CHAPTER 24

# Anesthesia for Patients with Respiratory Disease

## KEY CONCEPTS

1. In a patient with an acute asthma attack, a normal or high $Paco_2$ indicates that the patient can no longer maintain the work of breathing and is often a sign of impending respiratory failure. A pulsus paradoxus and electrocardiographic signs of right ventricular strain (ST-segment changes, right axis deviation, and right bundle branch block) are also indicative of severe airway obstruction.
2. Asthmatic patients with active bronchospasm presenting for emergency surgery should be treated aggressively. Supplemental oxygen, aerosolized $\beta_2$-agonists, and intravenous glucocorticoids can dramatically improve lung function in a few hours.
3. Intraoperative bronchospasm is usually manifested as wheezing, increasing peak airway pressures (plateau pressure may remain unchanged), decreasing exhaled tidal volumes, or a slowly rising waveform on the capnograph.
4. Other causes, such as obstruction of the tracheal tube from kinking, secretions, or an overinflated balloon; bronchial intubation; active expiratory efforts (straining); pulmonary edema or embolism; and pneumothorax, can simulate bronchospasm.
5. Chronic obstructive pulmonary disease (COPD) is currently defined as a disease state characterized by airflow limitation that is not fully reversible. The chronic airflow limitation of this disease is due to a mixture of small and large airway disease (chronic bronchitis/bronchiolitis) and parenchymal destruction (emphysema), with the representation of these two components varying from patient to patient.
6. Cessation of smoking is the long-term intervention that has been shown to reduce the rate of decline in lung function.
7. Preoperative interventions in patients with COPD aimed at correcting hypoxemia, relieving bronchospasm, mobilizing and reducing secretions, and treating infections may decrease the incidence of postoperative pulmonary complications. Patients at greatest risk of complications are those with preoperative pulmonary function measurements less than 50% of predicted.
8. Restrictive pulmonary diseases are characterized by decreased lung compliance. Lung volumes are typically reduced, with preservation of normal expiratory flow rates. Thus, both forced expiratory volume in 1 sec ($FEV_1$) and forced vital capacity (FVC) are reduced, but the $FEV_1$/FVC ratio is normal.
9. Intraoperative pulmonary embolism usually presents as unexplained cardiovascular collapse, hypoxemia, or bronchospasm. A decrease in end-tidal $CO_2$ concentration is also suggestive of pulmonary embolism, but is not specific.

The impact of preexisting pulmonary disease on respiratory function during anesthesia and in the postoperative period is predictable: Greater degrees of preoperative pulmonary impairment are associated with more marked intraoperative alterations in respiratory function and higher rates of postoperative pulmonary complications. Failure to recognize patients who are at increased risk is a frequent contributory factor leading to complications, as patients may not receive appropriate preoperative and intraoperative care. This chapter examines pulmonary risk in general and then reviews the anesthetic approach in patients with the most common types of respiratory disease.

## PULMONARY RISK FACTORS

**Certain risk factors (Table 24–1) may predispose patients to postoperative pulmonary complications**. The incidence of atelectasis, pneumonia, pulmonary embolism, and respiratory failure following surgery is quite high, but varies widely (from 6% to 60%), depending on the patient population studied and the surgical procedures performed. The two strongest predictors of complications seem to be operative site and a history of dyspnea, which correlate with the degree of preexisting pulmonary disease.

The association between smoking and respiratory disease is well established; abnormalities in maximal midexpiratory flow (MMEF) rates are often demonstrable well before symptoms of COPD appear. Although abnormalities can be demonstrated on pulmonary function tests (PFTs), because most patients who smoke do not have PFTs performed preoperatively, it is best to assume that such patients have some degree of pulmonary compromise. Even in normal individuals, advancing age is associated with an increasing prevalence of pulmonary disease and an increase in closing capacity. Obesity decreases functional residual capacity (FRC), increases the work of breathing, and predisposes patients to deep venous thrombosis.

Thoracic and upper abdominal surgical procedures can have marked effects on pulmonary function. Operations near the diaphragm often result in diaphragmatic dysfunction and a restrictive ventilatory defect (see below). Upper abdominal procedures consistently decrease FRC (60% to 70%); the effect is maximal on the first postoperative day and usually lasts 7–10 days. Rapid shallow breathing with an ineffective cough caused by pain (splinting), a decrease in the number of sighs, and impaired mucociliary clearance lead to microatelectasis and loss of lung volume. Intrapulmonary shunting promotes hypoxemia. Residual anesthetic effects, the recumbent position, sedation from opioids, abdominal distention, and restrictive dressings may also be contributory. Complete relief of pain with regional

**TABLE 24–1 Risk factors for postoperative pulmonary complications.**

| Patient-related Factors[1] | Procedure-related Factors[1] |
|---|---|
| **Supported by good evidence** | |
| Advanced age | Aortic aneurysm repair |
| ASA class ≥2 | Thoracic surgery |
| Congestive heart failure | Abdominal surgery |
| Functional dependency | Upper abdominal surgery |
| Chronic obstructive pulmonary disease | Neurosurgery |
| | Prolonged surgery |
| | Head and neck surgery |
| | Emergency surgery |
| | Vascular surgery |
| | Use of general anesthesia |
| **Supported by fair evidence** | |
| Weight loss | Perioperative transfusion |
| Impaired sensorium | |
| Cigarette use | |
| Alcohol use | |
| Abnormal chest exam | |
| **Good evidence *against* being a risk factor** | |
| Well-controlled asthma | Hip surgery |
| Obesity | Genitourinary/gynecologic surgery |
| **Insufficient data** | |
| Obstructive sleep apnea[2] | Esophageal surgery |
| Poor exercise capacity | |

ASA, American Society of Anesthesiologists.

[1]Within each evidence category, risk factors are listed according to strength of evidence, with the first factor listed having the strongest evidence.

[2]Subsequent evidence indicates that this is a probable risk factor.

Data from Smetana GW, Lawrence VA, Cornell JE, et al: Preoperative pulmonary risk stratification for noncardiothoracic surgery: systematic review for the American College of Physicians, Ann Intern Med 2006;144(8):581-595.

**TABLE 24–2 Recommendations of the American College of Physicians to reduce perioperative pulmonary complications in patients undergoing noncardiothoracic surgery.**

| |
|---|
| **Recommendation 1:**<br>• All patients undergoing noncardiothoracic surgery should be evaluated for the presence of the following significant risk factors for postoperative pulmonary complications in order to receive pre- and postoperative interventions to reduce pulmonary risk: chronic obstructive pulmonary disease, age older than 60 years, American Society of Anesthesiologists class of II or greater, functionally dependent, and congestive heart failure.<br>• The following are not significant risk factors for postoperative pulmonary complications: obesity and mild or moderate asthma. |
| **Recommendation 2:**<br>• Patients undergoing the following procedures are at higher risk for postoperative pulmonary complications and should be evaluated for other concomitant risk factors and receive pre- and postoperative interventions to reduce pulmonary complications: prolonged surgery (>3 hours), abdominal surgery, thoracic surgery, neurosurgery, head and neck surgery, vascular surgery, aortic aneurysm repair, emergency surgery, and general anesthesia. |
| **Recommendation 3:**<br>• A low serum albumin level (<35 g/L) is a powerful marker of increased risk for postoperative pulmonary complications and should be measured in all patients who are clinically suspected of having hypoalbuminemia; measurement should be considered in patients with one or more risk factors for perioperative pulmonary complications. |
| **Recommendation 4:**<br>• All patients who after preoperative evaluation are found to be at higher risk for postoperative pulmonary complications should receive the following postoperative procedures in order to reduce postoperative pulmonary complications: deep breathing exercises or incentive spirometry and the selective use of a nasogastric tube (as needed for postoperative nausea or vomiting, inability to tolerate oral intake, or symptomatic abdominal distention). |
| **Recommendation 5:**<br>• Preoperative spirometry and chest radiography should not be used routinely for predicting risk for postoperative pulmonary complications.<br>• Preoperative pulmonary function testing or chest radiography may be appropriate in patients with a previous diagnosis of chronic obstructive pulmonary disease or asthma. |
| **Recommendation 6:**<br>• The following procedures should not be used solely for reducing postoperative pulmonary complication risk: right heart catheterization and total parenteral nutrition or total enteral nutrition (for patients who are malnourished or have low serum albumin levels). |

Data from Qaseem A, Snow V, Fitterman N, et al: Risk assessment for and strategies to reduce perioperative pulmonary complication for patients undergoing noncardiothoracic surgery: a guideline from the American College of Physicians. Ann Intern Med 2006;144:576.

anesthesia can decrease, but does not completely reverse these abnormalities. Persistent microatelectasis and retention of secretions favor the development of postoperative pneumonia.

