2 in the normoxic compartment (from 1.0 to 0.5 to 0.3) causes an increase in normoxic lung vascular tone, thereby decreasing blood flow diversion from hypoxic to normoxic lung. 351 Indeed, with unilateral lung injury, ventilation of both lungs with a hypoxic gas mixture (FIO2 of 0.12) induces much more vasoconstriction in the normal, previously nonconstricted lung than in the injured and already hypoxically constricted lung, which redirects blood flow to, increases the shunt through, and increases edema in the injured lung. 352 In addition, the development of systemic hypoxemia, either when bilateral hypoxic ventilation is used or when there is a very largehypoxic compartment and a small normoxic compartment, may indirectly inhibit regional HPV by stimulation of arterial chemoreceptors. 353 At the other extreme, prolonged exposure to hyperoxia (FIO2 of 1.0) for 68 hours blunts a subsequent whole-lung HPV response. 354

8.

Older studies have suggested that the vasoconstrictor drugs (dopamine, epinephrine, phenylephrine) constrict normoxic lung vessels preferentially, thereby disproportionately increasing normoxic lung 325, 329, 335 PVR. The increase in normoxic lung PVR would be expected to decrease normoxic lung blood flow and increase atelectatic lung blood flow. The HPV-inhibiting effect of vasoconstrictor drugs is similar to that of decreasing normoxic lung FIO2 (see item 7). In recent years, dopamine has been extensively studied. Although one reasonably straightforward study 355 agrees well with previous studies, 325, 329, 335, 347 most recent studies 356, 357, 358 have shown no significant effect of dopamine on HPV and/or arterial oxygenation. On the basis of these latter more recent studies, dopamine appears to be a reasonable cardiovascular stimulant to use in patients with lung disease provided that arterial oxygenation is monitored.

9.

Hypocapnia has been thought to directly inhibit and hypercapnia to directly enhance regional HPV. 346, 359 In addition, during one-lung ventilation conditions, hypocapnia can be produced only by hyperventilation of the one lung. The hyperventilation requires an increased ventilated lung airway pressure, which may cause increased ventilated lung PVR, which in turn may divert blood flow back into thehypoxic lung. Hypercapnia during one-lung ventilation seems to act as a vasoconstrictor by selectively increasing ventilated lung PVR (which would divert blood flow back to the nonventilated lung). In addition, hypercapnia is ordinarily caused by hypoventilation of the ventilated lung, which greatly increases the risk of developing low V/Q and atelectatic regions in the dependent lung. However, it should be noted as a theoretical possibility that if hypoventilation of the dependent lung is associated with decreased ventilated lung airway pressure, ventilated lung pulmonary vascular resistance may be decreased, thus in turn promoting or enhancing HPV in the nonventilated lung.

10.

The effects of changes in airway pressure due to PEEP and tidal volume changes are discussed in detail in the sections *Dependent Lung PEEP* and *Selective Dependent Lung PEEP.* In brief, selective application of PEEP to only normoxic ventilated lung will selectively increase PVR in the ventilated lung and shunt blood flow back into thehypoxic nonventilated lung (i.e., decrease nonventilated lung HPV). 360, 361 On the other hand, high-frequency ventilation of the gas-exchanging lung is associated with low airway pressure and enhancement of HPV in the collapsed lung. 362

Finally, there is some evidence that certain types of infections (which may cause atelectasis), particularly granulomatous and pneumococcal infections, may inhibit HPV. 363, 364

Blood Flow to the Dependent Ventilated Lung

The dependent lung usually has an increased amount of blood flow due to both passive gravitational effects and active nondependent lung vasoconstrictor effects. However, the dependent lung may also have a hypoxic compartment (area of low V/Q ratio and atelectasis) that was present preoperatively or that developed intraoperatively. This hypoxic compartment may develop intraoperatively for several reasons. First, in the LDP the ventilated dependent lung usually has a reduced lung volume resulting from the combined factors of induction of general anesthesia and circumferential (and perhaps severe) compression by the mediastinum from above, by the abdominal contents pressing against the diaphragm from the caudad side, and by suboptimal positioning effects (rolls, packs, chest supports) pushing in from the dependent side and axilla 207, 231, 334, 365 (see Fig. 48–13). Second, absorption atelectasis can also occur in regions of the dependent lung that have low V/Q ratios when they are exposed to high inspired oxygen concentration.

366, 367 Third, difficulty in secretion removal may cause the development of poorly ventilated and atelectatic areas in the dependent lung. Finally, maintaining the LDP for prolonged periods may cause fluid to transude into the dependent lung (which may be vertically below the left atrium) and cause further decrease in lung volume and increase in airway closure in the dependent lung. 368

The development of low V/Q ratio and/or atelectatic areas in the dependent lung increases vascular resistance in the dependent lung 365, 369 (because of dependent lung HPV), 306 thereby decreasing dependent lung blood flow and increasing nondependent lung blood flow. 370 Stated differently, the PVR in the ventilated compartment of the lung determines the ability of the ventilated, and supposedly normoxic, lung to accept redistributed blood flow from the hypoxic lung. Clinical conditions that are independent of specific dependent lung disease but that may still increase dependent lung vascular resistance in a dose-dependent manner are a decreasing inspired oxygen tension in the dependent lung (from 1.0 to 0.5 to 0.3) 351, 370 and decreasing temperature (from 40 to 30°C). 371

Miscellaneous Causes of Hypoxemia During One-Lung Ventilation

Still other factors may contribute to hypoxemia during one-lung ventilation. Hypoxemia due to mechanical failure of the oxygen supply system or the anesthesia machine is a recognized hazard of any kind of anesthesia. Gross hypoventilation of the dependent lung can be a major cause of hypoxemia. Malfunction of the dependent lung airway lumen (blockage by secretions) and malposition of the DLT are, in our experience, frequent causes of increased P(A-a)O2 and hypoxemia. Resorption of residual oxygen from the nonventilated lung is time-dependent and accounts for a gradual increase in shunt and decrease in PaO2 after one-lung ventilation is initiated. 369 With all other anesthetic and surgical factors constant, anything that decreases the $P\bar{v}O_2$ (decreased cardiac output, increased oxygen consumption [excessive sympathetic nervous system stimulation, hyperthermia, shivering]) causes increased P(A-a)O2. 372, 373 Finally, transfusion of blood may cause pulmonary dysfunction, and the dysfunction has been attributed to the action of isoantibodies against leukocytes, which causes cellular aggregation, microvascular occlusion, and capillary leakage. Indeed, such a reaction has been described during prolonged one-lung ventilation. 374 Interestingly, the noncollapsed lung was preferentially injured, and the collapsed lung showed only minimal radiologic signs of edema after reexpansion. 374

Conventional Management of One-Lung Ventilation

The proper initial conventional management of one-lung ventilation is logically based on the preceding determinants of blood flow distribution during one-lung ventilation. In view of the fact that one-lung ventilation incurs a definite risk of causing systemic hypoxemia, it is extremely important that dependent lung ventilation, as it affects these determinants, be optimally managed. This section considers the usual management of one-lung ventilation in terms of the most appropriate FIO2, tidal volume, and respiratory rate (Table 48–15).

TABLE 48–15. Initial Conventional Ventilatory Management of One-Lung Anesthesia

Inspired Oxygen Concentration

Although the theoretical possibilities of absorption atelectasis and oxygen toxicity exist, the benefits of ventilating the dependent lung with 100 percent oxygen far exceed the risks. A high FIO2 in the single ventilated lung may critically increase PaO2 from arrhythmogenic and life-threatening levels to safer levels.

In addition, a high FIO2 in the dependent lung causes vasodilation, thereby increasing the dependent lung capability of accepting blood flow redistribution due to nondependent lung HPV. Direct chemical 100 percent oxygen toxicity does not occur during the operative period, 375 and absorption atelectasis in the dependent lung 375 is unlikely to occur in view of the remaining one-lung ventilation management characteristics (moderately large tidal volumes with intermittent positive-pressure, low-level PEEP). If the anesthesiologist is concerned about and wishes to limit the FIO2 in patients treated with either bleomycin or mitomycin, it is possible to carefully increase or decrease FIO2 to both lungs independently in an attempt to have the lowest FIO2 to both lungs (even when CPAP is administered to the nonventilated lung). 376

Tidal Volume

The dependent lung should be ventilated with a tidal volume of approximately 10 mL/kg. A much smaller tidal volume might promote dependent-lung atelectasis; a much greater tidal volume might excessively increase dependent lung airway pressure and vascular resistance 369 and thereby increase nondependent lung blood flow (decrease nondependent lung HPV). 360, 377, 378 If a tidal volume of 10 mL/kg causes excessive airway pressure, it should be lowered (after mechanical causes [i.e., tube malfunction] have been ruled out) and the respiratory rate increased (see later).

A dependent-lung tidal volume of 10 mL/kg is in the middle of a range of tidal volumes (8–15 mL/kg) that have been found in one study not to affect arterial oxygenation greatly during one-lung ventilation. 302 In that study, changes in PaO2 with alterations in tidal volume (during stable one-lung ventilation conditions) in individual patients were variable and unpredictable in both degree and direction (although the mean value for the group did not change). Thus, it appears that changing the tidal volume from 15 to 8 mL/kg during one-lung ventilation has an unpredictable but usually not great impact on arterial oxygenation. 302

Dependent Lung PEEP

No, or just a very low level of, dependent lung PEEP (<5 cm H2 O) should be used initially because of concern of unnecessarily increasing dependent lung PVR. Although this is unlikely to occur when dependent lung PEEP is less than 5 cm H2 O, 379 the presence of intrinsic PEEP during one-lung ventilation may make the total PEEP excessive 380 (see the section, *Selective Dependent-Lung PEEP*).

Respiratory Rate

The respiratory rate should be set so that the PaCO2 remains at 40 mm Hg. Because a dependent lung tidal volume of 10 mL/kg represents a 20 percent decrease from the usual two-lung tidal volume of 12 mL/kg, the respiratory rate usually has to be increased by 20 to 30 percent to maintain carbon dioxide hemostasis. The trade-off between decreased tidal volume and increased respiratory rate usually results in a constant minute ventilation; although ventilation and perfusion are considerably mismatched during one-lung ventilation, an unchanged minute ventilation during one-lung ventilation (as compared with two-lung ventilation) can continue to eliminate a normal amount of carbon dioxide because of the high diffusibility of carbon dioxide. 369, 381, 382, 383 Hypocapnia should be prevented because use of the airway pressure in the dependent lung necessary to produce systemic hypocapnia may excessively increase dependent lung vascular resistance. Furthermore, hypocapnia may directly inhibit HPV in the nondependent lung. 346, 359

In summary, at the commencement of one-lung ventilation, 100 percent oxygen, a tidal volume of 10 mL/kg, and a 20 percent increase in respiratory rate are used as initial ventilation settings (see Table 48–15). Ventilation and arterial oxygenation are monitored by use of arterial blood gases, end-tidal carbon dioxide concentration, and pulse oximetry. If there is a problem with either ventilation or arterial oxygenation, one or more of the differential lung management techniques described next are used.

Differential Lung Management of One-Lung Ventilation

Intermittent Inflation of the Nondependent Operative Lung

Intermittent inflation with oxygen of the collapsed lung during one-lung ventilation may be expected to increase PaO2 for a variable period of time. In a group of thoracic surgery patients undergoing one-lung ventilation with an inspired nitrous oxide fraction (FIN2O) of 0.5 and an FIO2 of 0.5, the collapsed lung was manually inflated with a breath every 5 minutes with 2 L of oxygen and the lung was then allowed to collapse again; PaO2 increased by more than 28 mm Hg following each inflation. 384 The beneficial effect of each inflation persisted to a large extent to the next breath even if at a gradually decreasing level. Although PaO2 decreased between inflations, it never reached the level observed in controls (no lung inflation) during 19 minutes of one-lung ventilation.

Selective Dependent Lung PEEP

Because the ventilated dependent lung often has decreased lung volume during one-lung ventilation (see Figs. 48–13, 48–28, and 48–30), it is not surprising that several attempts have been made to improve oxygenation by selectively treating the ventilated lung with PEEP. 207, 237, 304, 360, 377, 378 An accepted risk of selective dependent lung PEEP is that the PEEP-induced increase in lung volume can

cause compression of the small dependent lung intra-alveolar vessels and increase dependent lung PVR; this diverts blood flow from the ventilated lung to the nonventilated lung (see Fig. 48–30, upper right panel), increasing the shunt and decreasing the PaO2. That increases in both PEEP and tidal volume in the dependent ventilated lung have an additive effect in decreasing PaO2 during one-lung ventilation greatly supports the one-ventilated lung volume versus vascular resistance hypothesis. 377 Therefore, the effect of dependent lung PEEP on arterial oxygenation is a trade-off between the positive effect of increasing dependent lung FRC and V/Q ratio and the negative effect of increasing dependent lung PVR and shunting blood flow to the nonventilated lung. Not surprisingly, then, the various one-lung ventilation–PEEP studies have had patients who have had an increase, 304, 377, 385 no change, 377, 385, 386, 387 or a decrease 304, 377, 385, 388, 389, 390 in oxygenation. It may be expected that in patients with a very diseased dependent lung (low lung volume and low V/Q ratio), the positive effects of selective dependent lung PEEP (increased lung volume and increased V/Q ratio) might outweigh the negative effects (shunting of blood flow to the nonventilated, nondependent lung), whereas in patients with a normal dependent lung, the negative effects would outweigh the benefits. Indeed, in one study in which 10 cm H2 O PEEP was selectively applied to the dependent lung, PaO2 increased in those patients with an initial PaO2 of less than 80 mm Hg (FIO2 of 0.5), whereas PaO2 decreased or remained constant in patients with an initial PaO2 higher than 80 mm Hg (FIO2 of 0.5). 385, 390 Presumably, in the patients with PaO2 lower than 80 mm Hg, the dependent lung had a low FRC (low V/Q ratio and atelectatic regions); therefore, the positive effect of increased dependent lung volume predominated over the negative effect of shunting blood flow to the nonventilated lung. Conversely, the patients with the higher PaO2 presumably had a dependent lung with an adequate FRC and V/Q ratio, and the negative effect of shunting blood flow to the nonventilated lung predominated over the positive effect of increased dependent lung volume. Although a dose (ventilated lung PEEP) versus response (PaO2, QS/QT value) relationship has been poorly described, it seems reasonable to postulate on the basis of these results 304, 377, 385, 386, 387, 388, 389, 390 that the therapeutic margin of using PEEP to increase PaO2 during one-lung ventilation is quite narrow. PEEP only to the dependent ventilated lung may be delivered by the same anesthesia apparatus that is ordinarily used to deliver PEEP to the whole lung. Other studies have shown that high tidal volumes, 301 variations in the inspiratory/expiratory ratio, 386 and intermittent manual hyperventilation of the lower lung are not beneficial in increasing PaO2 during one-lung ventilation. 386

FIGURE 48–30 Four-part schematic diagram showing the effects of various differential lung management approaches. (A) The one-lung ventilation situation. The DOWN (dependent) lung is ventilated (VENT) but is compressed by the weight of the mediastinum (M) from above, the pressure of the abdominal contents against the diaphragm (D), and by positioning effects of rolls, packs, and shoulder supports (P). The UP (nondependent) lung is nonventilated (NONVENT), and blood flow through this lung is shunt flow. (B) The dependent lung has been selectively treated with PEEP, which improves V/Q relationships in the dependent lung but also increases dependent lung vascular resistance; this diverts blood to, and thereby increases shunt flow through, the nonventilated lung. (C) Selective application of CPAP to the nondependent lung permits oxygen uptake from this lung; even if the CPAP causes an increase in vascular resistance and diverts blood flow to the dependent lung, the diverted blood flow can still participate in gas exchange in the ventilated dependent lung. Consequently, selective nondependent lung CPAP can greatly increase PaO2. (D) With differential lung CPAP (nondependent lung)/PEEP (dependent lung), it does not matter where the blood flow goes, since both lungs can participate in O2 uptake. With this latter one-lung ventilation pattern, PaO2 can be restored to levels near those achieved by two-lung ventilation.

Selective Nondependent Lung CPAP

Low levels of positive pressure can be selectively and statically applied to only the nonventilated, nondependent lung. Because under these conditions the nonventilated lung is only slightly but constantly distended by oxygen, an appropriate term for this ventilatory arrangement is *nonventilated lung CPAP.* The application of CPAP (without tidal ventilation) to only the nonventilated lung significantly increases oxygenation. 388, 391 Also, the institution of 10 cm H2 O nondependent lung CPAP in patients has no significant hemodynamic effect. 385, 389 Low levels of CPAP simply maintain the patency of nondependent lung airways, allowing some oxygen distention of the gas exchanging alveolar space in the nondependent lung (Fig. 48–30C) without significantly affecting the pulmonary vasculature. In all clinical studies, 385, 388, 389, 392 the application of 5 to 10 cm H2 O CPAP has not interfered with the performance of surgery and may, in fact, facilitate intralobar dissection. This is not surprising in view of the fact that the initial compliance of a collapsed lung is only 10 mL/cm H2 O, and 5 to 10 cm H2 O CPAP should create only a slightly distended lung that occupies a volume of 50 to 100 mL, which is hardly or not at all noticed by the surgeon.

On the other hand, in a canine study, 391 15 cm H2 O of nondependent lung CPAP caused changes in PaO2 and shunt similar to those of 5 to 10 cm H2 O of nondependent lung CPAP, whereas blood flow to the nonventilated nondependent lung decreased significantly. Therefore, high levels of nonventilated lung CPAP act by permitting oxygen uptake in the nonventilated lung as well as by causing blood flow diversion to the ventilated lung, where both oxygen and carbon dioxide exchange can take place (see Fig. 48–30C). Because low levels of nonventilated lung CPAP are as efficacious as high levels and have less surgical interference and hemodynamic implications, it is logical to use low levels of nonventilated CPAP first.

