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Anesthesia for thoracic surgery

JAY B BRODSKY

PREOPERATIVE ASSESSMENT

All patients undergoing thoracic surgery must give a complete medical history and have a complete physical examination prior to surgery. The physical examination is directed primarily to the cardiovascular and pulmonary systems. The presence of wheezing, rales, rhonchi, or other abnormal breath sounds suggests the need for further medical intervention. Any deviation of the trachea from the midline should alert the anesthesiologist to a potentially difficult intubation or to the possibility of airway obstruction during induction of anesthesia.

The electrocardiogram (ECG) of the patient with chronic obstructive pulmonary disease may reveal right atrial or ventricular hypertrophy. An enlarged P-wave in lead II ("P" pulmonale) indicates right atrial enlargement. A low-voltage QRS complex may be due to lung hyperinflation.

Many patients undergoing thoracic surgery have pre-existing pulmonary problems, and alterations in pulmonary function are to be expected in every patient after thoracotomy. Pulmonary function tests and an arterial blood gas sample are usually indicated to establish baseline values before major surgery.[1]

[1]

Bedside spirometric tests for forced vital capacity (FVC) and forced expiratory volume (FEV) are usually adequate.[2] FVC can be compared with normal values based on sex, height, and age and then expressed as a percentage of the predicted vital capacity (%FVC). A %FVC greater than 80 is considered normal, 70–80 is borderline normal, 60–70 suggests pulmonary disease, and less than 60 indicates significant restriction of pulmonary function.

The FEV_1 is the volume of air forcefully expired in the first second of an FVC maneuver. FEV_1 can be compared with the actual measured FVC (ratio $FEV_1/FVC = \%FEV_1$) to differentiate between restrictive and obstructive pulmonary disease. The $\%FEV_1$ is normally greater than 70. In restrictive disease, both FEV_1 and FVC decrease, so $\%FEV_1$ remains normal.

In obstructive disease, only FEV_1 is reduced, so $\%FEV_1$ is lowered. A $\%FEV_1$ less than 70 suggests significant airway resistance, which increases the work of breathing during stress. A 15% improvement in $\%FEV_1$ after bronchodilator therapy indicates some reversibility of airway obstruction, and in these patients bronchodilators should be administered preoperatively.

Pulmonary function tests are used to predict the degree to which the pre-existing obstructive and restrictive components of pulmonary function may compromise the ability to ventilate adequately and to maintain clear lungs after thoracic surgery.

An FVC that is at least three times greater than tidal volume is necessary for an effective cough. A preoperative FVC of less than 20 mL kg^{-1}, a FEV_1 of less than 1.2 L, and a $\%FEV_1$ of less than 35 are each associated with postoperative respiratory problems. Improvements in operative and postoperative management of high-risk surgical patients have rendered these absolute measurements invalid. Thoracotomy and video-assisted thoracoscopic surgery are now safely performed in patients who were not previously considered surgical candidates based on the above spirometric criteria.[3]

Flow–volume loop measurements are another means of assessing airway status. The shape and peak air flow rates during expiration at high lung volumes are effort dependent and indicate the patency of the larger airways. Expiration at low lung volumes is effort independent and reflects resistance in smaller airways.

Split-lung ventilation/perfusion studies predict the amount of functional lung tissue remaining after pulmonary resection. These tests are especially useful before pneumonectomy. Radioactive xenon is injected intravenously, and the radioactivity subsequently measured in each lung is proportional to regional perfusion. Ventilation is measured by the inhalation of radioactive gas. The radioactivity measured in each lung area is proportional to the degree of regional ventilation.