Although many adverse effects of general anesthesia on pulmonary function have been described, the superiority of regional over general anesthesia in patients with pulmonary impairment is not firmly established.

Because of the prevalence of smoking and obesity, many patients may be at increased risk of developing postoperative pulmonary dysfunction. The risk of complications increases if the patient is having a thoracotomy or laparotomy, even if the patient has no risk factors. Patients with known disease should have their pulmonary function optimized preoperatively, with careful consideration given to the choice of general versus regional anesthesia.

The American College of Physicians has established guidelines to assist in the preoperative assessment of patients with pulmonary disease (see Table 24–2).

When patients with a history of dyspnea present without the benefit of a previous workup, the differential diagnosis can be quite broad and may include both primary pulmonary and cardiac pathologies. Diagnostic approaches to evaluating such patients are summarized in Figure 24–1.

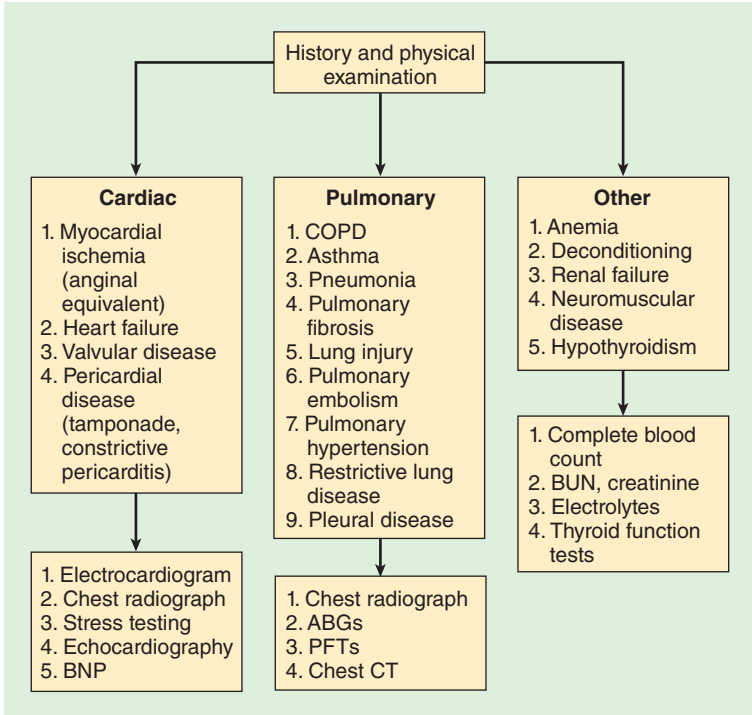


**FIGURE 24–1** Evaluation of dyspnea. ABGs, arterial blood gases; BNP, brain natriuretic peptide; BUN, blood urea nitrogen; COPD, chronic obstructive pulmonary disease; CT, computed tomography; PFTs, pulmonary function tests. (Reproduced, with permission, from Sweitzer BJ, Smetana GW: Identification and evaluation of the patient with lung disease. Anesthesiol Clin 2009;27:673.)

## Obstructive Pulmonary Disease

Obstructive and restrictive breathing are the two most common abnormal patterns, as determined by PFTs. Obstructive lung diseases are the most common form of pulmonary dysfunction. They include asthma, emphysema, chronic bronchitis, cystic fibrosis, bronchiectasis, and bronchiolitis. The primary characteristic of these disorders is resistance to airflow. An MMEF of <70% (forced expiratory flow [$FEF_{25–75\%}$]) is often the only abnormality early in the course of these disorders. Values for $FEF_{25–75\%}$ in adult males and females are normally >2.0 and >1.6 L/sec, respectively. As the disease progresses, both forced expiratory volume in 1 sec ($FEV_1$) and the $FEV_1$/FVC (forced vital capacity) ratio are less than 70% of the predicted values.

Elevated airway resistance and air trapping increase the work of breathing; respiratory gas exchange is impaired because of ventilation/perfusion ($\dot{V}/\dot{Q}$) imbalance. The predominance of expiratory airflow resistance results in air trapping; residual volume and total lung capacity (TLC) increase. Wheezing is a common finding and represents turbulent airflow. It is often absent with mild obstruction that may be manifested initially only by prolonged exhalation. Progressive obstruction typically results first in expiratory wheezing only, and then in both inspiratory and expiratory wheezing. With marked obstruction, wheezing may be absent when airflow has nearly ceased.

## ASTHMA

### Preoperative Considerations

Asthma is a common disorder, affecting 5% to 7% of the population. Its primary characteristic is airway (bronchiolar) inflammation and hyperreactivity in response to a variety of stimuli. Clinically, asthma is manifested by episodic attacks of dyspnea, cough, and wheezing. Airway obstruction, which is generally reversible, is the result of bronchial smooth muscle constriction, edema, and increased secretions.

Classically, the obstruction is precipitated by a variety of airborne substances, including pollens, animal dander, dusts, pollutants, and various chemicals. Some patients also develop bronchospasm following ingestion of aspirin, nonsteroidal antiinflammatory agents, sulfites, or tartrazine and other dyes. Exercise, emotional excitement, and viral infections also precipitate bronchospasm in many patients. Asthma is classified as acute or chronic. Chronic asthma is further classified as intermittent (mild) and mild, moderate, and severe persistent disease.

The terms extrinsic (allergic) asthma (attacks related to environmental exposures) and intrinsic (idiosyncratic) asthma (attacks usually occurring without provocation) were used in the past, but these classifications were imperfect; many patients show features of both forms. Moreover, overlap with chronic bronchitis (see below) is common.

## A. Pathophysiology

The pathophysiology of asthma involves the local release of various chemical mediators in the airway, and, possibly, overactivity of the parasympathetic nervous system. Inhaled substances can initiate bronchospasm through both specific and nonspecific immune mechanisms by degranulating bronchial mast cells. In classic allergic asthma, antigen binding to immunoglobulin E (IgE) on the surface of mast cells causes degranulation. Bronchoconstriction is the result of the subsequent release of histamine; bradykinin; leukotrienes C, D, and E; platelet-activating factor; prostaglandins (PG) $PGE_2$, $PGF_2\alpha$, and $PGD_2$; and neutrophil and eosinophil chemotactic factors. The parasympathetic nervous system plays a major role in maintaining normal bronchial tone; a normal diurnal variation in tone is recognized in most individuals, with peak airway resistance occurring early in the morning (at about 6:00 AM). **Vagal afferents in the bronchi are sensitive to histamine and multiple noxious stimuli, including cold air, inhaled irritants, and instrumentation (eg, tracheal intubation)**. Reflex vagal activation results in bronchoconstriction, which is mediated by an increase in intracellular cyclic guanosine monophosphate (cGMP).

During an asthma attack, bronchoconstriction, mucosal edema, and secretions increase resistance to gas flow at all levels of the lower airways. As an attack resolves, airway resistance normalizes first in the larger airways (main-stem, lobar, segmental, and subsegmental bronchi), and then in more peripheral airways. Consequently, expiratory flow rates are initially decreased throughout an entire forced exhalation, but during resolution of the attack, the expiratory flow rate is reduced only at low lung volumes. TLC, residual volume (RV), and FRC are all increased. In acutely ill patients, RV and FRC are often increased by more than 400% and 100%, respectively. Prolonged or severe attacks markedly increase the work of breathing and can fatigue respiratory muscles. The number of alveolar units with low ($\dot{V}/\dot{Q}$) ratios increases, resulting in hypoxemia. Tachypnea is likely due to stimulation of bronchial receptors and typically produces hypocapnia. A normal or high $Paco_2$ indicates that the patient can no longer maintain the work of breathing and is often a sign of impending respiratory failure. A pulsus paradoxus and electrocardiographic signs of right ventricular strain (ST-segment changes, right axis deviation, and right bundle branch block) are also indicative of severe airway obstruction.