In all patients in all clinical studies to date, 5 to 10 cm H2 O of nondependent lung CPAP has significantly increased PaO2 during one-lung ventilation. 385, 388, 389, 392, 393, 394, 395 It should be concluded that the single most efficacious maneuver to increase PaO2 during one-lung ventilation is to apply 5 to 10 cm H2 O of CPAP to the nondependent lung. In our experience, low levels of nonventilated lung CPAP have corrected severe hypoxemia (PaO2<50 mm Hg) more than 95 percent of the time, provided that the DLT was correctly positioned. However, the nondependent lung CPAP must be applied during the deflation phase of a large tidal volume so that the deflating lung can lock into a CPAP level with uniform expansion and obviate the need to overcome critical opening pressures of airways and alveoli.

In both the human 388 and canine 391 studies, oxygen insufflation at zero airway pressure did not significantly improve PaO2 and shunt, and this result was probably due to the inability of zero transbronchial airway pressure to maintain airway patency and overcome critical alveolar opening pressures. Although one study in patients has concluded that insufflation of O2 at zero airway pressure does increase PaO2, the study is difficult to interpret, because the patients did not serve as their own controls. 396

Several selective nondependent lung CPAP systems that are easy to assemble have been described. 389, 393, 394, 395 All these nondependent lung CPAP systems have three features in common (Fig. 48–31). First, there must be a source of oxygen to flow into the nonventilated lung. Second, there must be some sort of restrictive mechanism (hand-screw valve, pop-off valve, weight-loaded valve) to retard the egress of oxygen from the nonventilated lung so that the nonventilated lung may become distended. Thus, oxygen from a free-flowing pressurized source flows into a lung, but the escape of the oxygen is restricted; the unrestricted flow in and the restricted flow out create a constant distending pressure. Third, the distending pressure must be measured by a manometer. In practice, it is often simplest to keep the restrictive mechanism constant and adjust the distending pressure with a relatively fine sensitivity by changing the oxygen flow rate. If the nondependent lung CPAP system includes a reservoir bag (which is highly desirable), the reservoir bag will reflect the amount of CPAP (by distention), and the nondependent lung may also be ventilated with intermittent positive pressure whenever desired.
FIGURE 48–31 The three essential components of a nondependent lung CPAP system consist of (1) an oxygen source, (2) a pressure relief valve, and (3) a pressure manometer to measure the CPAP. The CPAP is created by the free flow of oxygen into the lung versus the restricted outflow of oxygen from the lung by the pressure relief valve. ZEEP, zero end-expiratory pressure. (From Benumof604)

The availability of a commercial nondependent nonventilated lung CPAP device (Mallinckrodt Medical, Inc.) replaces the need for all homemade devices. The Broncho-Cath CPAP System (Fig. 48–32) is similar in concept to the system shown in Figure 48–31 except that the restrictive mechanism is a continuously vented slot (opening); the larger the vent slot, the lower the CPAP level delivered, and the smaller the vent slot, the higher the CPAP level. The level of CPAP is selected by simply turning an outer calibrated cylinder (calibrated for an oxygen flow rate of 5 L/min), which progressively uncovers the inner (pop-off) slot. Because the dial is calibrated and marked with the CPAP level selected, a pressure manometer is unnecessary. The rest of the CPAP system consists of the other components shown in Figure 48–31 (oxygen tubing, reservoir bag). A unique feature of the Broncho-Cath CPAP System is the fact that the valve is continuously vented. Because the vent slot cannot be totally occluded, the danger of overpressurization of the nondependent lung is minimized. In addition, the CPAP system is calibrated from 1 to 10 cm H2 O pressure, which permits accurate delivery of very low levels of CPAP. This may be desirable with extremely compliant lungs or where maximum surgical exposure is required. This small, lightweight CPAP device can be included within the DLT package (the DLT is available with or without the CPAP device), is extremely easy to use, and is preassembled, cost-effective, sterile, reliable, and disposable. Perhaps its most important attribute is that a preassembled device is obviously more readily available than devices that require a search for spare parts, and the commercial product has built-in quality assurance that cannot be duplicated by individual remedies.

FIGURE 48–32 The Mallinckrodt Broncho-Cath CPAP System. (A) Photograph of entire CPAP system connected to a double-lumen tube. (B) Schematic of the entire CPAP system. (C) Close-up of the CPAP-generating restrictive mechanism. (Photography courtesy of Mallinckrodt Medical, Inc., St. Louis, MO.)

Differential Lung PEEP/CPAP

In theory and from the preceding considerations, it appears that the ideal way to improve oxygenation during one-lung ventilation is the application of differential lung PEEP/CPAP (see Fig. 48–30D) (see the following section for the step-by-step approach). In this situation, the ventilated (dependent) lung is given PEEP in the usual conventional manner in an effort to improve ventilated lung volume and V/Q relationships. Simultaneously, the nonventilated (nondependent) lung receives CPAP in an attempt to improve oxygenation of the blood perfusing this lung. Therefore, with differential lung PEEP/CPAP, it does not matter where the blood flow goes nearly as much as during simple one-lung ventilation, because wherever it goes (to either ventilated or nonventilated lung), it has at least some chance to participate in gas exchange with alveoli that are expanded with oxygen. In indirect support of this contention, arterial oxygenation has been increased significantly in patients during thoracotomy in the LDP (using two-lung ventilation) when PEEP has been added to the ventilated dependent lung, while the nondependent lung was also able to participate in gas exchange by virtue of being ventilated at zero end-expiratory pressure (ZEEP). 237 In direct support of this contention, in patients undergoing thoracotomy and one-lung ventilation, arterial oxygenation was unchanged by the application of 10 cm H_2O dependent lung PEEP alone (consistent with an equal positive/negative effect trade-off), was significantly improved by 10 cm H_2O nondependent lung CPAP alone, and was further and even more significantly increased by use of 10 cm H_2O nondependent lung CPAP and 10 cm H_2O dependent lung PEEP together (differential lung PEEP/CPAP ventilation). 385, 389 The use of 10 cm H_2O nondependent lung CPAP together with 10 cm H_2O dependent lung PEEP in patients caused only small, clinically insignificant hemodynamic effects. 385, 389

There are multiple reports of significant increases in oxygenation obtained with the application of differential lung ventilation and PEEP (either PEEP/PEEP, PEEP/CPAP, or CPAP/CPAP) through DLTs to intensive care unit patients with acute respiratory failure due to predominantly unilateral lung disease. 397 In all cases conventional two-lung therapy (mechanical ventilation, PEEP, CPAP) had been administered via a standard single-lumen tube and either failed to improve or actually decreased oxygenation. In these patients the single-lumen tube was replaced with a DLT. In most cases the amount of PEEP initially administered to each lung was inversely proportional to the compliance of each lung; ideally, this PEEP arrangement should result in equal FRC in each lung. In some cases, the amount of PEEP that each lung received was later adjusted and titrated in an effort to find a differential lung PEEP combination that resulted in the lowest right-to-left transpulmonary shunt. This treatment modality for severe unilateral lung disease is discussed more fully in Chapter 72.

Recommended Combined Conventional and Differential Lung Management of One-Lung Ventilation

Figure 48–33 summarizes the recommended plan for obtaining satisfactory arterial oxygenation during one-lung anesthesia. Two-lung ventilation is maintained for as long as possible (usually until the pleura is opened). When one-lung ventilation is commenced, a tidal volume of 10 mL/kg is used, and the respiratory rate is adjusted so that $PaCO_2$ equals 40 mm Hg. A high inspired oxygen concentration (FIO_2 of 0.8–1.0) should be used, and SaO_2 should be monitored continuously.
FIGURE 48–33 An overall one-lung ventilation plan. FIO_2, inspired oxygen concentration; TV, tidal volume; RR, respiratory rate; PEEP, positive end-expiratory pressure; CPAP, continuous positive airway pressure; ASAP, as soon as possible.

If hypoxemia is present after this initial conventional approach, two major causes of hypoxemia, namely malposition of the DLT and poor hemodynamic status, must be ruled out. Proper tube position should be confirmed via fiberoptic bronchoscopy. If the DLT is correctly positioned and the hemodynamic status is satisfactory, simple tidal volume and respiratory rate adjustments should be made. 302 For example, if the tidal ventilation is thought to be too high, it should be decreased, and if the tidal ventilation is thought to be too low, it should be increased. If these simple maneuvers do not quickly resolve the problem, the studies of selective nondependent lung CPAP 385, 388, 389, 390, 391, 392, 393, 394, 395 and differential lung PEEP 397 dictate that the next treatment should be to apply 5 to 10 cm H_2O of CPAP to the nondependent lung. Nondependent lung CPAP should be applied during the deflation phase of a large tidal volume breath to overcome critical opening pressures in the atelectatic lung. If oxygenation does not improve with

nondependent lung CPAP (as it does in the large majority of cases), 5 to 10 cm H2 O of PEEP should then be applied to the ventilated dependent lung. If dependent lung PEEP does not improve oxygenation, nondependent lung CPAP should be increased to 10 to 15 cm H2 O while the dependent lung is maintained at 5 to 10 cm H2 O of PEEP. If arterial oxygenation is still not satisfactory, the nondependent lung CPAP level should be matched with an equal amount of dependent lung PEEP. In this way, a differential lung PEEP/CPAP search for the maximum compliance and a minimum right-to-left transpulmonary shunt are made in an attempt to find the optimal end-expiratory pressure for each lung and the patient as a whole.

If severe hypoxemia persists after the application of differential lung PEEP/CPAP (which would be extremely rare), it should be remembered that the nondependent lung may be intermittently ventilated with positive pressure with oxygen (see Fig. 48–33). Finally, most of the V/Q imbalance is eliminated during a pneumonectomy by tightening a ligature around the nonventilated lung pulmonary artery as early as possible, thus directly eliminating all shunt flow through the nonventilated lung (see Fig. 48–33). Indeed, clamping the pulmonary artery to a collapsed lung functionally resects the entire lung, and the PaO2 is restored to a level not significantly different from a two-lung ventilation or postpneumonectomy one-lung ventilation value.

Because nondependent lung CPAP has been shown to relieve hypoxemia consistently and reliably during one-lung ventilation, 388, 389 its routine use to prevent hypoxemia during thoracic surgery using DLTs should be considered. Low levels of CPAP used in this manner have not compromised surgical conditions and have occasionally improved surgical exposure by facilitating the identification of intralobar planes. 385, 388, 389, 395

High-Frequency Ventilation Management of Thoracic Surgery

Conventional intermittent positive-pressure ventilation delivers relatively large tidal volumes (10–15 mL/kg) at respiratory rates usually less than 30 breaths/min. In contrast, high-frequency ventilation (HFV) delivers very small tidal volumes (<2 mL/kg) at rates between 60 and 2,400 breaths/min. Because HFV uses much smaller tidal volumes (and much lower peak inspiratory airway pressures), which can be delivered through very small airway catheters, it may be uniquely useful in facilitating the performance of thoracic surgery in the following three ways.

High-frequency ventilation is an umbrella term encompassing different delivery systems and respiratory rate ranges. Current methods used to provide HFV are quite diverse, but by using the criteria of ventilation rate and type of gas delivery mechanism, it is possible to separate HFV into three general categories (Table 48–16). With regard to oxygenation, all forms of HFV should be regarded as a method of achieving PEEP (or above ambient airway pressure) and thereby minimizing shunt without the necessity of imposing a large volume (and pressure) excursion on top of this to eliminate carbon dioxide. It is conceptually similar to the approach of holding the lung slightly to moderately distended with an increased mean airway pressure and thereby minimizing shunt while removing carbon dioxide by an extracorporeal membrane. 398, 399

TABLE 48–16. Characteristics of the Three Types of High-Frequency Ventilation

Use in Major Conducting Airway Surgery

The most important advantage of HFV in thoracic surgery is that the small rapid tidal volumes may be delivered through small airway tubes; thus, if a major conducting airway (trachea, carinal area, main stem bronchus) has to be divided, the transit of a small airway tube through the surgical field causes much less interference with surgery than the passage of a large standard or double-lumen endotracheal tube. The small airway catheters present the surgeon with a relatively unobstructed, accessible circumference of trachea and bronchus, so that the ends of a divided airway can be properly aligned for the construction of an unstressed and airtight anastomosis. Both high-frequency positive pressure ventilation (HFPPV) and high-frequency jet ventilation (HFJV) have been used successfully with small airway catheters for several different types of airway surgery. 400 In contradistinction to these findings with HFPPV, high-frequency oscillatory ventilation delivered through a standard single-lumen tube has been found to cause changes in the diameter of large airways and to produce a large “mediastinal bounce” with each oscillation, which “made surgery on the major airways and around the hilum and mediastinal structures almost impossible.” 401 With all types of HFV for airway surgery, the logistics of small catheter placement and securement are formidable, and suctioning the airway and distal lung may be problematic.

Use in Bronchopleural Fistula

The second proposed advantage of HFV for thoracic surgery is that the lower inspiratory pressures and tidal volumes may result in a smaller gas leak through pathologic low-resistance pathways such as bronchopleural fistulas and tracheobronchial disruptions. Consequently, air leaks (loss of tidal volume) and mediastinal and interstitial emphysema may be minimized with this form of ventilatory treatment, and HFV has been successfully used in the treatment of major bronchopleural fistulas and tracheobronchial disruptions. 400 In most of the bronchopleural fistula cases, $PaCO_2$ was unacceptably high despite a high minute ventilation on volume-controlled intermittent positive-pressure ventilation, whereas HFJV restored normocarbia. However, it appears that the reduction in air leak flow through the fistula is directly related to reduced airway pressure with HFV as compared with conventional mechanical ventilation; when peak and mean tracheal pressures are decreased by HFV, fistula leak is decreased, and when peak and mean tracheal pressures are increased by HFV, fistula flow increases. Thus, in a report by Albelda et al 402 of seven consecutive patients with an average bronchopleural fistula leak greater than 5 L/min, HFJV (125–150/min) caused only two patients to have clinically important decreases in airway pressure and air leak fistula flow, and none had significant improvement in gas exchange. The authors recommend measurement of tracheal pressures to predict what is happening to air leak fistula flow. 402 Thus, it is likely that investigations in the use of HFV to treat patients with pathologic low-resistance airway pathways will continue.

Use in Minimizing Movement of the Operative Field

The third proposed advantage of HFV for thoracic surgery is to minimize the tidal movement of the operative field; theoretically, the lower peak inspiratory pressures and tidal volumes of HFV should result in much smaller inflation and deflation movements of the lung. Therefore, HFV of the nondependent lung might provide a relatively “quiet” operative lung field, and HFV of the dependent lung could contribute to the quiet lung field by providing a minimally moving mediastinum. 400 Unfortunately, in no report was the use of HFV compared with conventional one-lung ventilation, with or without nondependent lung CPAP, in terms of operating conditions and efficiency of gas exchange.

The studies of selective nondependent lung CPAP indicate that delivery of any amount of oxygen to the nondependent lung alveolar space will cause some oxygen uptake from the nondependent lung and decrease the right-to-left shunt in this lung. Consequently, it is not surprising that selective HFV of the nondependent lung, while the dependent lung is ventilated with conventional intermittent positive-pressure breathing, increases PaO_2 compared with simple collapse of the nondependent lung and conventional mechanical ventilation of the dependent lung. 403 However, because the same increase in arterial oxygenation and operative conditions may be obtained by selective nondependent lung CPAP with much simpler equipment than HFV apparatus, it is more logical to use selective nondependent lung CPAP than HFV to improve arterial oxygenation and operative conditions during one-lung ventilation.

In one report the dependent lung was selectively ventilated with HFV while the operated lung was completely collapsed. 404 As compared with selective dependent lung intermittent positive-pressure ventilation, selective dependent lung HFV significantly improved arterial oxygenation, presumably because of lower dependent lung PVR (due to either lower dependent lung airway pressures 405 or release of dependent lung vasodilator prostaglandins 406) and enhanced operating conditions (because of minimal mediastinal movements). However, the use of dependent lung HFV to minimize mediastinal and hilar movements must remain controversial in view of a 1986 report describing a mediastinal bounce with each oscillation, which made surgery around these structures nearly impossible. 401

In summary, in view of the limitations of HFV, its use as a routine procedure for all three possible thoracic surgery situations (airway surgery, bronchopleural fistulas, minimizing operative field movement) cannot be recommended.

Low-Flow Apneic Ventilation (Apneic Insufflation)

The need for an absolutely quiet surgical field for short periods often arises during a thoracotomy in which a standard endotracheal tube and two-lung ventilation are used. This can be accomplished relatively safely by using the principle of apneic mass-movement oxygenation. Indeed, this method gained considerable

popularity around the time DLTs were being introduced into clinical practice. If ventilation is stopped during the administration of 100 percent oxygen and the airway is left connected to a fresh gas supply, oxygen will be drawn into the lung by mass movement to replace the oxygen that crossed the alveolocapillary membrane. There is usually no difficulty in maintaining an adequate PaO2 (especially if 5–10 cm H2 O of CPAP is used) during at least 20 minutes of apneic mass-movement oxygenation.

If the flow of oxygen into the lungs is relatively low (<0.1 L/kg/min) almost all the carbon dioxide produced is retained, and the arterial carbon dioxide tension (PaCO2) rises approximately 6 mm Hg in the first minute because of the washin of venous blood into the arterial compartment (venous blood has a carbon dioxide tension 6 mm Hg higher than that of arterial blood) and then 3 to 4 mm Hg each minute thereafter because of normal carbon dioxide production. 407, 408 On the basis of these considerations, if a patient had normal carbon dioxide production, was hyperventilated to a PaCO2 of 30 mm Hg, and was then made apneic and had oxygen insufflated into the lungs with a low flow, the PaCO2 after 10 minutes of apnea would be 63 to 72 mm Hg. Indeed, one report describes a series of eight patients in whom apneic mass-movement oxygenation was used for 18 to 55 minutes after normal ventilation. 407 Although the lowest arterial saturation that resulted was 98 percent, the PaCO2 in the five patients in whom it was measured ranged from 103 to 250 mm Hg, and the pH ranged from 6.72 to 6.97. Although severe degrees of hypercapnia and respiratory acidosis may be well tolerated in some healthy patients, it would appear that the safe period of low-flow apneic oxygenation during thoracotomy would be well below 10 minutes. Although it has not been studied, low-flow apneic oxygenation should theoretically be able to be used when a DLT is used. In all cases in which this technique is used, arterial oxygen saturation monitoring via pulse oximetry is mandatory.