Diffusing capacity is another sensitive measure of pulmonary function. Abnormal diffusing capacity measurements may reveal the existence of emphysematous changes in the lung, even when spirometric values are considered acceptable. Decreased diffusing capacity is a strong predictor of both pulmonary and cardiac complications following lung resection.[4]

Pulmonary and cardiac complications continue to present major sources of morbidity and mortality for patients undergoing thoracic operations. Determination of the specific factors associated with increased risk is therefore important so that those patients who might be predisposed to develop serious complications can be identified.[5]

Such factors include carbon monoxide diffusion capacity, peak oxygen (O_2) uptake, pulmonary artery pressure, oxygen tension ($P_{a}O_2$) during pulmonary artery occlusion, and presurgical exercise testing. Patient age, the site and extent of the operative procedure, duration of anesthesia, pre-existing cardiovascular or neurologic dysfunction, obesity, the degree of postoperative pain expected, and the interval between surgery and ambulation affect postoperative outcome.[6–9]

Patients with respiratory muscle weakness have a high incidence of postoperative pulmonary complications. Preoperative respiratory muscle testing and then training can reduce complications by increasing both inspiratory and expiratory muscle strength in patients undergoing thoracic surgery.[10]

A cardiopulmonary risk index (CPRI) has been used to predict postoperative outcome following thoracotomy (Table 47.1).[11] This system assigns numerical values to certain cardiac factors (congestive heart failure, recent myocardial infarction, arrhythmia, aortic stenosis) and to pulmonary factors [obesity, cough, elevated carbon dioxide (P_aCO_2), poor spirometric performance, cigarette smoking, and diffuse wheezing].

A CPRI score of 4 or higher is associated with a 22-fold increase in post-thoracotomy complications compared with patients with a score of less than 4. Other studies have failed to correlate a high CPRI score with an adverse outcome.[12,13]

For patients with significant chronic pulmonary disease or retained secretions, a significant improvement in vital capacity (VC) and FEV_1 can be obtained preoperatively with appropriate bronchial hygiene therapy (aerosol therapy with or without bronchodilator, chest physiotherapy, intermittent positive pressure breathing).[14] Every patient should be encouraged to cough, breathe deeply, and sit up and ambulate as soon as possible after surgery.

Preoperative spirometry used to predict postoperative FEV_1 is probably the best predictor of outcome.[15] However, no index or comorbidity is completely accurate. With appropriate perioperative management, even patients with severely compromised pulmonary function, such as those undergoing lung volume reduction surgery or pulmonary transplantation, can be safely anesthetized for pulmonary resection.[16]

CHOICE OF ANESTHETIC AGENT

General anesthesia normally increases airway resistance by reducing functional residual capacity (FRC). Airway resistance may be further increased during thoracic surgery by obstruction with secretions or tumor and from surgical trauma that can cause hemorrhage and bronchospasm. Associated medical conditions [chronic obstructive pulmonary disease (COPD), asthma, cystic fibrosis, or other lung diseases] can also affect airway caliber and reactivity. Intentional intubation of a bronchus with a double-lumen tube (DLT) or bronchial blocker can produce bronchospasm from direct mucosal stimulation.

These effects can be partially alleviated by using an inhalational anesthetic agent (Table 47.2). Halothane, enflurane, isoflurane, desflurane, and sevoflurane have direct bronchodilator properties. They also obtund bronchoconstrictive airway reflexes in patients with reactive airways.

Isoflurane, sevoflurane, or desflurane, which allow rapid changes in depth of anesthesia with fewer ventilatory arrhythmias, are preferred for thoracic surgery. No differences in oxygenation, shunt fraction, or hemodynamic stability result from the use of isoflurane or sevoflurane[17,18] or from isoflurane or desflurane[19] during selective one-lung ventilation.

Drugs that release histamine (thiopental, thiamylal, propanidid) can produce bronchospasm. These agents must be used with caution in patients with reactive airway disease. Methohexital, etomidate, and propofol do not cause histamine release and should be considered for anesthetic induction of the patient with asthma.[20]

Some muscle relaxants (vecuronium, mivacurium, curare) also cause histamine release. Pancuronium and cisatracurium are completely devoid of any chemically mediated histamine release and can be used for patients with reactive airways.[21]