## B. Treatment

Drugs used to treat asthma include β-adrenergic agonists, methylxanthines, glucocorticoids, anticholinergics, leukotriene blockers, and mast cell-stabilizing agents; with the exception of the last, these drugs may be used for either acute or chronic treatment of asthma. Although devoid of any bronchodilating properties, cromolyn sodium and nedocromil are effective in preventing bronchospasm by blocking the degranulation of mast cells.

Sympathomimetic agents (Table 24–3) are the most commonly used for acute exacerbations. They produce bronchodilation via $\beta_2$-agonist activity. Activation of $\beta_2$-adrenergic receptors on bronchiolar smooth muscle stimulates the activity of adenylate cyclase, which results in the formation of intracellular cyclic adenosine monophosphate (cAMP). These agents are usually administered via a metered-dose inhaler or by aerosol. Use of more selective $\beta_2$-agonists, such as terbutaline or albuterol, may decrease the incidence of undesirable $\beta_1$

**TABLE 24–3 A comparison of commonly used bronchodilators.[1]**

| Agent | Adrenergic Activity | |
|---|---|---|
| | $\beta_1$ | $\beta_2$ |
| Albuterol (Ventolin) | + | +++ |
| Bitolterol (Tornalate) | + | ++++ |
| Epinephrine | ++++ | ++ |
| Fenoterol (Berotec) | + | +++ |
| Formaterol (Foradil) | + | ++++ |
| Isoetharine (Bronkosol) | ++ | +++ |
| Isoproterenol (Isuprel) | ++++ | — |
| Metaproterenol (Alupent) | + | + |
| Pirbuterol (Maxair) | + | ++++ |
| Salmeterol (Serevent) | + | ++++ |
| Terbutaline (Brethaire) | + | +++ |

[1]+ Indicates level of activity.

cardiac effects, but are often not particularly selective in high doses.

Traditionally, methylxanthines are thought to produce bronchodilation by inhibiting phosphodiesterase, the enzyme responsible for the breakdown of cAMP. Their pulmonary effects seem much more complex and include catecholamine release, blockade of histamine release, and diaphragmatic stimulation. Oral long-acting theophylline preparations are used for patients with nocturnal symptoms. Unfortunately, theophylline has a narrow therapeutic range; therapeutic blood levels are considered to be 10–20 mcg/mL. Lower levels, however, may be effective. Aminophylline is the only available intravenous theophylline preparation.

Glucocorticoids are used for both acute treatment and maintenance therapy of patients with asthma because of their antiinflammatory and membrane-stabilizing effects. Beclomethasone, triamcinolone, fluticasone, and budesonide are synthetic steroids commonly used in metered-dose inhalers for maintenance therapy. Although they are associated with a low incidence of undesirable systemic effects, their use does not necessarily prevent adrenal suppression. Intravenous hydrocortisone or methylprednisolone is used acutely for severe attacks, followed by tapering doses of oral prednisone. Glucocorticoids usually require several hours to become effective.

Anticholinergic agents produce bronchodilation through their antimuscarinic action and may block reflex bronchoconstriction. Ipratropium, a congener of atropine that can be given by a metered-dose inhaler or aerosol, is a moderately effective bronchodilator without appreciable systemic anticholinergic effects.

## Anesthetic Considerations

### A. Preoperative Management

The emphasis in evaluating patients with asthma should be on determining the recent course of the disease and whether the patient has ever been hospitalized for an acute asthma attack, as well as on ascertaining that the patient is in optimal condition. Patients with poorly controlled asthma or wheezing at the time of anesthesia induction have a higher risk of perioperative complications. Conversely, well-controlled asthma has not been shown to be a risk factor for intraoperative or postoperative complications. A thorough history and physical examination are of critical importance. The patient should have no or minimal dyspnea, wheezing, or cough. Complete resolution of recent exacerbations should be confirmed by chest auscultation. Patients with frequent or chronic bronchospasm should be placed on an optimal bronchodilating regimen. A chest radiograph identifies air trapping; hyperinflation results in a flattened diaphragm, a small-appearing heart, and hyperlucent lung fields. PFTs—particularly expiratory airflow measurements such as $FEV_1$, $FEV_1$/FVC, $FEF_{25-75\%}$, and peak expiratory flow rate—help in assessing the severity of airway obstruction and reversibility after bronchodilator treatment. Comparisons with previous measurements are invaluable.

2 Asthmatic patients with active bronchospasm presenting for emergency surgery should be treated aggressively. Supplemental oxygen, aerosolized $\beta_2$-agonists, and intravenous glucocorticoids

can dramatically improve lung function in a few hours. Arterial blood gases may be useful in managing severe cases. Hypoxemia and hypercapnia are typical of moderate and severe disease; even slight hypercapnia is indicative of severe air trapping and may be a sign of impending respiratory failure.

Some degree of preoperative sedation may be desirable in asthmatic patients presenting for elective surgery—particularly in patients whose disease has an emotional component. In general, benzodiazepines are the most satisfactory agents for premedication. Anticholinergic agents are not customarily given unless very copious secretions are present or if ketamine is to be used for induction of anesthesia. In typical intramuscular doses, anticholinergics are not effective in preventing reflex bronchospasm following intubation. The use of an $H_2$-blocking agent (such as cimetidine, ranitidine, or famotidine) is theoretically detrimental, since $H_2$-receptor activation normally produces bronchodilation; in the event of histamine release, unopposed $H_1$ activation with $H_2$ blockade may accentuate bronchoconstriction.

Bronchodilators should be continued up to the time of surgery; in order of effectiveness, they are β-agonists, inhaled glucocorticoids, leukotriene blockers, mast-cell stabilizers, theophyllines, and anticholinergics. Patients who receive chronic glucocorticoid therapy with more than 5 mg/day of prednisone (or its equivalent) should receive a graduated supplementation schedule based on the severity of the illness and complexity of the surgical procedure. Supplemental doses should be tapered to baseline within 1–2 days.

### B. Intraoperative Management

The most critical time for asthmatic patients undergoing anesthesia is during instrumentation of the airway. General anesthesia by mask or regional anesthesia will circumvent this problem, but neither eliminates the possibility of bronchospasm. In fact, some clinicians believe that high spinal or epidural anesthesia may aggravate bronchoconstriction by blocking sympathetic tone to the lower airways (T1–T4) and allowing unopposed parasympathetic activity. Pain, emotional stress, or stimulation during light general anesthesia can precipitate bronchospasm. Drugs often associated with histamine release (eg, atracurium, morphine, and meperidine) should be avoided or given very slowly when used. The goal of any general anesthetic is a smooth induction and emergence, with anesthetic depth adjusted to stimulation.

The choice of induction agent is less important, if adequate depth of anesthesia is achieved before intubation or surgical stimulation. Thiopental may occasionally induce bronchospasm as a result of exaggerated histamine release. Propofol and etomidate are suitable induction agents; propofol may also produce bronchodilation. Ketamine has bronchodilating properties and is a good choice for patients with asthma who are also hemodynamically unstable. Ketamine should probably not be used in patients with high theophylline levels, as the combined actions of the two drugs can precipitate seizure activity. Halothane and sevoflurane usually provide the smoothest inhalation induction with bronchodilation in asthmatic children. Isoflurane and desflurane can provide equal bronchodilation, but are not normally used for inhalation induction. Desflurane is the most pungent of the volatile agents and may result in cough, laryngospasm, and bronchospasm.