THE POSTOPERATIVE PERIOD

Early Serious Complications Specifically Related to Thoracic Surgery

Herniation of the Heart

An intrapericardial approach may be used for radical pneumonectomy to facilitate access to major vessels and allow a wider hilar dissection. This technique may result in a large pericardial defect that the surgeon is not able to close after the pneumonectomy is completed. There have been several reports of herniation of the heart 409 through the pericardial opening into the empty hemithorax. The herniation can be into either the right or left hemithorax, and herniation into either side can cause profound cardiac functional impairment; the mortality rate is 50 percent.

With herniation, the heart protrudes through the defect into one of the hemithoraces, potentially causing twisting of the superior vena cava (superior vena cava syndrome), inferior vena cava (cardiovascular collapse), distal trachea (wheezing), pulmonary veins (pulmonary edema), and pericardial constriction of the heart (myocardial ischemia and ventricular arrhythmia). These symptoms may occur individually or in various combinations.

In most reports herniation of the heart has occurred either immediately after the patient is turned from the LDP to the supine position (75%) or during the first few hours of postoperative mechanical ventilation. However, this complication can also occur as late as several days after surgery. Events that increase intrapleural pressure in the nonsurgical (ventilated) hemithorax or that decrease intrapleural pressure in the surgical (empty) hemithorax may predispose the patient to cardiac herniation. Placing the patient with the empty hemithorax in a dependent position allows the heart to be pulled by gravity into the empty hemithorax. Use of high levels of pressure and volume during mechanical ventilation of the remaining lung can push the heart into the empty hemithorax. Similarly, coughing can increase pleural pressure in the remaining lung and thereby promote displacement of the heart into the empty hemithorax. Conversely, inadvertently applying suction to a chest drain in the empty hemithorax can pull the heart through a pericardial defect (tension vacuthorax).

The diagnosis of cardiac herniation is made by the sudden appearance of catastrophic symptoms in a patient who had a radical pneumonectomy, the presence of a predisposing condition (see earlier), and a chest radiograph that shows the apex of the heart to be pointing to a lateral chest wall at a right angle. 410 A diagnosis of cardiac herniation almost always necessitates immediate reexploration.

Five conservative measures may improve cardiopulmonary function before and during transfer to the operating room (they are the reverse of the causes of herniation of the heart discussed previously), and they should be instituted as soon as the diagnosis is entertained. First, the patient should be positioned so that the ventilated nonsurgical side is in a dependent position and the empty surgical side is in a nondependent position. Gravity may return the heart and mediastinum to their normal anatomic positions. Even if the heart does not reenter the pericardium, the repositioning may decrease atriocaval kinking and increase cardiac output. Second, avoidance of high levels of pressure and volume in the ventilated lung may allow the return of the heart to the pericardium. Consequently, the tidal volume should be reduced, PEEP should be removed, and the respiratory rate should be increased slightly. Third, suction to the empty hemithorax should be discontinued. Fourth, pharmacologic support of the circulation should be provided as needed. Fifth, injecting 1 to 2 L of air into the surgical hemithorax may push the heart and mediastinum back to a normal anatomic position and perhaps the heart back into the pericardium. Success with this latter technique has been variable. 409

Pulmonary Torsion

Pulmonary torsion refers to rotation of the parenchyma on its bronchovascular pedicle and is thought to be due to increased mobility of a lobe (as may occur to the remaining lobes in one hemithorax after a lobectomy). Any patient with atelectasis or an expanding intrathoracic mass and perhaps with severe chest pain following thoracotomy should have lung torsion included in the differential diagnosis, which also includes intrathoracic bleeding and progressive atelectasis. Because torsion compromises the pulmonary vasculature (both arteries and veins) as well as the bronchi, prompt recognition and surgical intervention are required to avoid the attendant morbidity and mortality (i.e., infarction and gangrene). 410

A double-lumen endotracheal tube should be inserted before surgery if pulmonary torsion is suspected because during surgical correction of this condition (either completion pneumonectomy or untwisting and stabilization of the lobe to another lobe or chest wall), massive hemorrhage into the airways may occur owing to release of entrapped blood and continuing bleeding from necrotic lung tissue (i.e., vessels and airways), drowning the dependent lung and producing severe hypoxia or death. Other intraoperative therapeutic modalities that the anesthesiologist should consider include use of intravenous steroids to decrease any reactive pulmonary inflammation, use of PEEP to reexpand atelectatic lung tissue, and saline lavage for the removal of any excess blood that might form clots in the normal airways and thereby obstruct healthy lung parenchyma.

Major Hemorrhage

Postoperative bleeding that necessitates emergency thoracotomy 409 may occur in as many as 3 percent of all thoracotomy patients. 411 The mortality of major postoperative bleeding is approximately 23 percent. 411 The potential sources of major postoperative hemorrhage are bleeding from divided pulmonary arteries and veins due to slippage of surgical ligatures, diffuse bleeding from raw surfaces, and systemic arterial bleeding (bronchial and intercostal arteries).

The drainage from a patent chest tube is an excellent monitor of the amount of intrathoracic bleeding. In addition, the hematocrit of the usual fluid in the chest drainage is always less than 20 percent, and intrathoracic bleeding increases the hematocrit to some higher value. Significant hemorrhage, of course, is accompanied by the hemodynamic signs of hypovolemia. Significant hemorrhage cannot always be ruled out by the absence of chest tube drainage, because the chest tube may be blocked by clotted blood or otherwise obstructed. Nevertheless, the patient will still show signs and symptoms of hypovolemia and a shift in the mediastinum to the opposite side.

Bronchial Disruption

Development of a bronchopleural fistula is a serious complication of pulmonary resection and as recently as 1978 carried a mortality of 23 percent. 412 The symptoms and signs are variable and depend on the size of the communication, the presence or absence of a chest drain, and the presence of fluid of any sort within the pleural space.

Gross disruption of a bronchial stump is usually signaled by massive bubbling of gas through the chest tube drainage system, if present, tension pneumothorax if the chest tube is not present, hypoxemia, and

hypercapnia. Prompt resuscitation is essential and may need to include mechanical and differential or one-lung ventilation with a DLT and support of the circulation. A functioning chest tube, if not present, must be inserted immediately to evacuate the air (to prevent a tension pneumothorax) and fluid (to prevent further contamination of the opposite lung) from the pneumonectomy cavity; a needle can decompress the hemithorax while a chest tube is being inserted. The patient should be recumbent, with the normal side in a nondependent position. The stump must be closed surgically as soon as possible. 412

It should be remembered that a tension pneumothorax may occur in the contralateral side if very high inflation pressures are used to expand a previously collapsed lung (as commonly occurs for one-lung ventilation); the tension pneumothorax may become apparent only after closure of the chest. Other causes of acute postoperative contralateral pneumothorax relevant to thoracic surgery include damage to the contralateral pleura during surgery, puncture of the pleura during insertion of a central venous cannula or thoracic epidural needle, or damage to the bronchus by use of an endobronchial catheter.

Development of a chronic bronchopleural fistula may be expected to occur in 1 to 3 percent of pulmonary resection patients. 412 A chronic bronchopleural fistula most frequently becomes evident within the first 2 weeks after operation and may run a fulminating course characterized by sepsis, empyema, purulent sputum, and respiratory insufficiency. Factors that predispose to the formation of a chronic bronchopleural fistula after pulmonary resection are preoperative irradiation, infection, residual neoplasm at the site of the closure, and the presence of a long or avascular stump.

Respiratory Insufficiency

Acute respiratory insufficiency (within 30 days of resection) is probably the most common serious complication after pulmonary resection. Recent data from a large thoracic surgery service indicate an incidence of 4.4 percent after bronchial carcinoma resections, and in this study, the patients who developed acute respiratory failure had a mortality rate of 50 percent. 413 Mortality from respiratory failure after right-sided pneumonectomy is greater than after pneumonectomy on the left side because the remaining functioning lung is smaller. 413 In addition to the usual mechanism of respiratory failure (Ch. 72), pulmonary resection per se involves additional new mechanisms of hypoxemia and hypercarbia.

First, the remaining lung in either hemithorax may be edematous and/or soiled with blood. The lung that was dependent during surgery may be edematous and/or may have aspirated blood (if a DLT was not used) as a result of gravitational effects (in zones 3 and 4). The lung that was nondependent during surgery may be edematous and hemorrhagic because of surgical compression and trauma. Second, the size of the remaining pulmonary vascular bed is decreased; this decrease can lead to pulmonary edema. The treatment of respiratory insufficiency is discussed in detail in Chapter 72.

Unilateral Reexpansion (Intraoperative) Pulmonary Edema

Sudden evacuation of a chronic or subacute pneumothorax or pulmonary effusion may cause edema of the ipsilateral lung (reexpansion pulmonary edema). 414, 415 Still other reports describe an even more acute form of reexpansion pulmonary edema associated with lung reexpansion after only several hours of atelectasis, 416, 417 including reexpansion of the nondependent lung after one-lung ventilation to facilitate thoracic surgery 418, 419, 420, 421 and following correction of an inadvertent main stem bronchial intubation. 416, 422

Most clinical and experimental observations support increased pulmonary vascular permeability as a major factor in the development of reexpansion pulmonary edema. 423, 424, 425 All the above clinical literature indicates that the rate of reexpansion may be more important than the duration of collapse in the development of reexpansion pulmonary edema. 414, 420 Thus, other mechanisms of reexpansion pulmonary edema include generation of markedly negative intrathoracic pressure during reexpansion and increased pulmonary capillary pressure and flow on lung reexpansion. Therefore, the most important factor in preventing reexpansion pulmonary edema is to reexpand the lung slowly and in a gradual fashion. The treatment of established reexpansion pulmonary edema is mechanical ventilation, PEEP, restriction of fluids, and diuretics.

Right Heart Failure

Major pulmonary resection results in a decrease in the cross-sectional area of the pulmonary vasculature and an increase in right ventricular afterload, which in some patients can result in acute right heart failure. Although patients at risk for developing right heart failure after pulmonary resection can usually be identified preoperatively, right heart failure may also occur if additional stresses, such as infection and increased pulmonary blood flow and new active pulmonary vasoconstriction (such as that caused by hypoxia, acidosis, vasoactive amines and peptides), are placed on the right side of the heart.

The diagnosis of selective right heart failure is established when the right atrial pressure exceeds the left atrial pressure (i.e., the pulmonary artery wedge pressure) in the presence of an abnormally low cardiac output. In addition, pulmonary hypertension with a pulmonary artery diastolic-wedge pressure gradient will usually be present, along with the systemic signs of left heart failure (oliguria, decreased mentation, peripheral edema). The treatment of acute right heart failure follows the same principles used to treat left-sided failure: control heart rate, optimize preload and the inotropic state of the right ventricle, and reduce PVR (Chs. 49 and 51).

Right-to-Left Shunting Across a Patent Foramen Ovale

Many adults have a patent foramen ovale 409 ; at autopsy, the incidence of a probe-patent foramen ovale is highest at 34 percent during the first 3 decades of life and decreases to 20 percent by the 9th and 10th decades. There is normally no right-to-left shunting across the foramen ovale because left atrial pressure exceeds right atrial pressure; this pressure gradient keeps the one-way flap valve of the foramen ovale pressed against the opening, resulting in a functionally competent seal. If right atrial pressure exceeds left atrial pressure (as may be the result of any factor that increases PVR and right ventricular pressure), the one-way flap valve can open, and right-to-left shunting can occur through the newly opened foramen ovale. 409 Thus, new onset right-to-left shunting across a patent foramen ovale or atrial septal defect after pneumonectomy and lobectomy, as a cause of otherwise unexplained postoperative dyspnea and systemic oxygen desaturation, has occurred. 409 Use of contrast two-dimensional echocardiography combined with a saline bubble injection study is an excellent method of diagnosing this process, if suspected. Contrast angiography and dye dilution curve analysis are other available diagnostic methods. Nonsurgical treatment consists of decreasing right atrial preload and afterload; this procedure permits functional closure of the foramen ovale in the majority of patients.

Neural Injuries

During radical hilar dissection or excision of mediastinal tumors, phrenic, vagus, and recurrent laryngeal nerves may be injured accidentally or may be sacrificed deliberately. 409 Phrenic nerve injury causes respiratory embarrassment by a flail chest effect and also causes elevation of the ipsilateral hemidiaphragm. The diagnosis should be suspected in patients who have relatively clear chest radiographs, have adequate gas exchange, and cannot be weaned from the ventilator. The diagnosis can be confirmed by paradoxic movements of the diaphragm on fluoroscopy. Injury to the vagus nerve causes gastric and intestinal atony, which usually is not problematic in the first few postoperative days. Bilateral partial injury of the recurrent laryngeal nerve causes adductor spasm of the vocal cords, which, after extubation, may result in upper airway obstruction. This must be diagnosed promptly and treated with immediate reintubation and possible tracheotomy until the dysfunction is resolved.

Aside from clamping of the thoracic aorta with resultant ischemia to the spinal cord, there are two other causes of paraplegia after thoracotomy. First, damage to the spinal branches of intercostal arteries by dissection or diathermy at the posterior end of a rib may cause spinal cord ischemia. 427 This is most likely to occur with damage to the intercostal arteries of the left lower lobe. 428 Second, surgical dissection may create a communication between the epidural space and the pleural cavity. Clotted blood, or blood that can later clot, can then enter the epidural space and cause postoperative spinal cord compression and ischemia. 429 Similarly, expansion of hemostatic material (such as Surgisil) may result in cord injury.

Management of Postoperative Mechanical Ventilation

Most relatively healthy patients who have not had extensive thoracic operations may have their tracheas safely extubated in the operating room or shortly after arrival in the recovery room (Chs. 68, 71, and 72). However, patients with severe chronic pulmonary disease who undergo extensive thoracic operations require a period of postoperative mechanical respiratory support. Careful clinical assessment and judgment

are necessary for postoperative tracheal extubation of patients who are intermediate in terms of their respiratory health and the extent of their thoracic procedures. Precise criteria for continued intubation and mechanical ventilatory support are presented in Chapters 39, 68, and 72. Whenever doubt exists, we urge caution in removing mechanical ventilatory support too rapidly from these patients; it is safest to support patients until clinical and laboratory data clearly indicate that extubation is safe. The following briefly describes our current practice of mechanical ventilation, weaning, and extubation.

For most postoperative thoracotomy patients who require respiratory support, controlled mechanical ventilation is initiated after reintubation with a single-lumen tube, with a tidal volume equal to 12 mL/kg, an intermittent mandatory ventilation (IMV) rate so that the PaCO2 is 40 mm Hg (which usually requires an initial rate of 8 to 12 breaths/min), and with an inspired oxygen concentration of 60 to 100 percent. IMV used in this way is a full-support ventilation modality. The patients may breathe spontaneously, but at this initial ventilator stage, the spontaneous ventilation is not regarded as significantly contributing to the minute ventilation. The spontaneous ventilation with IMV may increase cardiac output and thereby decrease intravenous fluid requirements as well as eliminate the need for paralysis and heavy sedation. IMV may also facilitate the transition to weaning. Arterial blood gas values and chest radiographs are obtained shortly after the institution of mechanical ventilation in the recovery room.

An FIO2 greater than 0.5 is toxic to the lungs, 430 and the first and compelling goal is to reduce the FIO2 to less than 0.5 while maintaining an acceptable PaO2 (Table 48–17). The decrease in FIO2 below 50 percent is achieved by performing a dose (PEEP)-response (PaO2) titration. PEEP is progressively added (range 0 to 20 mm Hg) until the PaO2 is relatively normal for that patient (or at least >60 mm Hg) with an FIO2 less than 0.5. PEEP should be raised in increments of 2.5 to 5.0 cm H2 O. The patient should be allowed to stabilize with regard to respiratory mechanics (peak inspiratory pressure and compliance), hemodynamics (pulse rate and rhythm, systemic and central filling pressures, and cardiac and urine output), and gas exchange (arterial blood gases). Usually each PEEP increment requires 0.5 to 1 hour to complete. Consequently, the performance of a PEEP-FIO2/PaO2 titration may take several hours. Of course, as the PEEP titration is being performed, other respiratory care modalities (see below), such as suctioning, turning, and administration of antibiotics, are instituted. The advent of fiberoptic bronchoscopy has eliminated the need for use of “super-PEEP” (PEEP >20 to 25 cm H2 O) because regions of collapsed lung that are resistant to opening by PEEP are usually obvious on chest radiograph and can be suctioned and lavaged open under direct vision via a bronchoscope.

TABLE 48–17. Mechanical Ventilation and Weaning Plan

If a patient requires more than 10 cm H2 O of PEEP to have a PaO2 greater than 60 mm Hg with an FIO2 of less than 0.5, the V/Q mismatch is so great that it is illogical to make the patient spontaneously ventilate to any significant degree. In other words, lungs that have a PEEP requirement greater than 10 cm H2 O are inefficient to the point that the work of breathing is likely to be excessive, and the patient will fatigue and fail.

Thus, the second goal is to reduce the PEEP to less than 10 cm H2 O; of course, it is assumed that what was achieved before (FIO2 <0.5, acceptable PaO2) is retained. This second goal is achieved primarily by instituting an intensive and aggressive respiratory care regimen. Such a regimen has four components (Table 48–18), and all four proceed in parallel. Although there are no hard data to prove that any one of these respiratory care treatments is beneficial when used alone, there is a widespread consensus that when used together and in the context of the other care necessary in the postoperative management of respiratory failure, they produce a clearing of atelectasis, eradication of infection, and a decrease in the PEEP requirements. 431, 432

TABLE 48–18. Aggressive and Intensive Respiratory Care Regimen

Use of the respiratory care regimen outlined above usually allows the physician, within a few days, to decrease PEEP to less than 10 cm H2 O with continued maintenance of an adequate PaO2 along with a low FIO2. Once the PEEP requirement is less than 10 cm H2 O, no special attempt is made to reduce it further. Maintenance of some PEEP is desirable in terms of maintaining normal FRC and lung compliance and providing a subjective sensation of good lung expansion. The achievement of an FIO2 less than 0.5 and a PEEP level less than 10 cm H2 O with an acceptable PaO2 means that the blood and gas within the lungs are matched well enough that the lungs perform their basic gas exchange function in a reasonably efficient

manner. It now makes sense to have the patient do some of the work of breathing on his own, and the patient is now a candidate for weaning from mechanical ventilation.