Reflex bronchospasm can be blunted before intubation by an additional dose of the induction agent, ventilating the patient with a 2–3 minimum alveolar concentration (MAC) of a volatile agent for 5 min, or administering intravenous or intratracheal lidocaine (1–2 mg/kg). Note that intratracheal lidocaine itself can initiate bronchospasm if an inadequate dose of induction agent has been used. Administration of an anticholinergic agent may block reflex bronchospasm, but causes excessive tachycardia. Although succinylcholine may on occasion induce marked histamine release, it can generally be safely used in most asthmatic patients. In the absence of capnography, confirmation of correct tracheal placement by chest auscultation can be difficult in the presence of marked bronchospasm.

Volatile anesthetics are most often used for maintenance of anesthesia to take advantage of their potent bronchodilating properties. Ventilation should incorporate warmed humidified gases whenever possible. Airflow obstruction during expiration is apparent on capnography as a delayed rise of the end-tidal $CO_2$ value (**Figure 24–2**); the

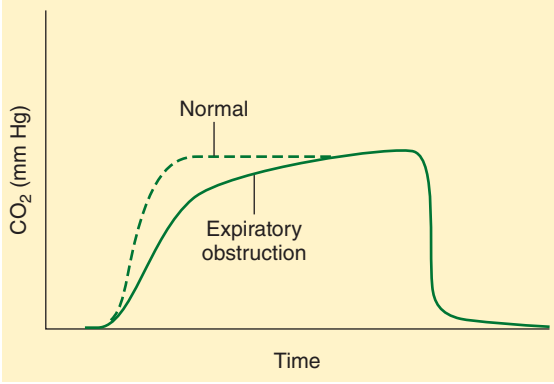


**FIGURE 24–2** Capnograph of a patient with expiratory airway obstruction.

severity of obstruction is generally inversely related to the rate of rise in end-tidal $CO_2$. Severe bronchospasm is manifested by rising peak inspiratory pressures and incomplete exhalation. Tidal volumes of 6–8 mL/kg, with prolongation of the expiratory time, may allow more uniform distribution of gas flow to both lungs and may help avoid air trapping. The $Paco_2$ may increase, which is acceptable if there is no contraindication from a cardiovascular or neurologic perspective.

3 Intraoperative bronchospasm is usually manifested as wheezing, increasing peak airway pressures (plateau pressure may remain unchanged), decreasing exhaled tidal volumes, or a slowly rising 4 waveform on the capnograph. Other causes can simulate bronchospasm: obstruction of the tracheal tube from kinking, secretions, or an overinflated balloon; bronchial intubation; active expiratory efforts (straining); pulmonary edema or embolism; and pneumothorax. Bronchospasm should be treated by increasing the concentration of the volatile agent and administering an aerosolized bronchodilator. Infusion of low dose epinephrine may be needed if bronchospasm is refractory to other interventions.

Intravenous hydrocortisone can be given, particularly in patients with a history of glucocorticoid therapy.

At the completion of surgery, the patient should ideally be free of wheezing. Reversal of nondepolarizing neuromuscular blocking agents with anticholinesterase agents does not precipitate bronchoconstriction, if preceded by the appropriate dose of an anticholinergic agent. Deep extubation (before airway reflexes return) reduces bronchospasm on emergence. Lidocaine as a bolus (1.5–2 mg/kg) may help obtund airway reflexes during emergence.

# CHRONIC OBSTRUCTIVE PULMONARY DISEASE

## Preoperative Considerations

COPD is the most common pulmonary disorder encountered in anesthetic practice, and its prevalence increases with age. The disorder is strongly associated with cigarette smoking and has a male 5 predominance. COPD is currently defined as a disease state characterized by airflow limitation that is not fully reversible. The chronic airflow limitation of this disease is due to a mixture of small and large airway disease (chronic bronchitis/bronchiolitis) and parenchymal destruction (emphysema), with representation of these two components varying from patient to patient.

Most patients with COPD are asymptomatic or only mildly symptomatic, but show expiratory airflow obstruction upon PFTs. In many patients, the obstruction has an element of reversibility, presumably from bronchospasm (as shown by improvement in response to administration of a bronchodilator). With advancing disease, maldistribution of both ventilation and pulmonary blood flow results in areas of low ($\dot{V}/\dot{Q}$) ratios (intrapulmonary shunt), as well as areas of high ($\dot{V}/\dot{Q}$) ratios (dead space).

### A. Chronic Bronchitis

The clinical diagnosis of chronic bronchitis is defined by the presence of a productive cough on most days of 3 consecutive months for at least 2 consecutive years. In addition to cigarette smoking, air pollutants, occupational exposure to dusts, recurrent pulmonary infections, and familial factors may be responsible. Secretions from hypertrophied bronchial mucous glands and mucosal edema from inflammation of the airways produce airflow obstruction. The term "chronic asthmatic

**TABLE 24–4 Signs and symptoms of chronic obstructive pulmonary disease.**

| Feature | Chronic Bronchitis | Emphysema |
|---|---|---|
| Cough | Frequent | With exertion |
| Sputum | Copious | Scant |
| Hematocrit | Elevated | Normal |
| $Paco_2$ (mm Hg) | Often elevated (>40) | Usually normal or <40 |
| Chest radiograph | Increased lung markings | Hyperinflation |
| Elastic recoil | Normal | Decreased |
| Airway resistance | Increased | Normal to slightly increased |
| Cor pulmonale | Early | Late |

bronchitis" may be used when bronchospasm is a major feature. Recurrent pulmonary infections (viral and bacterial) are common and often associated with bronchospasm. RV is increased, but TLC is often normal. Intrapulmonary shunting is prominent, and hypoxemia is common.

In patients with COPD, chronic hypoxemia leads to erythrocytosis, pulmonary hypertension, and eventually right ventricular failure (cor pulmonale); this combination of findings is often referred to as the blue bloater syndrome, but <5% of patients with COPD fit this description (Table 24–4). In the course of disease progression, patients gradually develop chronic $CO_2$ retention; the normal ventilatory drive becomes less sensitive to arterial $CO_2$ tension and may be depressed by oxygen administration (below).

### B. Emphysema

Emphysema is a pathological disorder characterized by irreversible enlargement of the airways distal to terminal bronchioles and destruction of alveolar septa. The diagnosis can be reliably made with computed tomography (CT) of the chest. Mild apical emphysematous changes are a normal, but clinically insignificant, consequence of aging. Significant emphysema is more frequently related to cigarette smoking. Less commonly, emphysema occurs at an early age and is associated with a homozygous deficiency of $\alpha_1$-antitrypsin. This is a protease inhibitor that prevents excessive activity of proteolytic enzymes (mainly elastase) in the lungs; these enzymes are produced by pulmonary neutrophils and macrophages in response to infection and pollutants. Emphysema associated with smoking may similarly be due to a relative imbalance between protease and antiprotease activities in susceptible individuals.

Emphysema may exist in a centrilobular or panlobular form. The centrilobular (or centriacinar) form results from dilatation or destruction of the respiratory bronchioles, is more closely associated with tobacco smoking, and has predominantly an upper lobe distribution. The panlobular (or panacinar) form results in a more even dilatation and destruction of the entire acinus, is associated with $\alpha_1$-antitrypsin deficiency, and has predominantly a lower lobe distribution.

Loss of the elastic recoil that normally supports small airways by radial traction allows premature collapse during exhalation, leading to expiratory flow limitation with air trapping and hyperinflation. Patients characteristically have increases in RV, FRC, TLC, and the RV/TLC ratio. The FRC is shifted rightward along the compliance curve of the lungs, toward the flat portion of the curve, in detriment of the pulmonary mechanics.

Disruption of the alveolar–capillary structure and loss of the acinar structure leads to decreased diffusion lung capacity (DLCO), $\dot{V}/\dot{Q}$ mismatch, and impairment of gas exchange. Also, normal parenchyma may become compressed by the hyperinflated portions of the lung, resulting in a further increase in the $\dot{V}/\dot{Q}$ mismatch. Due to the higher diffusibility of $CO_2$, its elimination is well preserved until $\dot{V}/\dot{Q}$ abnormalities become severe. Chronic $CO_2$ retention occurs slowly and generally results in a compensated respiratory acidosis on blood gas analysis. Arterial oxygen tension is usually normal or slightly reduced. Acute $CO_2$ retention is a sign of impending respiratory failure.