The third goal is to retain what was achieved before (FIO2 less than 0.5, PEEP less than 10 cm H2 O, and acceptable PaO2) and to reduce the IMV rate to less than 1 breath/min. This weaning process is accomplished by a progressive decrease in the IMV rate, allowing a gradual transition rate from 100 to 0 percent ventilator dependence. The rapidity by which the IMV rate can be reduced is dictated by and directly proportional to the vital capacity and peak inspiratory force. Other simple indices of readiness for decreasing the IMV rate are shown in Table 48–19. When the decrease in IMV rate is begun, the head of the bed should be elevated as much as possible to reduce the pressure of the abdominal contents on the diaphragm and also to allow the patient to reestablish contact with the environment. During the transition, IMV provides a progressively decreasing amount of partial ventilatory support. 433

TABLE 48–19. Bedside Physiologic Parameters Conventionally and Commonly Used to Predict Success in Weaning From Ventilatory Support and Weakness of Each Test

As the IMV rate is decreased, the patient is monitored by the spontaneous respiratory rate, vital capacity, peak inspiratory force, and PaCO2. In addition, the patient should be asked to indicate whether a sensation of dyspnea is perceived. When the rate of IMV withdrawal is appropriate and is being tolerated, the patient's spontaneous respiratory rate remains constant or only slightly increases, the vital capacity and peak inspiratory force remain constant or improve, the arterial blood gas concentrations do not deteriorate, and the patient remains reasonably comfortable. Conversely, when the IMV withdrawal rate is too rapid and is not being tolerated, the spontaneous respiratory rate increases greatly (which is usually the first indication of tolerance), the vital capacity and peak inspiratory force decrease, and the arterial blood gas values deteriorate; the patient becomes hypertensive and tachycardiac, may have arrhythmias, and communicates a sensation of air hunger.

The patient is ready for extubation when PaO2 is adequate, with the FIO2 less than 0.5 (goal 1), the PEEP level less than 10 cm H2 O (goal 2), VC greater than 15 mL/kg, peak inspiratory force (PIF) greater than –25 cm H2 O, IMV less than or equal to 1 breath/min, spontaneous respiratory rate less than 20 to 30/min, and PaO2 approximately 40 mm Hg (goal 3), when no other major organ systems are in acute major failure or are unstable, and when the chest roentgenogram findings are reasonably equivalent to the premorbid findings or are rapidly improving and no new changes have appeared (such as infiltrates or pneumothorax). It should be emphasized that throughout this entire mechanical ventilation and weaning process analgesics and sedatives must be administered and titrated so that the patient is comfortable, is not bucking on the endotracheal tube, and is without active expiratory efforts.

The logic of the preceding approach is that the patient's lungs are first required to have the ventilation and perfusion well enough matched, as indicated by an adequate or reasonable PaO2 with an FIO2 less than 0.5 and a PEEP level less than 10 cm H2 O, that the lungs function as an efficient gas exchange organ. Next, the patient's lungs are required to sustain this efficient gas exchange without respirator aid as indicated by an adequate or reasonable vital capacity, PIF, spontaneous respiratory rate, respiratory rate to tidal volume ratio of less than 105 breaths/min/L which defines absence of rapid shallow breathing, 434 and PaCO2 on a low IMV rate. Finally, the process requires that no new complicating factor has occurred as indicated by lack of new chest radiographic findings, resolution of previous pathologic chest radiographic findings, and the absence of any significant disorder or complication in any of the other major organ systems.

During postextubation, the ideal respiratory care maneuver, a high alveolar inflating pressure is sustained for a relatively long time, and this can be achieved only with a large inhaled volume. Such deep breathing exercises with emphasis on achieving a sustained inspiration to total lung capacity can be accomplished by using the incentive spirometer.

In addition, a variety of other techniques can help to minimize postoperative pulmonary complications. Both upright position in bed and early ambulation increase FRC and help restore a favorable FRC–closing volume relationship. Percussion and postural drainage aid in the mobilization of secretions in patients with chronic bronchitis. Continuing the administration of bronchodilating drugs or steroids that were given preoperatively is important in continuing to keep reactive airways quiescent.

Management of Postoperative Pain

The treatment of pain after thoracotomy (Ch. 69) is important not only to ensure patient comfort, but also to minimize pulmonary complications by enabling patients to breathe normally (without active exhalation and/or splinting) and deeply (so that they can cough) and to ambulate. Normal and deep breathing requires stretching of the skin incision, which is painful. Postoperative patients normally try to prevent stretching of the skin incision by contracting their expiratory muscles (splinting), thus limiting the stretch on the incision during inspiration, and by actively exhaling, thus rapidly diminishing whatever stretch was caused by inspiration. Failure to inspire deeply before a forceful exhalation results in an ineffective cough. Splinting, active exhalation, and failure to cough promote retention of secretions, airway closure, and atelectasis.

Two very efficacious methods of post-thoracotomy pain treatment have become widely used and are considered by many to be the methods of choice. They are cryoanalgesia and administration of epidural narcotics. In addition, interpleural regional analgesia has recently been introduced as an alternative method for treating pain after thoracotomy. This section discusses only these three methods.

Cryoanalgesia

Extremely long-lasting intercostal nerve block may be obtained by intercostal nerve freezing (cryoanalgesia). 435, 436, 437, 438, 439, 440, 441, 442, 443, 444, 445 Direct application of an ice ball to the nerve causes degeneration of nerve axons without damage to the support structure of the nerve (the neurolemma), thereby reversibly disrupting nerve activity. Thus, intraneural and perineural connective tissue are preserved, providing a scaffolding for regenerating capillaries, axons, and Schwann cells. 442 The area of anesthesia is along the dermatomes treated. During the 2 to 3 weeks after nerve freezing, nerve structure and function begin to recover in parallel, and by 1 to 3 months after freezing, they usually are fully restored, without untoward sequelae (neuritis or neuroma formation). The numbness that persists during this period is not especially bothersome. Young women, however, have admitted to some distress during the period of axonal regeneration due to loss of sensation in the nipple area if the fifth and higher intercostal nerves have been frozen. 441

Currently used cryoprobes are about the size of a pen; the core of the instrument has an exit port that permits rapid expansion of a gas (usually nitrous oxide). The rapid expansion of gas cools a surrounding metal sheath. Because the metal sheath is in contact with a fluid, an ice ball (temperature –60°C) forms at the tip of the cryoprobe. The intercostal nerves are approached from within the chest, because the probe has to pierce the parietal pleura to reach the intercostal nerves in the subcostal groove. This is easily accomplished by the surgeon‘s lifting the nerve out of the groove with a small nerve hook. The cryoprobe is applied directly to the nerves as far posteriorly as possible from within the chest at the level of the incision plus two or three interspaces above and below this level, just before closure of the thoracotomy wound. The cryoprobe is activated so that the center of the resultant ice ball encapsulates the nerve. During the freeze, nerve tissue temperature is approximately –20°C. 441 If 2 to 3 mm of tissue remains between the tip of the cryoprobe and the nerve to be blocked, the nerve will not become adequately frozen, and nerve function might not be totally eliminated. If the pleura is thickened, local peeling of the pleura is advisable. 441 Usually two 30-second freeze cycles, which are separated by a 5-second thaw period, are applied to each of the nerves selected. However, recent experience with one 30-second freeze exposure has resulted in no loss of postoperative pain control with a significant reduction in the period of numbness (from 3.0 to 1.2 months). 441

The use of cryoanalgesia in this way has effectively reduced postoperative narcotic requirements and improved pulmonary function. 434, 437, 438, 439 Cryoanalgesia patients have had moderate pain at the end of the first postoperative day and only slight pain after that (as compared with patients receiving intravenous narcotics, who experience moderate to severe pain at 24 hours). Most of the pain experienced by the cryoanalgesia patients has not been incisional discomfort, but rather shoulder or arm pain secondary to chest tube irritation of the pleura. Further advantage of the cryoanalgesic method can be gained if the chest tubes can be placed within an intercostal space whose nerves have been subjected to cryoanalgesia. 441 Once the chest tubes are removed, usually on the second or third postoperative day, pain in the cryoanalgesia patients has become barely noticeable. Consequently, these patients are more able to cooperate with the postoperative pulmonary physiotherapy maneuvers described in Chapter 72. Return of sensation occurs in most patients by the thirtieth postoperative day. Long-term follow-up for 6 months has shown that the procedure is associated with a certain worrisome incidence of dysesthesias and intercostal muscle paralysis, 443 but it is impossible to distinguish between injury to the nerves and muscle during

surgery (which does occur [e.g., 44% of patients in one long-term follow-up study had persistent post-thoracotomy pain 444]) and injury due to the cryoprobe 445 as the cause of these problems.

Because cryoanalgesia has been shown to reliably and effectively relieve pain and to allow significant improvements in postoperative pulmonary function, it has become an important addition to the modalities used in the treatment of post-thoracotomy pain. Cryoanalgesia may be a treatment of choice in thoracic pain situations that are expected to last a long time (e.g., pain from chest trauma) and to limit respiratory function significantly.

Epidural Narcotic Administration

Treatment of pain after thoracic surgery with epidural narcotics (Ch. 69) has several important advantages. First, there is no sympathetic blockade and neither motor nor sensory loss; second, success in relieving pain is usually predictable; and third, the duration of pain relief is generally much longer and the quality of pain relief better than those that result from using parenteral narcotics. 446, 447, 448, 449, 450, 451, 452

Epidural opioids have had a moderately extensive trial in relief of post-thoracotomy pain. The epidural catheter should be placed before the induction of general anesthesia and checked for correct position by using a small dose of local anesthetic. Alternatively, but less preferably, it may be placed in the lumbar region postoperatively before emergence from anesthesia, while the patient is still in the LDP. The initial injection can be made in the operating room (most usual), recovery room, or intensive care unit. Several important clinical points have emerged from the post-thoracotomy epidural opioid analgesia experience.

First, although the thoracic epidural route has been used, the procedure has risks (primarily dural puncture and spinal cord damage), and the lumbar area for catheter insertion has been found to be equally satisfactory for pain relief if a slightly higher dose of morphine and adequate diluent volumes are used. 448, 451, 452, 453, 454, 455, 456, 457 Morphine can be used with a lumbar epidural injection for the relief of thoracic pain because it is lipophobic and will therefore remain in, and have time to spread throughout, the epidural and cerebrospinal fluid (CSF) spaces. For relief of thoracic pain, instillation of 6 mg of morphine in 10 to 15 mL of diluent (preservative-free normal saline) in the lumbar epidural region has been used successfully. 451, 455 Although studied to a much smaller degree, fentanyl, 458, 459 methadone, 460 hydromorphone, 461 and nalbuphine 462 may also be used by either the thoracic or the lumbar epidural route. Not surprisingly, the lumbar route requires a greater diluent volume to push the narcotic into a wider distribution mechanically, because these lipophilic narcotics bind too quickly to the spinal cord at, and a few dermatomes above and below, the segmental level of introduction. Because the lumbar route is safer than the thoracic route and because fentanyl has not been associated with respiratory depression, use of the lumbar epidural route with fentanyl is considered by many to be the post-thoracotomy pain management technique of choice. It should be noted that low-volume thoracic instillation of fentanyl may be safer than large-volume lumbar morphine and is therefore also a widely used technique. However, there remains a wide selection of drugs (and dosages) for use by the lumbar and thoracic epidural routes (Table 48–20).

TABLE 48–20. Epidural Narcotics That Have Been Used for Postoperative Thoracic Surgery Pain Management: Routes, Dosages, and Pharmacodynamics

Second, epidural morphine 456 and fentanyl 453 have been reported to be effective after thoracic trauma. In patients with multiple fractured ribs, epidural morphine resulted in well over 6 hours of analgesia with each dose. Alternatively, continuous epidural infusion of fentanyl (1–2 ?g/kg/h after a 1- to 2-?g/kg bolus) is a good choice in the thoracic trauma situation (as well as for post-thoracotomy pain) because it has a decreased likelihood of causing respiratory depression. Because there is no sympathetic blockade with epidural opioids, there is no need for the patient to lie supine for top-up doses, and patients with some degree of hypovolemia may be managed more safely than with epidural local anesthetic administration.

Third, in the post-thoracotomy pain relief experience (summarized in Table 48–20), there have been relatively few significant side effects. Urinary retention is generally not a problem because most patients have a bladder catheter in place.

Fourth, catheters have been left *in situ* and used for as long as 5 days without development of tolerance. 463

Fifth, pain relief failures have usually been due to improper catheter localization because no pain relief could be demonstrated after injection of local anesthetics. 463

Sixth, the expected increase in the mean post-thoracotomy expiration flow rate, forced vital capacity, FRC, and ability to tolerate respiratory care maneuvers after epidural pain relief have been demonstrated. 463 Even larger increases in ventilatory capacity have been demonstrated in the much larger experience with epidural pain relief after upper abdominal surgery. 464 These increases are generally greater than or equal to those obtained by parenteral narcotic or epidural local anesthetic administration.

Finally, epidural narcotics have been used to treat intractable pain due to thoracic tumors. 465 Lower dosage is required, and longer duration of analgesia with each intermittent dose is obtained for intractable cancer pain as compared with acute postoperative pain. 465 However, although results have been satisfactory after the first few series of injections for intractable pain, tolerance has developed, and dosages have had to be increased because of continuous bathing of the spinal cord with a CSF concentration of opioid. 453 For treatment of chronic pain, permanent catheters have been implanted in the subarachnoid and epidural space and connected to reservoirs or perfusion pumps. 453 These systems have been found to be effective, but again tachyphylaxis has been observed. 453

Interpleural Regional Analgesia

Interpleural regional analgesia has recently been introduced for the treatment of pain due to a number of conditions, including rib fractures, 466 pancreatitis, 467 and postoperative pain from mastectomy, cholecystectomy, and renal operations. 468, 469, 470 In addition, this technique has been evaluated in patients who have undergone thoracic procedures. 471, 472, 473, 474

Interpleural regional analgesia is the percutaneous introduction of a catheter (usually an epidural catheter) into the thoracic cage between the parietal and visceral pleura. Because the catheter tip is located, and a local anesthetic is deposited, between the two layers of the pleura, this technique is better termed *interpleural* rather than intrapleural regional analgesia. 475

Bupivacaine, in concentrations ranging from 0.25 to 0.5 percent, usually containing epinephrine, is the local anesthetic that has been studied most often. Analgesia is thought to occur as a result of (1) diffusion of local anesthetic through the parietal pleura and the innermost intercostal muscles to reach the intercostal nerves where blockage occurs, (2) blockage of the intrathoracic sympathetic chain, and (3) direct action of local anesthetic on nerve endings within the pleura. A more detailed discussion of this technique can be found in Chapters 43 and 69.

For thoracic surgery patients, the catheter is usually placed intraoperatively by the surgeon at an interspace just below the level of the incision. 471, 472, 473 Alternatively, and less commonly, a chest tube can be used for the instillation of local anesthetic. 474 The chest tube should be clamped for 5 to 15 minutes after each administration of bupivacaine. If the chest tube is not clamped, approximately 30 to 40 percent of any administered dose of local anesthetic will be lost via the thoracostomy tube. 476 The patient should be kept in a supine position during the injection and for the next 10 to 15 minutes.

Most of the many studies have had good results with interpleural analgesia 466, 467, 468, 469, 470, 471, 472, 473, 474, 475 (good pain relief and/or increased pulmonary function and/or decreased narcotic requirements). However, several reports have been only mildly positive 476 or frankly negative. 472, 477, 478, 479 The most important reason for failure to achieve adequate analgesia after interpleural administration of local anesthetics has been failure to clamp the chest tube before administration causing the local anesthetic to be suctioned out of the pleural space into the chest tube. Indeed, when one group 472 changed technique to include clamping of the chest tube, 480 negative results were converted to positive results. Other reasons for failure of the technique are posterior placement of local anesthetic, preventing adequate analgesia for anterior thoracotomy, dilution of local anesthetic by pleural effusion, blood, and/or infected fluid; loculation of local anesthetic by adhesions, fibrosis, and/or infection; and loss of local anesthetic through a bronchopleural fistula.

A recent review covering a total of 703 cases has detailed the complications of interpleural analgesics. 481 Pneumothorax was the most frequently registered complication (2.0%), followed by signs of systemic toxicity (1.2% [in one patient seizures were thought to be due to rapid uptake because of the presence of a highly

inflamed pleura 482]) and pleural effusion (0.42%). Horner syndrome, pleural infections, and catheter rupture have also been reported. 481

Because the initial results of treating pain due to thoracotomy with interpleural regional analgesia have been mixed at best, the routine use of this technique cannot be recommended at this time. Its ultimate role in the treatment of post-thoracotomy pain will be determined by the results of further studies.

ANESTHESIA FOR SPECIAL THORACIC SURGERY PROCEDURES

Special Diagnostic Procedures

Mediastinoscopy

Indications

Mediastinoscopy is commonly performed before thoracotomy to establish a diagnosis and/or to determine the resectability of a lung carcinoma. After a suprasternal notch incision, a tunnel is created (through the pretracheal fascia) by blunt dissection along the anterior and lateral walls of the trachea into the mediastinum, behind (posterior to) the aortic arch down to the subcarinal area (Fig. 48–34). This procedure allows for direct inspection and biopsy of the superior mediastinal lymph nodes, which lie posterior to the aortic arch (the anterior and lateral para-mainstem bronchial, anterior subcarinal, anterior, and lateral paratracheal lymph nodes) (see Fig. 48–34). Tumors of the thymus and anterior mediastinum are not examined by the usual diagnostic mediastinoscopic approach because they are anterior to the great vessels; thus, an anterior mediastinotomy is required to examine this area (this procedure uses a parasternal second innerspace incision and is a less complicated procedure than mediastinoscopy). Previous mediastinoscopy is a near absolute contraindication to a repeat procedure because scarring eliminates the plane of dissection. Relative contraindications to mediastinoscopy include superior vena cava syndrome, severe tracheal deviation, cerebrovascular disease, and thoracic aortic aneurysm. 483 With the advent of CT and magnetic resonance imaging, the role of mediastinoscopy may diminish considerably in the future because negative scans may obviate the need for invasive staging.

FIGURE 48–34 Schematic diagram showing placement of a mediastinoscope into the superior mediastinum. The mediastinoscope passes anterior to the trachea but behind the thoracic aorta. This location allows for sampling of anterior and lateral para-mainstem bronchial lymph nodes, anterior subcarinal lymph nodes, and anterior and lateral paratracheal lymph nodes. Anatomic structures that can be compressed by the mediastinoscope (see areas marked by *) and that can cause major complications are the thoracic aorta (rupture, reflex bradycardia), innominate artery (decreased right carotid blood flow can cause cerebrovascular symptoms, and decreased right subclavian flow can cause loss of right radial pulse), trachea (inability to ventilate, stimulus to cough), and vena cava (risk of hemorrhage with superior vena cava syndrome).

Anesthetic Technique

In addition to the usual preanesthetic evaluation of patients, one should specifically look for the signs and symptoms of the relative contraindications to mediastinoscopy, such as obstruction or distortion of the upper airway and superior vena caval obstruction, signs and symptoms of impaired cerebral circulation (which may be compounded during mediastinoscopy by compression of the innominate artery), and evidence of the myasthenic syndrome caused by lung carcinoma. Because there is the potential risk of hemorrhage, a large-bore intravenous catheter should be inserted, and blood should be immediately available during the procedure. If the superior vena cava is obstructed, a lower extremity intravenous catheter is mandatory.

Although mediastinoscopy can be performed under local anesthesia, 484, 485 general anesthesia with controlled positive-pressure ventilation is preferred because it allows the surgeon more flexibility in the dissection, minimizes the potential for air embolus (see later), and facilitates management of major complications such as massive hemorrhage. 486 However, local anesthesia may be considered for patients with active cerebrovascular disease to continuously monitor cerebral function in the awake state.

After the intravenous induction of general anesthesia and paralysis with either succinylcholine, atracurium, or vecuronium, lidocaine is sprayed directly on the trachea and given intravenously to minimize coughing during orotracheal intubation and the procedure itself. A nonkinking tube should be considered in cases in

which tracheomalacia is a possibility. Neuromuscular blockade is also used during the procedure to prevent coughing, venous engorgement from straining, and movement during the procedure. The head-up position minimizes venous engorgement but maximizes the potential for venous air embolism. During mediastinoscopy the anesthesiologist's attention is primarily focused on detecting the occurrence of the complications of the procedure. This is performed by monitoring the right upper extremity blood pressure (vessels most commonly compressed are the innominate and the right subclavian and carotid arteries) and by observing for reflex bradycardia (compression of the aorta), arrhythmias (mechanical stimulation of the aorta), hypovolemia, tension pneumothorax, and compression of trachea (see later). Blood pressure and oxygen saturation should be measured in the left arm; if an arterial line is placed, the right wrist may be preferable because it can immediately signal when compression of the innominate artery has occurred. Alternatively, a pulse oximeter probe placed on a finger of the right hand can be monitored by quantitative changes in signal strength, indicating a diminution of blood flow caused by the mediastinoscope. Immediately after the procedure patients can usually be extubated. Postoperatively patients should be nursed in a head-up position to minimize venous engorgement.

The anesthetic considerations for anterior mediastinotomy are similar to those for mediastinoscopy except that the incision is larger, the incidence of complications is lower because structures can be visualized and controlled more readily, the position of the head is unimportant, and the blood pressure can be measured in either arm because there is little chance of compression of the innominate artery.

Complications

Although overall mortality from this procedure is low (0.1%), 487 and one institution has even undertaken to perform ambulatory (outpatient) mediastinoscopies in a hospital-based surgical suite, 488 serious complications can occur and the anesthesiologist must be prepared to diagnose and to treat them. In a series of 6,490 patients undergoing mediastinoscopy, the major complications reported (numbers of patients in parentheses) consisted of hemorrhage (48), pneumothorax (43), recurrent laryngeal nerve injury (22), infection (22), tumor implantation in the wound (8), phrenic nerve injury (3), esophageal injury (1), chylothorax (1), air embolism (1), and transient hemiparesis (1). 487 The overall complication rate in various studies has been 1.5 to 3.0 percent. 487, 488, 489, 490, 491, 492, 493, 494 At the time of occurrence, most of the complications listed required a specific anesthetic management response.

Significant (occasionally massive) hemorrhage has been the most frequent major problem encountered during mediastinoscopy. If it occurs, thoracotomy must be performed immediately. While preparations for median sternotomy are in progress, the surgeon should attempt to control hemorrhage by compressing the bleeding site with a sponge forceps or a small pack, although the relative inaccessibility of the operative field may make this maneuver difficult or ineffective. The anesthesiologist should (1) rapidly begin volume replacement thorough one (or more) large-bore intravenous cannulae that have been placed before the induction of anesthesia, (2) send for blood that was reserved for the patient preoperatively, (3) support the circulation pharmacologically until volume replacement is achieved, (4) ensure adequate oxygenation and ventilation, (5) administer atropine for reflex bradycardia from aortic compression (if it occurs), and (6) discontinue or reduce the dose of all anesthetic drugs until normovolemia is reestablished. Rarely, it may be necessary to induce deliberate hypotension to control bleeding in this setting. 490 If hemorrhage originates from a superior vena cava tear, volume replacement and drug treatment may be lost into the surgical field unless they are administered via a peripheral intravenous line placed rapidly in the lower extremity. 490

Pneumothorax is another relatively frequently encountered complication of mediastinoscopy. It is usually not apparent until the postoperative period, and the majority of patients do not require chest tube decompression. All patients should be monitored for signs of pneumothorax in the postoperative period and a chest radiograph obtained when doubt exists. Pneumothorax that occurs intraoperatively, as evidenced by increased peak inspiratory pressure, tracheal shift, distant breath sounds, hypotension, and cyanosis, requires immediate treatment by chest tube decompression. 495

When mediastinoscopy causes a recurrent laryngeal nerve injury (see Fig. 48–34), it is permanent in approximately 50 percent of patients. 487 If injury to the recurrent laryngeal nerve is suspected, the vocal cords should be visualized while the patient is spontaneously breathing (usually at the time of extubation). If the vocal cords are nonmoving and/or are in midline position, consideration has to be given to the problem of postoperative laryngeal obstruction.

During mediastinoscopy, the mediastinoscope tip is located intrathoracically and therefore directly exposed to pleural pressure. Venous air embolism (when venous bleeding is present) can occur much more easily if patients are breathing spontaneously because of the development of negative intrathoracic pressure during inspiration; therefore, controlled positive-pressure ventilation during this procedure minimizes the risk of air embolism.

As noted, the mediastinoscope can exert pressure against the innominate artery and cause diminished blood flow to the right carotid and right subclavian arteries (see Fig. 48–34). This phenomenon may be of special significance in patients with preexisting compromised cerebral circulation. Compression of the right carotid artery has been proposed as the cause of a left hemiparesis that occurred in one patient and subsequently cleared 48 hours after the procedure. 487 In another patient compression of the right subclavian artery caused the loss of the pulse and blood pressure in the right arm and was misdiagnosed as an intraoperative cardiac arrest. 496 In another study, blood pressure in the right arm was significantly decreased from 15 to 360 seconds in four of seven patients who underwent mediastinoscopy. 497 This last report, therefore, recommended that blood pressure be measured in the left arm and the right radial artery be continuously monitored by palpation or finger plethysmography during mediastinoscopy. A right radial arterial line, of course, would very sensitively and continuously monitor the occurrence of innominate or right subclavian artery compression. An oxygen saturation monitor would do this task less sensitively. Any decrease in the right radial artery pressure requires repositioning of the mediastinoscope, especially in patients with cerebral vascular insufficiency. Preventing excessive extension of the neck, which might contribute to pinching of neck vessels, is also important in this group of patients.

Autonomic reflexes may result from compression or stretching of the trachea, vagus nerve, or great vessels. Sudden changes in pulse and/or blood pressure during mediastinoscopy may initially be empirically treated by repositioning the mediastinoscope. Atropine is given for persistent bradycardia.

Thoracoscopy

Indications

Diagnostic thoracoscopy (pleuroscopy) permits an examination of the intrathoracic cavity and is most commonly performed to aid in the diagnosis of pleural and parenchymal disease, to help establish the staging of suspected neoplasms, and to determine the etiology of recurrent pleural effusions. 498, 499, 500, 501, 502 It is most often performed after thoracocentesis or closed-chest pleural or lung biopsy has been performed and a diagnosis has still not been established.

The development of endoscopic video systems and instrumentation stapling devices, dissectors, coagulators, autotyping sutures and the neodymium:yttrium-aluminum-garnet [Nd:YAG] laser has permitted therapeutic thoracoscopy for a wide variety of major thoracic procedures. These procedures include biopsy of many intrathoracic structures, peripheral wedge and sublobar resections, lobectomy, removal of mediastinal cysts, closure of persistent/or recurrent pneumothoraces and leaking blebs, pleurodesis, dorsal thoracic sympathectomy, drainage of spinal abscesses, resection of posterior mediastinal neurogenic tumor, retrieval of intrathoracic (pleural) foreign bodies, definitive treatment of a postpneumonectomy chylothorax, and facilitation of pericardectomy. 503, 504, 505, 506, 507, 508 Thoracoscopy is done by making a small incision in the lateral thoracic wall (usually at the level of the sixth intercostal space) and then introducing a thorascope, laparoscope, or mediastinoscope into the pleural cavity. The procedure allows for complete inspection of the pleural space of a hemithorax. Fluid and biopsy specimens can usually be obtained easily through the incision, although many surgeons prefer to use a separate incision for additional instruments such as biopsy forceps.

Anesthetic Technique

Thoracoscopy can be done with either local, regional, or general anesthesia with one-lung ventilation. Local anesthetic infiltration of the lateral thoracic wall and parietal pleura is the simplest way to provide anesthesia, 509 although some patients may experience considerable discomfort when this method is used. Partial collapse of the lung on the operated side occurs when air enters the pleural cavity. This allows for good visualization of the pleural space by the surgeon. It is both hazardous and unnecessary to insufflate gases under pressure into the hemithorax under examination to increase visualization of the pleural space. Surprisingly, even though many of these patients suffer from advanced pulmonary disease, changes in

PaO_2, $PaCO_2$, and cardiac rhythm are usually minimal during the procedure when it is performed under local anesthesia and the patient is breathing spontaneously. 510, 511 However, it is prudent to use a high FIO_2 to overcome the loss in lung volume due to the unavoidable pneumothorax.

Intercostal nerve blocks performed at the level of the incision and for two interspaces above and below may provide more complete analgesia for thoracoscopy, especially if they are placed far enough posteriorly to anesthetize the parietal pleura. The addition of an ipsilateral stellate ganglion block helps to prevent the cough reflex that is sometimes elicited during visualization and manipulation of the hilum.

When thoracoscopy is performed under general anesthesia, a DLT should be used. Positive-pressure ventilation seriously interferes with visualization of thoracic contents, and thoracoscopy is therefore a relatively strong indication for one-lung ventilation. If the procedure is short and the ipsilateral lung needs to be deflated for only a brief period, blood gases are not routinely monitored during the procedure. However, for patients with marginal pulmonary status in whom the period of one-lung ventilation lasts for more than a few minutes, the usual monitoring precautions should be taken.

Complications

Complications are rare during this simple procedure, although it is possible that any structure that the surgeon has to manipulate may be damaged. A few critically ill spontaneously breathing patients may experience impaired gas exchange during the procedure. Some air may transiently remain in the pleural space after the procedure, and this situation may require chest tube insertion.

Special Elective Procedures

Tracheal Resection

General Considerations

Tracheal resection is indicated, if technically feasible, in patients who have tracheal obstruction due to a primary tracheal tumor (the majority are carcinomas), prior tracheal trauma (e.g., stenosis due to prolonged intubation), congenital anomalies, and vascular lesions. For patients who have operable tumors, approximately 80 percent have a segmental resection (may include carina or larynx) with primary anastomosis, 10 percent have segmental resection with prosthetic reconstruction, and 10 percent have insertion of a T-tube stent. Adjuncts to surgical extirpation include preoperative and postoperative external radiation, internal radioactive seed radiation (transferred by endobronchial catheter or directly placed by thoracotomy), and preoperative laser debulking therapy.

Many previous technical limitations to the performance of tracheal surgery can now be overcome by careful preoperative delineation of the site and degree of obstruction, close intraoperative communication between the surgeon and anesthesiologist, improved anesthetic management techniques, and meticulous postoperative care. All these components contribute to the ability to provide adequate ventilation throughout the perioperative period. Although the results of this very complicated surgery on primary tracheal tumors depend on tumor cell type, location, and method of resection, it is generally accepted that in the few institutions that have a reasonable degree of surgical experience, a worthwhile survival rate can be achieved in most patients. This section discusses the care of patients undergoing tracheal resection and describes several different methods of airway management 512, 513, 514, 515, 516, 517, 518, 519, 520, 521, 522, 523, 524, 525, 526, 527, 528, 529, 530, 531, 532, 533, 534, 535, 536 (Chs. 39 and 63).

Anesthetic Considerations

Unless airway obstruction is imminent, pulmonary function should be routinely studied preoperatively. The presence of preoperative lung disease that is severe enough to indicate a need for postoperative ventilatory support is a relative contraindication to tracheal resection, because the trauma of positive airway pressure and an endotracheal tube cuff at the tracheal suture line may cause wound dehiscence. 515 Obtaining a history of position-dependent airway obstruction is important because the induction of anesthesia should be accomplished with those patients in a position that does not cause airway obstruction. Preoperative evaluation should also include tracheal tomograms, CT (to define the exact position of the lesion), bronchoscopy (usually deferred until the time of operation in order not to precipitate airway obstruction due

to edema or hemorrhage), flow-volume loops (upper airway obstructions have characteristic shapes to the loop—extrathoracic obstructions cause an inspiratory limb plateau, and intrathoracic obstructions cause an expiratory limb plateau), and arterial blood gas determinations.

During surgery, all patients should have an arterial catheter placed to facilitate analysis of arterial blood gases. It should be placed in the left radial artery because the innominate artery (which supplies the right radial artery) crosses the trachea and may be compressed during surgery. A variety of methods for providing adequate oxygenation and carbon dioxide elimination have been used during tracheal resection and are described in Table 48–21. These can be divided into five approaches: (1) standard orotracheal intubation, (2) insertion of a tube into the opened trachea distal to the area of resection, (3) HFJV through the stenotic area, (4) HFPPV, and (5) cardiopulmonary bypass.

TABLE 48–21. Approaches to Airway Management During Tracheal Resection

The first technique uses a standard but uncut long orotracheal tube, which is placed above the tracheal lesion after the induction of general anesthesia and is merely manipulated by the surgeon past (distal to) the area of stenosis or mass. 516, 518 Although this method is relatively easy to use, a large tube may traumatize the lesion and cause bleeding or dislodgement of tissue, resulting in further airway obstruction. Furthermore, the technique is limited to patients with relatively mild stenosis, and the presence of an endotracheal tube in the surgical field makes completing the tracheal anastomosis more difficult.

To overcome these problems, endotracheal or endobronchial tubes have been inserted into the opened trachea distal to the site of resection (second approach). 515, 519, 520, 521, 522, 523, 524, 525 With this second approach, initially either a small endotracheal tube is passed distal to the obstruction or a standard endotracheal tube is placed proximal to it (see Figs. 48–35A and 48–36A; Fig. 48–37A). All further tracheal and endobronchial intubations are performed with armored tubes passed into the airway, which is surgically opened distal to the lesion. The surgeon must have a complete set of sizes of endotracheal tubes to choose from because either mainstem bronchus or any lobar bronchus may need to be intubated.
FIGURE 48–37 Airway management and surgical procedure for resection of a carinal lesion. (A) Initial intubation above the lesion. (B) Left endobronchial intubation distal to the lesion after the left mainstem bronchus has been severed. (C) The trachea is anastomosed to the right mainstem bronchus. (D) The left endobronchial tube has been removed to allow for anastomosis between the trachea and left mainstem bronchus. Ventilation is accomplished via the original endotracheal tube. (Modified from Geffin et al515)
FIGURE 48–35 Airway management and surgical procedure for resection of a high tracheal lesion. (A) Initial intubation above the lesion. (B) Second endotracheal intubation distal to the lesion after the trachea has been opened. (C) Placement of sutures for the posterior anastomosis. (D) The second endotracheal tube has been removed, and the original endotracheal tube has been advanced distal to the anterior anastomosis. (Modified from Geffin et al515)
FIGURE 48–36 Airway management and surgical procedure for resection of a low tracheal lesion. (A) Initial intubation above the lesion. (B) Left endobronchial intubation distal to the lesion after the trachea has been opened. (C) Placement of sutures for the posterior anastomosis. (D) The endobronchial tube has been removed, and the original endotracheal tube has been advanced distal to the anterior anastomosis into an endobronchial position. (Modified from Geffin et al515)

With a high tracheal lesion, a cervical incision, possibly combined with a median sternotomy, provides adequate surgical exposure. An opening is made in the trachea distal to the area to be resected, and a sterile endotracheal tube is inserted by the surgeon into the distal trachea. 515, 519, 520 (see Fig. 48–35B). This second endotracheal tube is connected to a Y-piece and a second set of anesthetic hoses and handed off to the anesthesiologist in order to continue ventilation. After excision of the tracheal lesion and placement of the posterior tracheal sutures, the second (distal) endotracheal tube is removed from the trachea, the original (first) endotracheal tube is advanced past the anastomosis line and reconnected to the anesthetic circuit, and the anastomosis is completed Figs. 48–35A (see Fig. 48–35C and D).