Destruction of pulmonary capillaries in the alveolar septa leads to the development of pulmonary hypertension. However, the degree of

pulmonary hypertension is usually low to moderate, rarely exceeding 35-40 mm Hg.

When dyspneic, patients with emphysema often purse their lips to delay closure of the small airways, which accounts for the term "pink puffers" that is often used (Table 24–4). However, as mentioned above, most patients diagnosed with COPD have a combination of bronchitis and emphysema.

### C. Treatment

Treatment for COPD is primarily supportive. 6 Cessation of smoking is the long-term intervention that has been shown to reduce the rate of decline in lung function. Patients demonstrating a reversible element in airway obstruction (>15% improvement in $FEV_1$ following administration of a bronchodilator) should be started on long-term bronchodilator therapy. Inhaled $\beta_2$-adrenergic agonists, glucocorticoids, and ipratropium are very useful; ipratropium may play a more important role in the management of these patients than in patients with asthma. Even patients who do not show improvement in their PFTs from the use of bronchodilators may improve clinically with bronchodilator therapy. Treatment with systemic corticosteroids may be required in patients with acute exacerbations of COPD. However, systemic corticosteroids in patients with stable COPD is discouraged due to the lack of added benefit and the potential for systemic side effects. COPD exacerbations may be related to bouts of bronchitis, heralded by a change in sputum; frequent treatment with broad-spectrum antibiotics may be necessary. Hypoxemia should be treated carefully with supplemental oxygen. Patients with chronic hypoxemia ($Pao_2$ <55 mm Hg) and pulmonary hypertension require low-flow oxygen therapy (1–2 L/min). Oxygen treatment during acute COPD exacerbations to a $Pao_2$ above 60 mm Hg may lead to $CO_2$ retention, most likely due to an inhibition of the hypoxic vasoconstriction in areas with low $\dot{V}/\dot{Q}$ and the Haldane effect.

When cor pulmonale is present, diuretics are used to control peripheral edema; beneficial effects from vasodilators are inconsistent. Pulmonary rehabilitation may improve the functional status of the patient by improving physical symptoms and exercise capacity. Some studies suggest that the ability to increase oxygen consumption during exercise is inversely related to postoperative complications.

## Anesthetic Considerations

### A. Preoperative Management

Patients with COPD should be prepared prior to elective surgical procedures in the same way as patients with asthma (above). They should be questioned about recent changes in dyspnea, sputum, and wheezing. Patients with an $FEV_1$ less than 50% of predicted (1.2–1.5 L) usually have dyspnea on exertion, whereas those with an $FEV_1$ less than 25% (<1 L in men) typically have dyspnea with minimal activity. The latter finding, in patients with predominantly chronic bronchitis, is also often associated with $CO_2$ retention and pulmonary hypertension. PFTs, chest radiographs, and arterial blood gas measurements, if available, should be reviewed carefully. The presence of bullous changes on the radiograph should be noted. Many patients have concomitant cardiac disease and should also receive a careful cardiovascular evaluation.

In contrast to asthma, only limited improvement in respiratory function may be seen after a short period of intensive preoperative preparation. 7 Nonetheless, preoperative interventions in patients with COPD aimed at correcting hypoxemia, relieving bronchospasm, mobilizing and reducing secretions, and treating infections may decrease the incidence of postoperative pulmonary complications. Patients at greatest risk of complications are those with preoperative pulmonary function measurements less than 50% of predicted. The possibility that postoperative ventilation may be necessary in high-risk patients should be discussed with both the patient and the surgeon.

**Smoking should be discontinued for at least 6–8 weeks before the operation to decrease secretions and to reduce pulmonary complications.** Cigarette smoking increases mucus production and decreases clearance. Both gaseous and particulate phases of cigarette smoke can deplete glutathione and vitamin C and may promote oxidative injury to tissues. Cessation of smoking for as little as 24 hr has theoretical beneficial effects on the oxygen-carrying capacity of hemoglobin; acute inhalation of cigarette smoke releases carbon monoxide, which increases

carboxyhemoglobin levels, as well as nitric oxide, and nitrogen dioxide, which can lead to formation of methemoglobin.

Long-acting bronchodilators and mucolytics should be continued, including on the day of surgery. COPD exacerbations should be treated aggressively.

Preoperative chest physiotherapy and lung expansion interventions with incentive spirometry, deep breathing exercises, cough, chest percussion, and postural drainage may be beneficial in decreasing postoperative pulmonary complications.

### B. Intraoperative Management

Although regional anesthesia is often considered preferable to general anesthesia, high spinal or epidural anesthesia can decrease lung volumes, restrict the use of accessory respiratory muscles, and produce an ineffective cough, leading to dyspnea and retention of secretions. Loss of proprioception from the chest and positions such as lithotomy or lateral decubitus may accentuate dyspnea in awake patients.

Concerns about diaphragmatic paralysis may make interscalene blocks a less attractive option in the lung disease patient.

Preoxygenation prior to induction of general anesthesia prevents the rapid oxygen desaturation often seen in these patients. The selection of anesthetic agents and general intraoperative management must be tailored to the specific needs and goals of every patient. Unfortunately, the use of bronchodilating anesthetics improves only the reversible component of airflow obstruction; significant expiratory obstruction may still present, even under deep anesthesia. Expiratory airflow limitation, especially under positive pressure ventilation, may lead to air trapping, dynamic hyperinflation, and elevated intrinsic positive end-expiratory pressure (iPEEP). Dynamic hyperinflation may result in volutrauma to the lungs, hemodynamic instability, hypercapnia, and acidosis. Interventions to mitigate air trapping include: (1) allowing more time to exhale by decreasing both the respiratory rate and I:E ratio; (2) allowing permissive hypercapnia; (3) applying low levels of extrinsic PEEP; and (4) aggressively treating bronchospasm.

Intraoperative causes of hypotension include pneumothorax, and right heart failure due to hypercapnia and acidosis. A pneumothorax may manifest as hypoxemia, increased peak airway pressures, decreasing tidal volumes, and abrupt cardiovascular collapse unresponsive to fluid and vasopressor administration.

Nitrous oxide should be avoided in patients with bullae and pulmonary hypertension. Inhibition of hypoxic pulmonary vasoconstriction by inhalation anesthetics is usually not clinically significant at the usual doses. However, due to increased dead space, patients with severe COPD have unpredictable uptake and distribution of inhalational agents, and the end-tidal volatile anesthetic concentration is inaccurate.

Measurement of arterial blood gases is desirable for extensive intraabdominal and thoracic procedures. Although pulse oximetry accurately detects significant arterial desaturation, direct measurement of arterial oxygen tensions may be necessary to detect more subtle changes in intrapulmonary shunting. Moreover, arterial $CO_2$ measurements should be used to guide ventilation because increased dead space widens the normal arterial-to-end-tidal $CO_2$ gradient. Moderate hypercapnia with a $Paco_2$ of up to 70 mm Hg may be well tolerated in the short term, assuming a reasonable cardiovascular reserve. Hemodynamic support with inotropic agents may be required in more compromised patients. Hemodynamic monitoring should be dictated by any underlying cardiac dysfunction, as well as the extent of the surgery. In patients with pulmonary hypertension, measurements of central venous pressure reflect right ventricular function rather than intravascular volume.

At the end of surgery, the timing of extubation should balance the risk of bronchospasm with that of respiratory failure, but evidence suggests that early extubation (in the operating room) is beneficial. Successful extubation at the end of the procedure depends on multiple factors: adequate pain control, reversal of neuromuscular blockade, absence of significant bronchospasm and secretions, absence of significant hypercapnia and acidosis, and absence of respiratory depression due to residual anesthetic agents. Patients with an $FEV_1$ below 50% may require a period of postoperative ventilation, particularly following upper abdominal and thoracic operations.