With a low tracheal lesion, a right thoracotomy provides the necessary surgical exposure. If there is sufficient trachea distal to the area of resection, a Foley catheter with the tip cut off just distal to the balloon may be used as a single-lumen endotracheal tube. It is inserted by the surgeon and secured just above the carina, avoiding endobronchial intubation and the need for one-lung anesthesia. 521 Otherwise, if there is not enough distance between the tracheal lesion and carina to provide placement of even this homemade endotracheal tube, endobronchial intubation and one-lung ventilation are necessary 522, 523, 524 (see Fig.

48–36B). If oxygenation or ventilation is inadequate, it may be possible to decrease blood flow to the atelectatic lung by tightening reversible snares around the pulmonary artery of the nonventilated lung 515, 516; however, this maneuver may be technically difficult. An alternative technique is to pass a second endobronchial tube into the other bronchus to provide ventilation to both lungs 524 (see later). As with the high tracheal lesion, after the posterior anastomosis is completed, the endobronchial tube(s) is removed and the original endotracheal tube pushed past the site of resection; in this situation, however, it is likely that an endobronchial intubation may again be required to complete the anastomosis (see Fig. 48–36C and D).

Several methods have been described for managing the airway during carinal resection. 515, 516, 534 While the affected segment is being resected, left lung ventilation may be carried out via an endobronchial intubation of the left mainstem bronchus below this lesion 515 (see Fig. 48–37B). After the right mainstem bronchus and trachea have been reattached, the left endobronchial tube is removed, and the original endotracheal tube is advanced distal to the suture line. The right lung is then ventilated via this tube, while the left main bronchus is reanastomosed to the trachea at a different point of origin (see Fig. 48–37 C and D). Again, blood flow to the nonventilated lung can be reduced by tightening ties placed around the appropriate pulmonary artery. An alternate method of ventilation during carinal resection is to perform endobronchial intubation of both severed bronchi, a technique that allows two-lung ventilation for a much longer period during the procedure. 516 This latter technique requires the use of two ventilating systems. As the posterior anastomosis is being made, ventilation is achieved through both the distal mainstem bronchi. During repair of the anterior wall, ventilation is performed via the original endotracheal tube (above the anastomosis site). The air leak that initially occurs through the anastomosis site diminishes progressively as the anterior sutures are placed. If the anastomosis site leak is excessive, the endotracheal tube can be pushed into an endobronchial position until the placement of additional sutures reduces the leak.

It is apparent that the conventional techniques of airway management for tracheal resection are fraught with hazard. During operation a slight head-down tilt helps to minimize aspiration of blood and secretions. Intermittent sighs help prevent bronchiolar obstruction and atelectasis. A high FIO2 is used, as an oxygen-filled FRC permits a few extra minutes to correct relatively common episodes of airway obstruction and/or tube displacement. Ventilation is continuously monitored by pulse oximetry, capnography, auscultation and observation of the chest, measurement of compliance (peak inspiratory pressure), and arterial blood gas determinations. Several different sizes of armored endotracheal tubes must be available for use throughout the procedure. Finally, close communication must exist between the surgery and anesthesia teams. Disadvantages of these complex airway techniques include soiling of the lung with blood and debris, the presence of tubes in the surgical field, and the occasional necessity for using one-lung (or less) ventilation.

In an effort to overcome these problems, a third approach to airway management during tracheal resection was developed, which consists of HFJV through small-bore endotracheal tubes or catheters. 525, 526, 527, 528, 529, 533 With this technique a small-bore uncuffed catheter is placed through the stenotic area, and ventilation is accomplished by exposing the lung to rapid intermittent, high-flow, fresh gas through the catheter. Oxygen jets entrain ambient air, which in turn provides the volume necessary for adequate ventilation. With this technique, acceptable blood gases have been maintained, and there have been no deleterious effects on the circulation. With only a small catheter in the field, the surgeon can more easily perform the tracheal resection and anastomosis. The disadvantages of this technique, however, include possible inadequate escape of air around the jet catheter during exhalation when the catheter is passed through a tight stenotic area, plugging of the catheter with blood, displacement of the catheter, aspiration of blood, and technical difficulties with high-pressure injectors.

The fourth approach to airway management during tracheal resection uses HFPPV. 514 This technique uses small tidal volumes (50–250 mL) delivered via a small catheter at relatively rapid rates (50–150/min). Advantages of using HFPPV for tracheal resection include a relatively unobstructed surgical field, no interruption of ventilation during operation, minimized contamination of the lungs from blood and debris by a continuous outflow of gas, and minimized lung and mediastinal movement. In addition, production of CPAP lessens the risk of alveolar collapse. This technique has been used successfully by others for both tracheal 512, 513 and carinal resections. 512 During carinal operations HFPPV to the left lung alone generally provides adequate oxygenation and ventilation, although a system of two catheters and bilateral HFPPV could be used if necessary. 514

The fifth approach to airway management during tracheal resection, especially in cases of carinal resection, has been the institution of cardiopulmonary bypass either at the time of resection 530 or before the start of

surgery. 531 After resection, anesthesia can be continued by conventional techniques using a standard endotracheal tube. Although many surgical teams perform difficult tracheal resections with the cardiopulmonary bypass team standing by, the risk of intrapulmonary hemorrhage due to heparinization precludes its use in most cases. 515

Helium-oxygen breathing mixtures can significantly decrease the resistance to gas flow through stenotic areas and may be considered preoperatively for some of these patients. 531 However, the use of helium-oxygen mixtures prevents the use of a high FIO2 during anesthesia, and it is therefore not often recommended. 515

Postoperatively, most patients are kept in a position of head flexion to reduce tension on the suture line. If ventilatory support is necessary postoperatively, the endotracheal tube must be positioned so that the cuff does not rest on any suture line. Early extubation is highly desirable to minimize the compromise of blood flow to the trachea, which might be caused by an inflated tracheal cuff. Chest physiotherapy to remove secretions should not be too vigorous and may need augmentation by fiberoptic bronchoscopy. Systemic antibiotics or steroids are not routinely given unless infection or excessive edema, respectively, is strongly anticipated. If massive bleeding into the airway or chest occurs postoperatively, it is likely due to erosion into the pulmonary artery or aorta (high incidence after insertion of a tracheal prosthesis) and is usually fatal.

Giant Bullous Emphysema and Air Cysts

General Considerations

A *bulla* is defined as an air-filled, thin-walled space within the lung that results from the destruction of alveolar tissue. The walls of bullae are formed by connective tissue septa, compressed lung parenchyma, or pleura. A bulla usually represents a local end-stage area of emphysematous destruction. As separate but related entities, air cysts in the lung have their own epithelial margins, and they may also be associated with COPD 537 or found in the absence of other pulmonary pathology. 538

Bullectomy is the surgical resection of one or more bullae and is performed only in selected patients. The most important physiologic effect of the bullae is to compress the surrounding lung, and the release of lung compression following bullectomy is considered to be the most important factor in symptom relief (rather than eliminating dead space ventilation). Indications for bullectomy in COPD patients include intolerable breathlessness even after full medical therapy, rapidly enlarging bullae, and repeated occurrence of pneumothorax. 539 Patients with otherwise healthy lungs may undergo removal of a giant air cyst or bulla if it compresses a large area of normal lung and causes functional impairment. The compression of a large area of lung can be visualized on the chest roentgenogram (as well as on an angiogram) as the crowding together of pulmonary vessels. A strong case for functional impairment can be made if radioisotope studies show that the compressed area has good perfusion and some, but reduced, ventilation. 540, 541

The goal of surgery is to remove the bullae while preserving as much functioning lung as possible. Therefore lobectomy is not only undesirable but often unnecessary. Patients who are considered too compromised to tolerate an open approach may tolerate a closed thoracoscopic approach; the reason thoracoscopic carbon dioxide laser ablation of bullae has been so successful is that it maximizes preservation of normal lung better than any open thoracotomy approach. It is likely that this approach to bullectomy will be used with increasing frequency in the next few years. 542

Anesthetic Considerations

Anesthesia for the removal of bullae involves several specific ventilation hazards. First, most of these patients have severe generalized chronic lung disease with little or no ventilatory reserve. Thus, ventilation (which must be controlled once the chest is opened) of one severely diseased lung (the one without the giant bullae) may be hazardous and entails the risks of hypoxemia, hypercarbia, and pneumothorax on the ventilated side. If the ventilated lung also contains bullae, the risks are obviously even greater. In addition, because general anesthesia is necessary for the procedure, most patients with severe lung disease will be committed to at least a short period of postoperative mechanical ventilatory support. Second, when a bulla or air cyst is in communication with a bronchus, positive-pressure ventilation may cause it to increase in size. 543 If a significant portion of the tidal volume enters the bullous cavity, alveolar dead space ventilation will be greatly increased, and unless there is an equivalent increase in minute ventilation, the rest of the lung

may be inadequately ventilated. This complication is most likely to occur when the chest is opened, because the chest wall no longer limits the expansion of the bulla. Third, because of the rapidity with which closed air spaces take up nitrous oxide and expand in size, 544 this agent is best avoided (especially in patients whose bullae are thought to have poor communication with the bronchial system). 545 Fourth, if a check valve is present in the airway that communicates with the cavity, overinflation and air trapping may occur within the cavity. Fifth, positive pressure within a bulla might cause it to rupture, creating a pneumothorax, which would likely be under tension if the chest were closed (especially in patients whose bullae are thought to have good communication with the bronchial system). 543 Tension pneumothorax in these patients is usually a catastrophic event because of the impairment of venous return and cardiac output as well as further compromise of ventilation. Insertion of a chest tube at this point would create, in effect, a large bronchopleural cutaneous fistula that could divert much of the ventilation out through the chest tube. HFV with low tidal volumes and airway pressure has been used successfully to prevent positive-pressure rupture of a bulla according to a 1985 report. 546 Sixth, after bullectomy the remaining lung may be so overexpanded (as a result of residual bullae and/or bronchospasm) that considerable force is required to "stuff" the lung back into the hemithorax. The resultant obstruction of venous return and physical encroachment on the heart may require the use of inotropes to maintain adequate hemodynamics. Seventh, it is common for multiple small air leaks to be present after bullectomy. Collectively, these leaks cause considerable loss of delivered tidal ventilation through a chest tube. Effective ventilation and oxygenation are accomplished by trial and error using a variety of tidal volumes, airway pressures, inspiratory/ expiratory ratios, flow rates, and ventilation rates or, alternatively, HFV.

The cornerstone of the anesthetic management of patients with giant bullae or cysts is the insertion of a DLT to allow differential treatment of the two lungs. Thus, in patients with unilateral disease, the DLT can allow adequate ventilation of the nondiseased side while preventing rupture of the diseased side. In patients with bilateral disease the DLT still allows differential lung treatment to maximize gas exchange as well as providing an increased capability to deal with the complications of a ruptured bulla. For example, a DLT allows all possible permutations of HFV, CPAP, PEEP, and ZEEP to the two lungs, depending on the pathology in each lung. In addition, as each bulla is resected, the DLT allows ventilation to the operated lung to be reestablished for short periods, enabling the surgeon to identify and suture any air leaks that may be present. The versatility of the DLT is particularly important in thoracoscopic procedures, which may average 3 hours in duration. 542

A DLT can be inserted with the patient either awake with the airway topically anesthetized (for those with histories of repeated pneumothorax and/or severe bilateral bullae) or under general anesthesia (for the majority of patients) to isolate the affected lung and provide positive-pressure ventilation to the contralateral lung. 547, 548 While the depth of general anesthesia is being increased, spontaneous ventilation may be maintained (primarily indicated in patients with a history of repeated pneumothorax or bilateral bullae), but it should be realized that spontaneously breathing patients with significant pulmonary disease under general anesthesia may not be able to ventilate themselves adequately. Alternatively, and preferably in the majority of patients, the patient can be anesthetized and both lungs ventilated by using a limited amount of positive airway pressure; gentle ventilation by hand is the best way to ensure low airway pressures. If a major air leak develops intraoperatively while positive pressure ventilation is being used with a volume cycled ventilator, inadequate ventilation may occur (the machine delivers the preset volume but it does not go into the lungs). Under these circumstances a pressure cycled high inspiratory flow ventilator must be used (e.g., Siemens Servo 900C, Solna, Sweden). 542

If limited positive-pressure ventilation is chosen, it is important that the anesthesiologist be able to diagnose and treat a pneumothorax rapidly. External stethoscopes should be attached over each hemithorax at the points where breath sounds are maximal to monitor for pneumothorax on each side. 549 However, advanced bullous disease may completely prevent breath sounds from being heard externally at all. In addition to a decrease in breath sounds, a pneumothorax or check-valve mechanism in a cyst may be signaled by an increase in airway pressure, tracheal shift to the opposite side, or hypotension disproportionate to the depth of anesthesia. Thus, equipment for chest tube placement must be immediately available for these patients.

Theoretically, after bullectomy the patient's pulmonary status should be improved since the "healthy" lung tissue that was previously compressed by the bullae should now be able to expand. However, in our experience and that of others, 542 the weaning and extubation process often takes up to several days in those patients with advanced disease. When mechanical ventilation is required postoperatively, positive

airway pressure should again be minimized to decrease the possibility of producing a pneumothorax from rupture of suture lines and/or residual bullae 550 and to minimize air leaks from remaining lung tissue.

When bilateral bullectomy is performed because of extensive disease in both lungs, a sternal splitting incision with the patient supine is usually made. 551 However, sequential posterolateral thoracotomies may be planned if it is desired to see how the patient responds to the first bullectomy. With bilateral bullectomy, the same anesthetic principles apply as with unilateral bullectomy.

Lung Volume Reduction Surgery

General and Surgical Considerations

Advanced emphysema is a debilitating lung disease that afflicts 2 million people in the United States. 552 In emphysema, the lungs are hyperinflated with poor elastic recoil, the airways are dynamically compressed, the flattened diaphragms contract poorly, and cardiac filling may be impaired. These pathophysiologic changes result in hypoxemia, hypercapnia, an increased work of breathing, and a debilitating shortness of breath. The goal of lung volume reduction surgery is to partially reverse these pathophysiologic mechanisms. 553

Current approaches to bilateral lung volume reduction surgery include median sternotomy and thoracoscopy, using either a linear cutting stapler or laser ablation, and reinforcement of incised lung parenchyma with exogenous buttressing materials (such as bovine pericardial strips). 553 The lung tissue to be resected is determined by visual assessment of the most diseased lung (which remains inflated during contralateral one-lung ventilation); it most often consists of multiple small regions, is nonanatomic, and usually composes 20 to 30 percent of that lung. 553 In some patients, however, only a few large blebs or bullae are resected. In either case, the most abnormally appearing tissue is resected to allow expansion of the more normal remaining lung tissue. The majority of patients who have undergone this operation have experienced improvement in lung function and quality of life. The two principal causes of morbidity and mortality in patients undergoing this procedure are respiratory impairment due to the induction of general anesthesia and the trauma of the operative incision in these already critically ill patients, and the complications arising from the resection of emphysematous lung tissue, most notably persistent air leaks.

Anesthetic Management

The overriding goal of anesthetic management is to have the patient adequately and quietly breathing spontaneously as soon as possible at the end of surgery to minimize positive pressure–induced lung air leaks. The main components of the anesthetic management that allow achievement of this goal are to use general anesthesia supplemented with thoracic epidural anesthesia intraoperatively and continued after surgery to provide postoperative analgesia, minimal to no intravenous narcotics, fluid restriction, and often a prolonged emergence and recovery (can be a few hours) with mask ventilation assistance is required in the operating room.

Essential invasive monitoring consists of arterial and central venous catheters. The usual indications for a pulmonary artery catheter remain. A thoracic epidural catheter is placed before induction of general anesthesia and the position of the catheter pharmacologically and/or fluoroscopically determined. Optimally the catheter tip is at an upper thoracic (T3–T5) level, being located in the mid-dermatome area of the incision. These very respiratorily debilitated patients are either unpremedicated or receive only small doses of midazolam, if they are anxious. Anesthesia is induced intravenously (without narcotics), the patient paralyzed, a left-sided double-lumen tube passed, and the position confirmed with fiberoptic bronchoscopy. Anesthesia is maintained via the thoracic epidural catheter and a propofol infusion, with or without an added low-dose of volatile anesthetic. During one-lung ventilation, the tidal volume should be adjusted to achieve a peak inspiratory pressure of approximately 25 cm H2 O and long expiratory times utilized. With these ventilator settings, hypercapnia is often unavoidable and entirely permissible as long as patients are normoxemic. 554 After periods of one-lung ventilation, the previously atelectatic lung should be very slowly and gently reexpanded. Hypotension is treated with vasopressors and fluid infusion is restricted to less than 1,000 mL (blood loss is usually minimal). The patients are usually tracheally extubated in a surgical plane of anesthesia and adequate gas exchange maintained via assisted mask ventilation until adequate spontaneous ventilation returns. During the prolonged assisted emergence, intensive respiratory care, consisting of bronchodilators and chest physical therapy, may be required/desired. Postoperatively pain

control is achieved by infusion of a dilute local anesthetic solution (e.g., 0.125 percent bupivacaine) in an effort to block afferent pain signals with minimal effect on motor function (Ch. 69). If required, a very low dose of narcotic can be added to the infusion for a synergistic analgesic effect.

Unilateral Bronchopulmonary Lavage

General Considerations

Unilateral bronchopulmonary lavage, or massive irrigation of the tracheobronchial tree of one lung, has been used with good success in patients with pulmonary alveolar proteinosis as a means of removing the enormous accumulations of alveolar lipoproteinaceous material characteristically present in these patients. 555, 556, 557, 558, 559 The lipoproteinaceous material is thought to be surfactant, 560 and the abnormal accumulation is due to failure of clearance mechanisms rather than to enhanced formation. 561 The abnormal accumulation of alveolar lipoproteinaceous material is bilateral and symmetric and causes the classic chest radiographic picture of air space consolidation with patchy, poorly defined shadows throughout the lung. 562 The radiographic picture reflects the course of the disease. The air space consolidation causes progressive hypoxemia and shortness of breath (first on exertion and then at rest), 562, 563 and the lungs have a low compliance. The diagnosis of alveolar proteinosis is made by correlating the clinical, radiologic, and laboratory data with the results of a lung biopsy. The indication for lung lavage is PaO2 less than 60 mm Hg at rest or hypoxemic limitation of normal activity. 564, 565 Infrequently, lung lavage may be performed in patients with asthma, cystic fibrosis, and radioactive dust inhalation. 552, 557, 565, 566, 567

Unilateral lung lavage is performed under general anesthesia with a double-lumen endotracheal tube, allowing lavage of one lung while the other lung is ventilated (Fig. 48–38). In patients with alveolar proteinosis, lavage is performed on one lung and then, after a few days' rest, on the other lung. After lung lavage these patients usually have marked subjective improvement, which correlates with increases in PaO2 during rest and exercise, increased vital capacity and diffusion capacity, and clearing of the chest radiograph. 568, 569 Some patients require lavage every few months, whereas others remain in remission for several years, and the disease may even eventually completely remit. 568, 569
FIGURE 48–38 Technique for providing unilateral bronchopulmonary lavage. A left clear plastic double-lumen endotracheal tube allows ventilation to the left lung during lavage of the right lung (and vice versa). Normal saline is infused and drained by gravity; clamps on the connection tubes determine direction of fluid flow. (See text for details.) (From Benumof604)

Anesthetic Considerations

This section discusses the technique for anesthetic management of bronchopulmonary lavage in patients with pulmonary alveolar proteinosis. 260 When the patient is admitted to the hospital, V/Q scans of the lung are obtained. Ventilation can be maximized during lung lavage by performing the first lavage on the most severely affected lung, allowing the "better" lung to provide gas exchange. If the scan indicates relatively equal involvement (as is usually the case), the left lung is lavaged first, leaving the larger right lung to support gas exchange.

After several minutes of preoxygenation (see below), general anesthesia is induced with thiopental in divided doses and inhalation of either isoflurane or halothane in 100 percent oxygen. Isoflurane is relatively indicated in patients in whom therapeutic levels of theophylline (and the risk of arrhythmias) are present. Neuromuscular blockade is induced with a nondepolarizing muscle relaxant. When a suitable level of anesthesia has been reached, the trachea is intubated with the largest left-sided DLT that can be passed atraumatically through the glottis. A clear plastic disposable left-sided DLT is used because of the ease and certainty with which it is correctly positioned, the reliable left cuff seal obtained (the right endobronchial cuff is small and inflates asymmetrically), and the ability to continuously observe the tidal movements of respiratory gas moisture (ventilated lung) and the lavage drainage fluid for leaking air bubbles (lavaged lung). The largest tube is used because the left endobronchial cuff will make contact over a greater bronchial mucosal area with less air in the left cuff (as compared with a small DLT). In addition, a large tube facilitates suctioning, which is an important consideration at the end of the case when the lungs must be made as clear as possible. Precise placement of the tube and detection of leaks are essential because of the serious hazard of spillage during the lavage procedure. The position of the DLT must be confirmed with a fiberoptic bronchoscope, and left cuff seal must be demonstrated to hold against 50 cm H2 O left lung air pressure by

a right lumen to catheter-under-water technique (see the section *Quantitative Determination of Cuff Seal Pressure Hold*).

The question of patient position during unilateral lung lavage is important, for there are major advantages and disadvantages related to each position (Table 48–22). The LDP with the lavaged lung dependent minimizes the possibility of accidental spillage of lavage fluid from the dependent lavaged lung to the nondependent ventilated lung. However, during periods of lavage fluid drainage, pulmonary blood flow, which is gravity-dependent, would preferentially perfuse the nonventilated dependent lung, and the right-to-left transpulmonary shunt would be maximal. The LDP with the lavaged lung nondependent minimizes blood flow to the nonventilated lung, but, on the other hand, increases the possibility of accidental spillage of lavage fluid from the lavaged lung to the dependent ventilated lung. As a compromise, the supine position is used to balance the risk of aspiration with the risk of hypoxemia.

TABLE 48–22. Unilateral Lung Lavage: Position of Lavaged Lung

After insertion and checking of the DLT and positioning of the patient, baseline total and individual lung compliances are measured. Airway pressure can be electronically transduced and continuously recorded on a paper write-out, and a Wright spirometer should be placed in the expiratory limb of the anesthesia circle system to measure tidal volume accurately. A volume ventilator that can deliver relatively high inflation pressures is required, because these patients have diseased and noncompliant lungs. Prelavage total dynamic compliance (chest wall and lung) of both lungs together (using 15 mL/kg tidal volume) and then of each lung separately (using 10 mL/kg tidal volume) is measured. After measurement of total and individual lung compliance and with the patient breathing 100 percent oxygen, baseline arterial blood gases are measured.

The patients are completely preoxygenated before induction of anesthesia and lavage for two reasons. First, as with the induction of general anesthesia in any patient, an oxygen-filled FRC greatly minimizes the risk of hypoxemia during the apneic period required for laryngoscopy and endotracheal intubation. This consideration has added importance for patients with alveolar proteinosis because they are already severely hypoxemic. Second, preoxygenation eliminates nitrogen from the lung that is to be lavaged. Alveolar gas is then composed only of oxygen and carbon dioxide. During fluid filling, these gases will be absorbed, allowing the lavage fluid maximal access to the alveolar space. Failure to remove nitrogen from the lung before filling the lavage fluid may leave peripheral nitrogen bubbles in the alveoli and thus limit the effectiveness of the lavage.

Warmed isotonic saline is used as the lavage fluid and is infused by gravity from a height of 30 cm above the midaxillary line. After the lavage fluid ceases to flow (i.e., lung filling is complete), drainage is accomplished by clamping the inflow line and unclamping the drainage line, which runs to a collection bottle placed 20 cm below the midaxillary line (see Fig. 48–38). The inflow and outflow fluid lines are connected to the appropriate endotracheal tube lumen by a Y-adapter. Each tidal lavage filling is accompanied by mechanical chest percussion and vibration to the lavaged hemithorax before drainage. The lavage fluid that is drained is typically light brown, and the sediment layers out at the bottom of the collection bottle after a short time. Filling and drainage of approximately 500- to 1,000-mL aliquots are repeated until the lavage effluent clears (see Fig. 48–38). Volumes delivered to and recovered from each tidal lavage are recorded. Total lavage fluid volumes of 10 to 20 L are usually used.

Most patients studied have been hemodynamically stable throughout the entire lavage procedure. In particular, lavage itself has caused no significant changes in systemic and transmural pulmonary artery pressures and cardiac output. 570 In these patients, the arterial saturation, as measured by pulse oximetry, has increased and decreased, with each lung filling and drainage, respectively. 570 In addition, cardiac output has decreased and increased with each lung filling and drainage, respectively. Arterial saturation increases during lung filling because blood flow to the nonventilated lung is decreased by the lavage fluid infusion pressure. 571 The opposite set of events (increased nonventilated lung blood flow, decreased arterial oxygen saturation) occurs during lung drainage. 571 Not surprisingly, the cardiac output decreases with each lavage fluid instillation because of the increase in PVR. 570 The opposite set of events (increased nonventilated lung blood flow, decreased arterial oxygen saturation) occurs during drainage. 571 In addition, with drainage of the lavage fluid, PVR decreases and cardiac output increases. 570 An adequate degree of neuromuscular blockade must be maintained because unexpected vigorous coughing during the procedure could alter DLT position and allow spillage of fluid from the lavaged lung to the ventilated lung.

If a small leak should occur during lavage, the following may be observed sequentially: (1) the appearance of bubbles in the lavage fluid draining from the lavaged lung, (2) rales and rhonchi in the ventilated lung, (3) a difference between lavage volumes administered and those drained from the lavaged lung (the former exceeds the latter), and (4) a fall in arterial oxygen saturation. If a small leak is suspected or detected by any of these signs and the lavaged lung has been only minimally treated, the lavaged lung should be drained of all fluid, and the position of the DLT, the adequacy of cuff seal, and separation of the lungs should be rechecked with a fiberoptic bronchoscope. Before beginning the lavage procedure again and no matter what the DLT malposition was, the functional separation of the two lungs and adequacy of cuff seal should be tested and found adequate by using the previously described air bubble leak detection method.

Massive spillage of fluid from the lavaged lung to the ventilated lung is not a subtle event and results in a dramatic decrease in ventilated lung compliance and a rapid and profound decrease in arterial oxygen saturation. Under these circumstances, the lavage procedure must be terminated no matter how much treatment has been accomplished. The patient should be moved quickly to the LDP with the lavaged side dependent, and the operating room table should be placed in a head-down position to facilitate removal of lavage fluid. Vigorous suctioning and inflation of both lungs should be carried out. The DLT should be changed to a standard single-lumen tube, and the patient should additionally receive a period of mechanical ventilatory support with PEEP. Timing of further unilateral lung lavage attempts will be dictated by the patient‘s subsequent clinical course and gas exchange status.

After the effluent lavage fluid becomes clear, the procedure is terminated. The lavaged lung is thoroughly suctioned, and ventilation to that lung is resumed. Because the compliance of the lavaged side will be much less than that of the ventilated side at this time, large tidal ventilations (sighs) (15–20 mL/kg) to that side alone (with the nonlavaged side temporarily nonventilated) are necessary to reexpand alveoli. Arterial blood oxygenation may decrease precipitously during this time, but this can be minimized by clamping the nonlavaged side after a large inspiration of 100 percent oxygen.

After lavage, the recovery procedure consists of repetitive periods of large tidal ventilations, suctioning and chest wall percussion to the previously lavaged side, conventional two-lung ventilation with PEEP, and bilateral suctioning and postural drainage while intermittently measuring combined (total) and individual lung dynamic compliance. As the compliance of the lavaged lung returns to prelavage values, ventilation with an air-oxygen mixture may help lavaged lung alveoli with low V/Q ratios to remain open. When the compliance of the hemothorax of the lavaged side returns to its prelavage value, the neuromuscular blockade is reversed. Mechanical ventilation and extubation guidelines are the same as for any patient with pulmonary disease; most patients are able to be extubated while still in the operating room.

In the immediate postlavage period, deep breathing (incentive spirometry), coughing exercises, chest percussion, and postural drainage are used to remove remaining fluid and secretions and to reexpand the lavaged lung further. After 3 to 5 days of recovery, the patient is returned to the operating room to have the opposite side lavaged. The anesthetic considerations for the second lavage are the same as for the first lavage, although oxygenation is usually not nearly as severe a problem as during the first lavage because the treated and now near normal lung will be used to support gas exchange.

Special problems associated with pulmonary lavage that may be encountered are that a few very critically ill patients may be unable to tolerate the conventional procedure, and unilateral lavage through a DLT is not easily done in children. Alternative (and more complicated) ways of accomplishing lung lavage in these patients include use of extracorporeal membrane oxygenation and lobar lavage via a fiberoptic bronchoscope inserted under topical anesthesia. 260

Tumors at the Confluence of the Superior, Anterior, and Middle Mediastinum

General Considerations

At the confluence of the superior, anterior, and middle mediastinum are the middle portion of the superior vena cava, the tracheal bifurcation, the main pulmonary artery, the aortic arch, and parts of the cephalad surface of the heart. 260 In adults, the majority of tumors in this region originate from involvement of the hilar lymph nodes with bronchial carcinoma or lymphoma, whereas in babies the masses are most often benign bronchial cysts, esophageal duplication, or teratoma. Tumors of this region can cause compression

and obstruction of three of the vital mediastinal structures: the tracheobronchial tree in the region of the tracheal carina, the main pulmonary artery and atria, and the superior vena cava. A CT scan of the chest is probably the single most important diagnostic procedure because it defines the size and degree of compression of these vital structures. The most common complication that occurs during anesthesia for masses involving these three vital mediastinal structures is airway obstruction; this was a feature in 20 of 22 patients recently reviewed (1969–1983) and succinctly summarized. 572 Although airway obstruction has been predominant in terms of symptomatology, it is not uncommon for compression of two or three of the major organs to be present in varying degrees and to cause complications in the same patient. 572 Each of these complications is life-threatening and can cause acute deterioration and death during anesthesia if not handled with the most extreme caution and expertise. Each major complication and anesthetic management problem is discussed separately below, and Figure 48–39 shows the overall strategy for managing these three problems.

FIGURE 48–39 Flow diagram showing the essential management strategies for the three major anesthetic problems that mediastinal masses may cause. TBT, tracheobronchial tree; SVC, superior vena cava; PA, pulmonary artery; FOB, fiberoptic bronchoscopy; ETT, endotracheal tube; CPB, cardiopulmonary bypass. (From Benumof604)

Compression of the Tracheobronchial Tree

Most anterior mediastinal masses that cause airway obstruction 260 are lymphomatous in origin. However, a number of benign conditions such as cystic hygroma, teratoma, thymoma, and thyroid tumors can occur in a similar fashion. A tissue diagnosis, therefore, should be made before radiation or chemotherapy can be undertaken. Thus, most patients with a mediastinal mass causing airway obstruction will first require anesthesia for a diagnostic procedure (e.g., cervical or scalene node biopsy, staging laparotomy for Hodgkin‘s disease). Importantly, not all patients who have developed severe intraoperative respiratory problems have respiratory symptoms and signs preoperatively.

The anesthetic management of these patients is based on two overriding considerations. First, the obstruction of major airways by a tumor is usually life-threatening because the obstruction usually occurs around the bifurcation of the tracheobronchial tree and is therefore distal to the endotracheal tube. The loss of spontaneous ventilation seems to precipitate the airway obstruction. It may be that loss of chest wall tone and the distending forces of active inspiration after administration of muscle relaxants release extrinsic support of a critically narrowed airway. Alternatively, intubation in the presence of distortion or compression of the trachea may cause complete obstruction if the orifice of the tube impinges on the tracheal wall or if the lumen of the tube is occluded where it passes a narrowed section or turns a sharp angle. In view of the potential for fatal airway obstruction during general anesthesia, all possible attempts to perform the procedure under local anesthesia should be undertaken.

Second, the response of lymphomatous tumors to radiation or chemotherapy is normally dramatic. Chest radiographs reveal a marked decrease in tumor size, and the symptoms are usually improved. Consequently, it behooves the treating physicians to use radiotherapy or chemotherapy if at all possible (sometimes the cell type can be known with a reasonable degree of certainty without a biopsy) before general anesthesia is used. 573, 574 It should be noted that during radiation, a small window can be created to spare some tissue for adequate histologic diagnosis. 575, 576

The following management plan is based on the principles outlined 560, 572, 573, 574, 575, 576 (Table 48–23 and see Fig. 48–39). If a patient with a mediastinal mass near the confluence of the superior, anterior, and middle mediastinum exhibits dyspnea and/or intolerance of the supine position and is scheduled for biopsy, it should be performed under local anesthesia if at all possible. If the cell type is thought to be radiosensitive or chemosensitive, appropriate types of therapy should be undertaken before any further surgery is performed. After these types of therapy, the radiologic appearance of the tumor must be reviewed along with a dynamic evaluation of pulmonary function (see later).

TABLE 48–23. Important Management Principles for Tumors at the Confluence of the Superior, Anterior, and Middle Mediastinum

If the patient does not have dyspnea or intolerance of the supine position (i.e., is asymptomatic), a CT scan, flow-volume loop, and echocardiogram should be obtained to evaluate the anatomic and functional position

of the tumor. If any of these three tests has positive results, local anesthesia should be used for biopsy even if the patient is asymptomatic.

If general anesthesia is to be used, the airway should be evaluated by fiberoptic bronchoscopy with topical anesthesia before induction of general anesthesia. 576 The fiberoptic bronchoscope should be jacketed with an armored endotracheal tube, and after the fiberoptic bronchoscopy examination has been completed, the patient is intubated. General anesthesia should be induced with the patient in the semi-Fowler position. The patient should be allowed to breathe spontaneously throughout the procedure; muscle relaxants should be avoided. Large changes in intrathoracic pressure, which may promote collapse of a weakened tracheobronchial tree, must be prevented. The operating room team should retain the capability of changing the patient's position rapidly to the lateral or prone position. A rigid ventilating bronchoscope should be on hand to bypass distal tracheal and carinal obstructions, and the appropriate personnel and equipment for cardiopulmonary bypass also should be available.

These patients must be watched extremely closely in the first few postoperative hours. Airway obstruction requiring reintubation and mechanical ventilation has occurred, possibly secondary to an increase in tumor size due to tumor edema after instrumentation.

Compression of the Pulmonary Artery and Heart

Compression of the pulmonary artery and heart is very rare, because the pulmonary trunk is more or less protected by the aortic arch and tracheobronchial tree; there are only a few case reports of this problem in the literature. 260

Principles similar to those for compression of the tracheobronchial tree apply to compression of the pulmonary artery. Most patients have their first anesthetic experience because they require a diagnostic procedure (e.g., a biopsy). These patients should be evaluated preoperatively in a manner similar to that used for patients with compression of the tracheobronchial tree. If the cell type is known or is highly suspected, preoperative irradiation should be seriously considered. All diagnostic procedures should be performed under local anesthesia if at all possible. If general anesthesia is required and if the symptoms worsen in the supine position, the sitting, leaning forward, or even face-down position is advised, and spontaneous ventilation should be maintained throughout the procedure. Measures to maintain venous return, pulmonary artery pressure, and cardiac output, such as volume loading and use of ketamine, should be considered. Arrangements for extracorporeal oxygenation should be completed preoperatively.