## Restrictive Pulmonary Disease

8 Restrictive pulmonary diseases are characterized by decreased lung compliance. Lung volumes are typically reduced, with preservation of normal expiratory flow rates. Thus, both $FEV_1$ and FVC are reduced, but the $FEV_1$/FVC ratio is normal.

Restrictive pulmonary diseases include many acute and chronic intrinsic pulmonary disorders, as well as extrinsic (extrapulmonary) disorders involving the pleura, chest wall, diaphragm, or neuromuscular function. Reduced lung compliance increases the work of breathing, resulting in a characteristic rapid, but shallow, breathing pattern. Respiratory gas exchange is usually maintained until the disease process is advanced.

## ACUTE INTRINSIC PULMONARY DISORDERS

Acute intrinsic pulmonary disorders include pulmonary edema (including the acute respiratory distress syndrome [ARDS]), infectious pneumonia, and aspiration pneumonitis.

### Preoperative Considerations

Reduced lung compliance in these disorders is primarily due to an increase in extravascular lung water, from either an increase in pulmonary capillary pressure or pulmonary capillary permeability. Increased pressure occurs with left ventricular failure, whereas fluid overload and increased permeability are present with ARDS. Localized or generalized increases in permeability also occur following aspiration or infectious pneumonitis.

### Anesthetic Considerations

#### A. Preoperative Management

Patients with acute pulmonary disease should be spared elective surgery. In preparation for emergency procedures, oxygenation and ventilation should be optimized preoperatively to the greatest extent possible. Fluid overload should be treated with diuretics; heart failure may also require vasodilators and inotropes. Drainage of large pleural effusions should be considered. Similarly, massive abdominal distention should be relieved by nasogastric compression or drainage of ascites. Persistent hypoxemia may require mechanical ventilation.

#### B. Intraoperative Management

Selection of anesthetic agents should be tailored to each patient. Surgical patients with acute pulmonary disorders, such as ARDS, cardiogenic pulmonary edema, or pneumonia, are critically ill; anesthetic management should be a continuation of their preoperative intensive care. Anesthesia is most often provided with a combination of intravenous and inhalation agents, together with a neuromuscular blocking agent. High inspired oxygen concentrations and PEEP may be required. The decreased lung compliance results in high peak inspiratory pressures during positive-pressure ventilation and increases the risk of barotrauma and volutrauma. Tidal volumes for these patients should be reduced to 4–6 mL/kg, with a compensatory increase in the ventilatory rate (14–18 breaths/min), even if the result is an increase in end-tidal $CO_2$. Airway pressure should generally not exceed 30 cm $H_2O$. Airway pressure release ventilation may improve oxygenation in the ARDS patient. The ventilator on the anesthesia machine may prove inadequate for patients with severe ARDS because of its limited gas flow capabilities, low pressure-limiting settings, and the absence of certain ventilatory modes. A more sophisticated intensive care unit ventilator should be used in such instances. Aggressive hemodynamic monitoring is recommended.

## CHRONIC INTRINSIC PULMONARY DISORDERS

Chronic intrinsic pulmonary disorders are also often referred to as interstitial lung diseases. Regardless of etiology, the disease process is generally characterized by an insidious onset, chronic inflammation of alveolar walls and perialveolar tissue, and progressive pulmonary fibrosis. The latter can eventually interfere with gas exchange and ventilatory function. The inflammatory process may be primarily confined to the lungs or may be part of a generalized multiorgan process. Causes include hypersensitivity pneumonitis from occupational and environmental

pollutants, drug toxicity (bleomycin and nitrofurantoin), radiation pneumonitis, idiopathic pulmonary fibrosis, autoimmune diseases, and sarcoidosis. Chronic pulmonary aspiration, oxygen toxicity, and severe ARDS can also produce chronic fibrosis.

### Preoperative Considerations

Patients typically present with dyspnea on exertion and sometimes a nonproductive cough. Symptoms of cor pulmonale are present only with advanced disease. Physical examination may reveal fine (dry) crackles over the lung bases, and, in late stages, evidence of right ventricular failure. The chest radiograph progresses from a "ground-glass" appearance to prominent reticulonodular markings, and, finally, to a "honeycomb" appearance. Arterial blood gases usually show mild hypoxemia with normocarbia. PFTs are typical of a restrictive ventilatory defect (see above), and carbon monoxide diffusing capacity is reduced.

Treatment is directed at abating the disease process and preventing further exposure to the causative agent (if known). Glucocorticoid and immunosuppressive therapy may be used for idiopathic pulmonary fibrosis, autoimmune disorders, and sarcoidosis. If the patient has chronic hypoxemia, oxygen therapy may be started to prevent, or attenuate, right ventricular failure.

### Anesthetic Considerations

#### A. Preoperative Management

Preoperative evaluation should focus on determining the degree of pulmonary impairment as well as the underlying disease process. The latter is important in determining the potential involvement of other organs. A history of dyspnea on exertion (or at rest) should be evaluated further with PFTs and arterial blood gas analysis. A vital capacity of less than 15 mL/kg is indicative of severe dysfunction (normal is >70 mL/kg). A chest radiograph is helpful in assessing disease severity.

#### B. Intraoperative Management

The management of these patients is complicated by a predisposition to hypoxemia and the need to control ventilation to ensure optimum gas exchange; anesthetic drug selection is generally not critical. The reduction in FRC (and oxygen stores) predisposes these patients to rapid hypoxemia following induction of anesthesia. Because these patients may be more susceptible to oxygen-induced toxicity, particularly patients who have received bleomycin, the inspired fractional concentration of oxygen should be kept to the minimum concentration compatible with acceptable oxygenation ($Spo_2$ of >88% to 92%). High peak inspiratory pressures during mechanical ventilation increase the risk of pneumothorax and should prompt adjustment of the ventilatory parameters. In patients with severe restrictive disease, using an I:E ratio of 1:1 (or even an inverse ratio ventilation) and dividing the minute ventilation to a higher respiratory rate (10–15 breaths/minute) may help to maximize the inspiratory time per tidal volume and minimize the peak and plateau ventilatory pressures.

## EXTRINSIC RESTRICTIVE PULMONARY DISORDERS

Extrinsic restrictive pulmonary disorders alter gas exchange by interfering with normal lung expansion. They include pleural effusions, pneumothorax, mediastinal masses, kyphoscoliosis, pectus excavatum, neuromuscular disorders, and increased intraabdominal pressure from ascites, pregnancy, or bleeding. Marked obesity also produces a restrictive ventilatory defect. Anesthetic considerations are similar to those discussed for intrinsic restrictive disorders.

## Pulmonary Embolism

### Preoperative Considerations

Pulmonary embolism results from the entry of blood clots, fat, tumor cells, air, amniotic fluid, or foreign material into the venous system. Clots from the lower extremities, pelvic veins, or, less commonly, the right side of the heart are usually responsible. Venous stasis or hypercoagulability is often contributory in such cases (Table 24–5). Pulmonary embolism can also occur intraoperatively in normal individuals undergoing certain procedures.

**TABLE 24–5 Factors associated with deep venous thrombosis and pulmonary embolism.**

| |
|---|
| Prolonged bed rest |
| Postpartum state |
| Fracture of the lower extremities |
| Surgery on the lower extremities |
| Carcinoma |
| Heart failure |
| Obesity |
| Surgery lasting more than 30 min |
| Hypercoagulability |
| Antithrombin III deficiency |
| Protein C deficiency |
| Protein S deficiency |
| Plasminogen-activator deficiency |

### A. Pathophysiology

Embolic occlusions in the pulmonary circulation increase dead space, and, if minute ventilation does not change, this increase in dead space should theoretically increase $Paco_2$. However, in practice, hypoxemia is more often seen. Pulmonary emboli acutely increase pulmonary vascular resistance by reducing the cross-sectional area of the pulmonary vasculature, causing reflex and humoral vasoconstriction. Localized or generalized reflex bronchoconstriction further increases areas with low ($\dot{V}/\dot{Q}$) ratios. The net effect is an increase in $\dot{V}/\dot{Q}$ mismatch and hypoxemia. The affected area loses its surfactant within hours and may become atelectatic within 24–48 hr. Pulmonary infarction occurs if the embolus involves a large vessel and collateral blood flow from the bronchial circulation is insufficient for that part of the lung (incidence <10%). In previously healthy persons, occlusion of more than 50% of the pulmonary circulation (massive pulmonary embolism) is necessary before sustained pulmonary hypertension is seen. Patients with preexisting cardiac or pulmonary disease can develop acute pulmonary hypertension with occlusions of lesser magnitude. A sustained increase in right ventricular afterload can precipitate acute right ventricular failure. If the patient survives acute pulmonary thromboembolism, the thrombus usually begins to resolve within 1–2 weeks.