Superior Vena Cava Syndrome

The superior vena cava syndrome is caused by mechanical obstruction of the superior vena cava. The causes of superior vena caval obstruction, in order of decreasing incidence, are bronchial carcinoma (87%), malignant lymphoma (10%), and benign causes (3%), such as central venous hyperalimentation and pacemaker catheter–induced thrombosis of the superior vena cava, idiopathic mediastinal fibrosis, mediastinal granuloma, and multinodular goiter. 260 The classic features of the superior vena cava syndrome include dilated distended veins in the upper half of the body due to increased peripheral venous pressure (which can be as high as 40 mm Hg); edema of the head, neck, and upper extremities; dilated venous collateral channels in the chest wall; and cyanosis. Venous distention is most prominent in the recumbent position, but in most instances the veins do not collapse in the normal manner with the patient upright. Edema can be so great as to cause swelling of periorbital fissures, preventing patients from opening their eyes. Such advanced cases of edema usually obscure the dilated veins as well. The majority of patients have respiratory symptoms (shortness of breath, cough, orthopnea), which are due to obstruction of the airways by engorged veins and mucosal edema, and these are ominous signs. Similarly, a change in mentation, due to cerebral venous hypertension and edema, is a sign of particularly significant obstruction. In some cases the superior vena cava becomes occluded quite slowly, and the signs and symptoms may be insidious in onset. When the occlusion occurs relatively rapidly, all clinical manifestations are more prominent. The most common radiologic sign is widening of the superior mediastinum. Venography confirms the diagnosis (but not the cause). Determination of etiology may require thoracotomy, sternotomy, bronchoscopy, lymph node biopsy, etc.

Most patients with superior vena cava syndrome due to a malignant process are treated with irradiation and chemotherapy (for patients with incomplete obstruction). 577 However, in patients with near complete to

complete obstruction (who usually have signs of cerebral venous hypertension and/or airway obstruction) or in whom irradiation or chemotherapy proves ineffective, surgical bypass or resection of the lesion via median sternotomy is indicated. 577 These operations are usually technically quite difficult because tissue planes are poorly delineated, anatomy is grossly distorted, central venous pressures are abnormally high, and varying degrees of fibrosis are present.

The preoperative anesthetic evaluation of a patient for superior vena caval decompression should include careful assessment of the airway. The same degree of edema that is present externally in the face and neck can be expected to be present in the mouth, oropharynx, and hypopharynx. In addition, the airway may be compromised by external compression, fibrosis limiting normal movement, or recurrent laryngeal nerve involvement. If tracheal compression is suspected, it should be evaluated by CT.

The patient is transported to the operating room in the head-up position to minimize airway edema. A radial artery catheter is inserted in all patients and depending on the medical condition of the patient, a central venous or pulmonary artery catheter is inserted via the femoral vein before induction of anesthesia. At least one large-bore intravenous cannula should be inserted into the leg or femoral vein before operating. Premedication is best limited to an antisialagogue to reduce airway secretions. The method chosen for induction of anesthesia and intubation depends on the preoperative airway evaluation. If it is necessary for the patient to maintain the sitting position to achieve adequate ventilation before induction, intubation with the patient awake may be facilitated by using a fiberoptic laryngoscope or bronchoscope.

The most significant intraoperative problem encountered is bleeding. Substantial venous blood loss results from the abnormally high central venous pressure. Further, unexpected arterial bleeding may occur because of the difficulty of dissecting in a distorted surgical field. Crossmatched blood should therefore be available in the operating room at the time of sternotomy.

Postoperatively, especially after diagnostic procedures such as mediastinoscopy and bronchoscopy wherein the superior vena caval obstruction has not been relieved, acute severe respiratory failure requiring intubation and mechanical ventilation may occur. 260 The mechanisms of the acute respiratory failure are obscure, but the most likely ones that are unique to the superior vena cava syndrome are acute laryngospasm and/or acute bronchospasm (both due to continued and perhaps increased obstruction of the superior vena cava), impaired respiratory muscle function (patients with malignant disease may have an abnormal response to muscle relaxants), and increased airway obstruction by the tumor (due to tumor swelling). Consequently, these patients must be closely monitored in the first few postoperative hours.

Emergency Thoracic Procedures

Massive Hemoptysis

General Considerations

Massive hemoptysis 578 is uncommon, occurring in less than 0.5 percent of patients admitted to a large pulmonary medicine service. It has been arbitrarily defined, on the basis of the amount of daily volume of blood expectorated, as 200 to 600 mL in 24 to 48 hours, or from the standpoint of causing acute airway obstruction or major hypotension. 578

More than 90 percent of reported cases of massive hemoptysis have a chronic infectious cause 579 because chronic inflammation leads to profuse vascularization of the high-pressure bronchial artery system. Subsequently, any erosion or rupture of enlarged bronchial arteries will result in massive hemoptysis. Active tuberculosis is the most common and bronchiectasis the second most common infection causing massive hemoptysis. 580 The majority of the remaining causes of hemoptysis are due to bleeding neoplasms.

Surgical Considerations

In a series of 55 pulmonary resections performed for massive hemoptysis (600 mL in 16 hours), a mortality rate of 18 percent was reported 581 ; this was markedly better than with conservative treatment, which resulted in a mortality rate of 75 percent in patients who bled 600 mL or more in 16 hours and 54 percent in those who bled 600 mL or more in 48 hours. 582, 583 However, routine use of surgery has been debated, as other authors have found that somewhat lesser degrees of hemoptysis may be successfully managed

conservatively regardless of the amount of bleeding in the first 24 hours. 578 Nevertheless, surgery (resection) is probably indicated in patients who require multiple transfusions, in those in whom bleeding results in progressive impairment of pulmonary function (aspiration should be evaluated by serial chest radiographs and arterial blood gases), and in those in whom hemoptysis persists for several days despite optimum medical treatment. Contraindications to surgery include inoperable carcinoma of the lung, inability to localize the bleeding site, and the presence of severe bilateral pulmonary disease and systemic disease (debilitation). These patients are candidates for bronchial artery embolization (Fig. 48–40) (see later).
FIGURE 48–40 Treatment algorithm for massive hemoptysis. The most important function of emergency bronchoscopy is to establish or to diagnose the cause and site of bleeding. In addition, the amount and spread of bleeding can be controlled during emergency bronchoscopy (see items listed in the algorithm under “Diagnosis of Cause”). (Modified from Benumof604)

Bronchoscopy during active bleeding is the single most important technique for determining the cause and location of bleeding and should be performed in all patients; the procedure should be done in the operating room so that immediate resection can be performed 583 (see Fig. 48–40). Most surgeons use a rigid bronchoscope because of the much greater suctioning and ventilating capability. However, the flexible fiberoptic bronchoscope may be used if there is no active bleeding and/or the site of bleeding is thought to be in the upper lobes.

The surgeon may be able to control bleeding and the spread of bleeding during bronchoscopy (see Fig. 48–40). Topical iced saline and vasoconstrictors can be administered through the bronchoscope to control bleeding, provided the bleeding is not so massive as to preclude visualization of its origin. 584 Control of spread of blood from one lung to the other can be achieved by use of a bronchial blocker (e.g., balloon-tipped Fogarty catheter) in the main bronchus of the bleeding side or by use of a gauze packing of the bleeding segment or side. 578 The Nd:YAG laser has proved efficacious in the treatment of hemoptysis in patients with lung cancer. 585, 586 In a very exciting new development in pulmonary medicine, two recent reports describe complete cessation of bleeding in almost all patients by selective intrabronchial spraying of fibrin precursors through a catheter in the suction port of a fiberoptic bronchoscope without any adverse effect. 587, 588 During bronchoscopy, the surgeon should frequently restore adequate oxygenation and ventilation by intubating the uninvolved main stem bronchus. If the patient can withstand surgery and has an identified operable lesion, surgery should be performed. If the patient cannot withstand surgery and/or has an inoperable lesion, bronchial embolization should be attempted (see Fig. 48–40).

Anesthetic Considerations

There are several important preoperative priorities. For patients with massive hemoptysis, asphyxiation must be prevented by administering 100 percent oxygen; placing the bleeding lung in a dependent position; separating the lungs with a DLT, endobronchial single-lumen tube, or bronchial blocker; and using intermittent positive-pressure ventilation and vigorous suction. After the airway is secured and the bleeding lung isolated, the patient is placed so that the bleeding lung is in a nondependent position.

Coughing may increase bleeding. The advisability of using sedatives and cough suppressants is time-dependent. In the unintubated patient, the ability to cough may be lifesaving, and suppressants should be avoided. In the intubated patient, suctioning can replace the cough mechanism, and suppression of cough may decrease bleeding. A coagulation profile should be drawn early; if any abnormalities are noted, they should be corrected. During and after bronchoscopy, the surgeon and pulmonologist can control bleeding by iced saline lavage, placement of topical vasoconstrictors, placement of a bronchial blocker or gauze packing, laser coagulation, fibrin seal, and use of bronchial artery embolization (see earlier).

As soon as possible, several large-bore intravenous cannulae are inserted. The patient‘s blood is typed and cross-matched for adequate amounts of blood products (whole blood, packed red blood cells, platelets, fresh frozen plasma). Transfusion should begin if appropriate. Antibiotics are administered preoperatively, and antituberculous drugs are started in patients with tuberculosis. Finally, the appropriate monitoring (e.g., arterial line, central venous line) is instituted. Many of these procedures should be undertaken simultaneously.

If the patient with massive hemoptysis is without an indwelling endotracheal tube, preoxygenation should be instituted immediately. Adequate suctioning must be available. It may be necessary for the patient to be awake for intubation during massive, active, spontaneous bleeding to prevent the hazard of trying to

visualize a blood-obscured airway in a paralyzed patient. Intubation performed in the semiupright position may minimize coughing that results in the presence of blood in the upper airway and thereby may provide a clearer field of vision.

If the patient without an indwelling endotracheal tube is to be anesthetized, aspiration precautions (e.g., cricoid pressure) should be used, because many patients have swallowed expectorated blood. Because these patients are likely to be hypovolemic, the induction of anesthesia should be accomplished with a small dose of short-acting barbiturate or ketamine or with narcotics followed in rapid sequence by relaxation. If the larynx can be visualized, insertion of a DLT is preferable to insertion of a single-lumen tube. It should be remembered that if the patient is not actively bleeding but has a blood-filled cavity (i.e., a hemorrhagic lobe), this cavity will likely empty its contents into the dependent unsoiled lung when the patient is turned to the LDP; therefore, this situation is a strong indication for placement of a DLT. If a single-lumen tube is inserted, it might as well be the Univent bronchial blocker tube, which additionally allows lung separation.

If a single-lumen tube is already in place, consideration should be given to conversion to a DLT, addition of a bronchial blocker, and/or achieving an endobronchial position of the existing tube. Once the airway has been secured, the patient must be placed in the LDP with the bleeding lung in the nondependent position. Use of this position, of course, emphasizes the importance of separating the lungs. In all situations if active tuberculosis is present or suspected, contamination precautions should be taken.

At the end of surgery, the endotracheal tube should be left in place and the patient should be ventilated mechanically. Most of these patients will have impaired gas exchange postoperatively as a result of preexisting lung disease, the probability that the nonbleeding lung has been soiled by the recent hemoptysis from the diseased lung, and the physiologic consequence of having just undergone a major anesthetic and surgical experience.

Bronchopleural Fistula

General Considerations

A bronchopleural fistula may be caused by the rupture of a lung abscess, bronchus, bulla, cyst, or parenchymal tissue (as in the case of high levels of PEEP during mechanical ventilation) into the pleural space; by erosion of a bronchus by a carcinoma or chronic inflammatory disease; and by breakdown of a bronchial suture line after pulmonary resection.

Surgical Considerations

The diagnosis of bronchopleural fistula is usually made clinically. In early postpneumonectomy patients, the diagnosis is based on sudden dyspnea, subcutaneous emphysema, contralateral deviation of the trachea, and disappearance of the fluid level on radiographs of the chest. In patients after lobectomy, persistent air leak, purulent drainage, and expectoration of purulent material are usually diagnostic. When the fistula appears after removal of the chest tube, the diagnosis of a bronchopleural fistula is made on the basis of fever, purulent sputum, and a new air fluid level in the pleural cavity on the chest radiograph. The diagnosis is confirmed by bronchoscopic examination in most, bronchography in a few, and sinograms (of the fistula) in occasional patients. 589 Other methods consist of injection of an indicator such as methylene blue into the pleural space and its recovery from the sputum, accumulation of radionuclide in the pleural space after inhalation of xenon, or a bronchogram showing spillage of contrast into the vacant hemithorax.

In postpneumonectomy patients, if the disruption occurs early, it is possible to resuture the stump. Late postpneumonectomy bronchial disruption associated with empyema has been managed by conservative drainage, but definitive operative closure is now considered the treatment of choice.

In non-postpneumonectomy cases, if the lung expands to fill the thoracic cavity, the leak can usually be controlled with chest tube drainage alone. However, if the fistula is large and a significant leak through a large persistent pleural space occurs, it is unlikely that the fistula will close, and surgical resection is usually necessary. 589 An empyema complicating a bronchopleural fistula should, if possible, be drained before surgery (see next section).

Finally, a spontaneous pneumothorax (no prior lung resection involved) is pathophysiologically similar to a bronchopleural fistula. There are three situations in which definitive surgical treatment is indicated in management of spontaneous pneumothorax. First, surgery is required when conventional tube drainage and suction have been unsuccessful in clearing the pleural space and when, in effect, a bronchopleural fistula has formed. Second, surgical intervention is usually indicated when a second ipsilateral or first contralateral spontaneous pneumothorax occurs. Third, considering that a spontaneous pneumothorax has a recurrence rate of 10 to 25 percent, if after the initial event the patient's life-style is such that a recurrence might be life-threatening or highly inconvenient, definitive treatment is indicated. Surgical procedure options are pleurectomy and chemical pleurodesis. 590

Anesthetic Considerations

Preoperatively, it is useful to estimate the loss of tidal volume through the bronchopleural fistula. This may be done in two ways. First, one should determine whether air bubbles intermittently or continuously through the chest tube. If air bubbles intermittently, it means the fistula is small. In contrast, when a patient has a large, low-resistance bronchopleural fistula or bronchial rupture, air may bubble continuously through the water seal chamber of the chest tube drainage system. Second, the size of the bronchopleural fistula may be quantitated by the difference between inhaled and exhaled tidal volumes. In the nonintubated patient, this may be determined with a tight-fitting mask and a fast-responding spirometer; in the intubated patient it is determined by direct attachment of the spirometer to the endotracheal tube. The larger the leak, the greater need to isolate the bronchopleural fistula (DLT, bronchial blocker).

There have been several nonsurgical approaches (use of various mechanical ventilation–chest tube drainage systems) to the treatment of patients with bronchopleural fistula. These approaches consist of one-lung ventilation and differential lung ventilation, including HFV, PEEP to the pleural cavity equal to the intrathoracic PEEP, and unidirectional chest tube valves.

For patients undergoing operative repair, the ability to deliver intraoperative positive-pressure ventilation adequately must be carefully considered preoperatively. If a fistula is obviously small, chronic, and uninfected, a standard endotracheal tube can likely be used safely. When in doubt, positive-pressure ventilation can be tested, and if it is found inadequate, the standard endotracheal tube can be replaced with a DLT. During the induction of anesthesia, suction on the chest tube should be discontinued to decrease the loss of tidal volume with the initiation of positive-pressure ventilation. If, after the chest is opened, an excessive leak is encountered when using a standard endotracheal tube, ventilation can be improved by lung packing and manual control of the air leak. 591

For large fistulas or fistulas of unknown size and for those in which an associated abscess or empyema is known or suspected to be present, intraoperative use of a DLT has the dual benefits of permitting positive-pressure ventilation of the normal lung without loss of the minute ventilation through the fistula and preventing the hazard of contamination of the uninfected lung with infected material when the patient is turned to the LDP. 592, 593, 594 Indeed, in one series of 22 patients undergoing operations for bronchopleural fistula after pulmonary resection for tuberculosis or tuberculous empyema, management with a single-lumen endotracheal tube, despite intubation in the head-up position and frequent suctioning, resulted in extensive contamination of normal lung in two patients. 595 In one patient the operation had to be terminated, and in the other emergency bronchoscopy had to be performed. The use of a DLT effectively isolates the leaking and perhaps infected lung cavity from the normal lung. For patients who cannot have a DLT (e.g., very small children, patients with inability to tolerate being taken off the ventilator, those with anatomic difficulties), bronchial blockade and endobronchial intubation are less satisfactory alternatives.

Lung Abscesses and Empyema

General Considerations

Pulmonary aspiration secondary to alcoholic stupor has classically been reported as the most common factor that precipitates lung abscess. Other historical factors that predispose patients to lung abscess formation consist of abuse of other drugs, prior pneumonia, lung carcinoma, immunosuppression with steroid drugs, diabetes mellitus, the presence of a distant septic focus (hematogenous spread), and COPD. 596

Empyema is the accumulation of pus in the pleural cavity. All the causes of a lung abscess described above may produce empyema. Empyema may also be caused by infection of residual clotted blood after a hemothorax (which was treated by chest tube placement) and after diagnostic thoracentesis, especially if there is a concurrent intra-abdominal injury or infection. 597 Both a lung abscess and an empyema may erode a bronchus and cause a bronchopleural fistula (see earlier).

Surgical Considerations

Simple empyemas (without abscess) may be treated by repeat thoracentesis, tube thoracostomy, thoracoscopy, or open drainage with rib resection. 598 In any patient who does not improve with any of these therapies, an open drainage procedure followed by either lobectomy or segmentectomy is required. 599, 600

Anesthetic Considerations

If a surgical procedure is to be performed with the patient under general anesthesia in a patient with either a lung abscess or an empyema, DLT intubation is absolutely indicated to prevent contamination of the uninfected lung by the infected lung. In addition, if an empyema is to be treated with thoracoscopy, collapse of the diseased lung greatly aids access to the empyema. 601, 602 The seal of the endobronchial cuff should be tight and can be quantitated by a bubble-underwater technique. The position of the tube should be determined by fiberoptic bronchoscopy. Both these maneuvers should be done before the patient is turned to the LDP.

During the surgical procedure, the diseased side should be suctioned frequently (fiberoptically if necessary). Whenever possible, but particularly at the end of the procedure, the diseased lung should be fully expanded manually. When the lung has been fully expanded, the surgeon should check carefully for the presence of a bronchopleural fistula. The pleural cavity may be irrigated with antibiotics at the end of the procedure, and one must be mindful of the neuromuscular blockade effects of antibiotics.