### B. Diagnosis

Clinical manifestations of pulmonary embolism include sudden tachypnea, dyspnea, chest pain, or hemoptysis. The latter generally implies lung infarction. Symptoms are often absent or mild and nonspecific unless massive embolism has occurred. Wheezing may be present on auscultation. Arterial blood gas analysis typically shows mild hypoxemia with respiratory alkalosis (the latter due to an increase in ventilation). The chest radiograph is commonly normal, but may show an area of oligemia (radiolucency), a wedge-shaped density with an infarct, atelectasis with an elevated diaphragm, or an asymmetrically enlarged proximal pulmonary artery with acute pulmonary hypertension. Cardiac signs include tachycardia and wide fixed splitting of the $S_2$ heart sound; hypotension with elevated central venous pressure is usually indicative of right ventricular failure. The electrocardiogram frequently shows tachycardia and may show signs of acute cor pulmonale, such as new right axis deviation, right bundle branch block, and tall peaked T waves. Ultrasound studies of the lower extremities also may be helpful in demonstrating deep venous thrombosis. The diagnosis of embolism is more difficult to make intraoperatively (see below).

Pulmonary angiography is still the gold standard criterion for diagnosing a pulmonary embolism, but it is invasive and difficult to perform. Therefore, the less invasive spiral computed tomography angiography (CTA) is the initial imaging of choice in stable patients with suspected pulmonary embolism. Ventilation-perfusion ($\dot{V}/\dot{Q}$) scanning may also be used when CTA cannot be performed. High-, intermediate-, and low-probability criteria have been established for the diagnosis of pulmonary embolism by $\dot{V}/\dot{Q}$ scan.

### C. Treatment and Prevention

The best treatment for pulmonary embolism is prevention. Heparin (unfractionated heparin 5000 U subcutaneously every 12 h begun preoperatively or immediately postoperatively in high-risk patients), enoxaparin or other related compounds, oral anticoagulation (warfarin), aspirin, or dextran therapy, together with early ambulation, can all be used to reduce the incidence of deep vein thrombosis. The use of high elastic stockings and pneumatic compression of the legs may also decrease the incidence

of venous thrombosis in the legs, but not in the pelvis or the heart.

After a pulmonary embolism, systemic anticoagulation prevents the formation of new blood clots or the extension of existing clots. Heparin therapy is begun with the goal of achieving an activated partial thromboplastin time of 1.5–2.4 times normal. Low molecular-weight heparin (LMWH) is as effective and is given subcutaneously at a fixed dose (based on body weight) without laboratory monitoring. In high-risk patients, LMWH is started either 12 hr before surgery, 12–24 hr after surgery, or at 50% the usual dose 4–6 hr after surgery. All patients should start warfarin therapy concurrent with starting heparin therapy, and the two should overlap for 4–5 days. The international normalized ratio should be within the therapeutic range on two consecutive measurements, at least 24 hr apart, before the heparin is stopped. Warfarin should be continued for 3–12 months. Thrombolytic therapy with tissue plasminogen activator or streptokinase is indicated in patients with massive pulmonary embolism or circulatory collapse. Recent surgery and active bleeding are contraindications to anticoagulation and thrombolytic therapy. In these cases, an inferior vena cava umbrella filter may be placed to prevent recurrent pulmonary emboli. Pulmonary embolectomy may be indicated for patients with massive embolism in whom thrombolytic therapy is contraindicated.

## Anesthetic Considerations

### A. Preoperative Management

Patients with acute pulmonary embolism may present in the operating room for placement of an IVC filter, or, rarely, for pulmonary embolectomy. In most instances, the patient will have a history of pulmonary embolism and presents for unrelated surgery; in this group of patients, the risk of interrupting anticoagulant therapy perioperatively is unknown. If the acute episode is more than 1 year old, the risk of temporarily stopping anticoagulant therapy is probably small. Moreover, except in the case of chronic recurrent pulmonary emboli, pulmonary function has usually returned to normal. The emphasis in the perioperative management of these patients should be in preventing new episodes of embolism (see above).

### B. Intraoperative Management

Vena cava filters are usually placed percutaneously under local anesthesia with sedation.

Patients presenting for pulmonary embolectomy are critically ill. They are usually already intubated, but tolerate positive-pressure ventilation poorly. Inotropic support is necessary until the clot is removed. They also tolerate all anesthetic agents very poorly. Small doses of an opioid, etomidate, or ketamine may be used, but the latter can theoretically increase pulmonary artery pressures. Cardiopulmonary bypass is required.

### C. Intraoperative Pulmonary Embolism

Significant pulmonary embolism is a rare occurrence during anesthesia. Diagnosis requires a high index of suspicion. Air emboli are common, but are often overlooked unless large amounts are entrained. Fat embolism can occur during orthopedic procedures; amniotic fluid embolism is a rare, unpredictable, and often fatal, complication of obstetrical delivery. Thromboembolism may occur intraoperatively during prolonged procedures. The clot may have been present prior to surgery or may form intraoperatively; surgical manipulations or a change in the patient's position may then dislodge the venous thrombus. Manipulation of tumors with intravascular extension can similarly produce pulmonary embolism.

9 Intraoperative pulmonary embolism usually presents as sudden cardiovascular collapse, hypoxemia, or bronchospasm. A decrease in end-tidal $CO_2$ concentration is also suggestive of pulmonary embolism, but is not specific. Invasive monitoring may reveal elevated central venous and pulmonary arterial pressures. Depending on the type and location of an embolism, a transesophageal echocardiogram may be helpful. TEE may not reveal the embolus but will often demonstrate right heart distention and dysfunction. **If air is identified in the right atrium, or if it is suspected, emergent central vein cannulation and aspiration of the air may be lifesaving**. For all other emboli, treatment is supportive, with intravenous fluids and inotropes. Placement of a vena cava filter should be considered postoperatively.

## CASE DISCUSSION

### Laparoscopic Surgery

**A 45-year-old woman is scheduled for a laparoscopic cholecystectomy. Known medical problems include obesity and a history of smoking.**

#### *What are the advantages of laparoscopic cholecystectomy compared with open cholecystectomy?*

Laparoscopic techniques have rapidly increased in popularity because of the multiple benefits associated with much smaller incisions than with traditional open techniques. These benefits include decreased postoperative pain, less postoperative pulmonary impairment, a reduction in postoperative ileus, shorter hospital stays, earlier ambulation, and smaller surgical scars. Thus, laparoscopic surgery can provide substantial medical and economic advantages.

#### *How does laparoscopic surgery affect intraoperative pulmonary function?*

The hallmark of laparoscopy is the creation of a pneumoperitoneum with pressurized $CO_2$. The resulting increase in intraabdominal pressure displaces the diaphragm cephalad, causing a decrease in lung compliance and an increase in peak inspiratory pressure. **Atelectasis, diminished FRC, ventilation/perfusion mismatch, and pulmonary shunting contribute to a decrease in arterial oxygenation.** These changes should be exaggerated in this obese patient with a long history of tobacco use.

The high solubility of $CO_2$ increases systemic absorption by the vasculature of the peritoneum. This, combined with smaller tidal volumes because of poor lung compliance, leads to increased arterial $CO_2$ levels and decreased pH.

#### *Why does patient position affect oxygenation?*

A head-down (Trendelenburg) position is commonly requested during insertion of the Veress needle and cannula. This position causes a cephalad shift in abdominal viscera and the diaphragm. FRC, total lung volume, and pulmonary compliance will be decreased. Although these changes are usually well tolerated by healthy patients, this patient's obesity and presumed preexisting lung disease increase the likelihood for hypoxemia. A head-down position also tends to shift the trachea upward, so that a tracheal tube anchored at the mouth may migrate into the right mainstem bronchus. This tracheobronchial shift may be exacerbated during insufflation of the abdomen.

After insufflation, the patient's position is usually changed to a steep head-up position (reverse Trendelenburg) to facilitate surgical dissection. The respiratory effects of the head-up position are the opposite of the head-down position.

#### *Does laparoscopic surgery affect cardiac function?*

Moderate insufflation pressures usually leave heart rate, central venous pressure, and cardiac output unchanged or slightly elevated. This seems to result from increased effective cardiac filling because blood tends to be forced out of the abdomen and into the chest. **Higher insufflation pressures (>25 cm $H_2O$ or 18 mm Hg), however, tend to collapse the major abdominal veins (particularly the inferior vena cava), which decreases venous return and leads to a drop in preload and cardiac output in some patients.**

Hypercarbia, if allowed to develop, will stimulate the sympathetic nervous system and thus increase blood pressure, heart rate, and the risk of arrhythmias. Attempting to compensate by increasing the tidal volume or respiratory rate will increase the mean intrathoracic pressure, further hindering venous return and increasing mean pulmonary artery pressures. These effects can prove particularly challenging in patients with restrictive lung disease, impaired cardiac function, or intravascular volume depletion.

Although the Trendelenburg position increases preload, mean arterial pressure and cardiac output usually either remain unchanged or decrease. These seemingly paradoxical responses may be explained by carotid and aortic baroreceptor-mediated reflexes. The reverse Trendelenburg position decreases preload, cardiac output, and mean arterial pressure.

***Describe the advantages and disadvantages of alternative anesthetic techniques for this patient.***

Anesthetic approaches to laparoscopic surgery include infiltration of local anesthetic with an intravenous sedative, epidural or spinal anesthesia, or general anesthesia. Experience with local anesthesia has been largely limited to brief gynecologic procedures (laparoscopic tubal sterilization, intrafallopian transfers) in young, healthy, and motivated patients. Although postoperative recovery is rapid, patient discomfort and suboptimal visualization of intraabdominal organs preclude the use of this local anesthesia technique for laparoscopic cholecystectomy.

Epidural or spinal anesthesia represents another alternative for laparoscopic surgery. A high level is required for complete muscle relaxation and to prevent diaphragmatic irritation caused by gas insufflation and surgical manipulations. An obese patient with lung disease may not be able to increase spontaneous ventilation to maintain normocarbia in the face of a T2 level regional block during insufflation and a 20° Trendelenburg position. Another disadvantage of a regional technique is the occasional occurrence of referred shoulder pain from diaphragmatic irritation. General anesthesia would therefore be the preferred technique in this patient.

***Does a general anesthetic technique require tracheal intubation?***

Tracheal intubation with positive-pressure ventilation is usually favored for many reasons: the risk of regurgitation from increased intraabdominal pressure during insufflation; the necessity for controlled ventilation to prevent hypercapnia; the relatively high peak inspiratory pressures required because of the pneumoperitoneum; the need for neuromuscular blockade during surgery to allow lower insufflation pressures, provide better visualization, and prevent unexpected patient movement; and the placement of a nasogastric tube and gastric decompression to minimize the risk of visceral perforation during trocar introduction and optimize visualization. The obese patient presented here would benefit from intubation to decrease the likelihood of hypoxemia, hypercarbia, and aspiration.

***What special monitoring should be considered for this patient?***

Monitoring end-tidal $CO_2$ normally provides an adequate guide for determining the minute ventilation required to maintain normocarbia. This assumes a constant gradient between arterial $CO_2$ and end-tidal $CO_2$, which is generally valid in healthy patients undergoing laparoscopy. This assumption would not apply if alveolar dead space changes during surgery. For example, any significant reduction in lung perfusion increases alveolar dead space and therefore increases the gradient between arterial and end-tidal $CO_2$. This may occur during laparoscopy if cardiac output drops because of high inflation pressures, the reverse Trendelenburg position, or gas embolism. Furthermore, abdominal distention lowers pulmonary compliance. Large tidal volumes are usually avoided because they are associated with high peak inspiratory pressures and can cause considerable movement of the surgical field. The resulting choice of lower tidal volumes and higher respiratory rates may lead to poor alveolar gas sampling and erroneous end-tidal $CO_2$ measurements. In fact, end-tidal $CO_2$ values have been found to be particularly unreliable in patients with significant cardiac or pulmonary disease undergoing laparoscopy. Thus, placement of an arterial catheter should be considered in patients with cardiopulmonary disease.

***What are some possible complications of laparoscopic surgery?***

Surgical complications include hemorrhage, if a major abdominal vessel is lacerated, or peritonitis, if a viscus is perforated during trocar introduction. Significant intraoperative hemorrhage may go unrecognized because of the limitations of laparoscopic visualization. Fulguration has been associated with bowel burns and bowel gas explosions. The use of pressurized gas introduces the possibility of extravasation of $CO_2$ along tissue planes, resulting in subcutaneous emphysema,

pneumomediastinum, or pneumothorax. Nitrous oxide should be discontinued and insufflating pressures decreased as much as possible. Patients with this complication may benefit from the continuation of mechanical ventilation into the immediate postoperative period.

Venous $CO_2$ embolism resulting from unintentional insufflation of gas into an open vein may lead to hypoxemia, pulmonary hypertension, pulmonary edema, and cardiovascular collapse. Unlike air embolism, end-tidal $CO_2$ may transiently increase during $CO_2$ gas embolism. Treatment includes immediate release of the pneumoperitoneum, discontinuation of nitrous oxide, insertion of a central venous catheter for gas aspiration, and placement of the patient in a head-down left lateral decubitus position.

Vagal stimulation during trocar insertion, peritoneal insufflation, or manipulation of viscera can result in bradycardia and even sinus arrest. Although this usually resolves spontaneously, elimination of the stimulus (eg, deflation of the peritoneum) and administration of a vagolytic drug (eg, atropine sulfate) should be considered. Intraoperative hypotension may be more common during laparoscopic cholecystectomy than during cholecystectomy by laparotomy. Preoperative fluid loading has been recommended to avoid this complication.

Even though laparoscopic procedures are associated with less muscle trauma and incisional pain than open surgery, pulmonary dysfunction can persist for at least 24 hr postoperatively. For example, forced expiratory volume, forced vital capacity, and forced expiratory flow are reduced by approximately 25% following laparoscopic cholecystectomy, compared with a 50% reduction following open cholecystectomy. The cause of this dysfunction may be related to diaphragmatic tension during the pneumoperitoneum.

Nausea and vomiting are common following laparoscopic procedures, despite routine emptying of the stomach with a nasogastric tube. Pharmacological prophylaxis is recommended.

## GUIDELINES

Qaseem A, Snow V, Fitterman N, et al: Risk assessment for and strategies to reduce perioperative pulmonary complication for patients undergoing noncardiothoracic surgery: a guideline from the American College of Physicians. Ann Intern Med 2006;144:576.

See www.guidelines.gov for additional guidelines from multiple organizations on deep vein thrombosis prophylaxis and pulmonary embolism.

## SUGGESTED READING

Hurford WE: The bronchospastic patient. Int Anesthesiol Clin 2000;38:77.

Reilly JJ Jr: Evidence-based preoperative evaluation candidates for thoracotomy. Chest 1999;116:474.

Smetana G. Postoperative pulmonary complications: an update on risk assessment and reduction. Cleveland Clin J Med 2009;76(S4):S60-S65.

Sweitzer B, Smetana G: Identification and evaluation of the patient with lung disease. Anesthesiol Clin 2009;27:673.