Ketamine has direct bronchodilatory effects and antagonizes bronchoconstriction from histamine without depressing respiration. It also maintains the hypoxic pulmonary vasoconstrictive (HPV) response.[22] It has a rapid onset of action while tending to maintain cardiovascular stability. Ketamine is a particularly useful agent for induction of anesthesia in unstable patients undergoing emergency thoracotomy.[23] Ketamine offers no advantages for routine thoracic surgery.[24]

In the lungs, regional hypoxia causes arteriolar constriction with diversion of blood flow away from a hypoxic segment to areas that are better oxygenated. The HPV response improves gas exchange by matching perfusion to ventilation. The primary stimuli for HPV are alveolar oxygen tension (P_AO_2) and mixed venous oxygen tension (P_vO_2).[25] [2]

The overall effects of anesthetics on HPV are complex.[26] In both *in vitro* and *in vivo* animal studies, all intravenous drugs (barbiturates, hypnotics, droperidol, ketamine, opioids) do not depress HPV. In experimental models, all inhalational anesthetics, including the newer agents sevoflurane and desflurane, directly inhibit HPV.[27–30]

The direct depression of HPV by the inhalational anesthetics is believed to be due to release of endogenous nitric oxide (NO).[31] Conversely, inhibition of NO production results in an increased HPV response, even in the presence of an inhalational anesthetic agent.[32]

Although in experiments the inhalational agents all inhibit HPV, clinically a wide range of effects are seen with those same agents. This may be due in part to their effects on cardiac output, oxygen consumption, regional wasted perfusion of the collapsed lung during one-lung ventilation ("shunt"), and P_vO_2, and to mechanical factors such as manipulation of the lung and the use of positive end-expiratory pressure (PEEP) during thoracic surgery.

By directly increasing the shunt by depression of HPV, inhalational

Table 47.1 *Cardiopulmonary risk index (CPRI) for lung resection*

CRI		PRI	
Variable	**Points**	**Variable**	**Points**
Congestive heart failure	11	Obesity	1
Myocardial infarction (within 6 months of surgery)	10	Cigarette smoking (within 8 weeks of surgery)	1
> 5 PVCs min^{-1}	7	Productive cough (within 5 days of surgery)	1
Other arrhythmia	7	Diffuse wheezing or rhonchi (within 5 days or surgery)	1
Age > 70 years	5	$FEV_1/FVC < 70\%$	1
Aortic stenosis	3	$P_aCO_2 > 45$ mmHg	1
Poor general medical condition	3		
Thoracic operation	3		

Score = 1 (0–5 CRI points)
2 (6–12 CRI points)
3 (12–25 CRI points)
4 (>25 CRI points)

CRI, cardiac risk index; PRI, pulmonary risk index; PVCs, ; FEV, forced expiratory volume; FVC, forced vital capacity. CPRI score is obtained by adding the CRI and PRI scores. A minimum CRI score of 3 is assigned to each patient because they are undergoing a thoracic operation. A CPRI score ≥4 was associated with a 22-fold increase in postoperative complications.

Table 47.2 *Properties of anesthetic agents used for thoracic surgery*

Agent	Desirable	Undesirable
Inhalational anesthetics	Permit high $F_{I}O_2$	Inhibit HPV
	Bronchodilator properties	Myocardial depression
	Diminished airway reflexes	
	Rapidly eliminated	
Opioids	Do not inhibit HPV	May depress ventilation
	Postoperative analgesia	
	No myocardial depression	
Nitrous oxide	Rapidly eliminated	Reduces $F_{I}O_2$
	No effect on HPV	May expand blebs, tube cuffs
Ketamine	Diminishes airway irritability	Emergence delirium
	Does not inhibit HPV	
	Cardiovascular stability during hypovolemia	
Thiopental	Does not inhibit HPV	Releases histamine
Propofol	Does not inhibit HPV	
	Rapid emergence	
Muscle relaxants	Facilitate mechanical ventilation	Potential postoperative weakness
	Improve surgical exposure	Release histamine
	Reduced dose of anesthetic	Requires reversal agent

HPV, hypoxic pulmonary vasoconstriction.

agents produce modest reductions in P_aO_2. However, if the anesthetic lowers cardiac output more than it decreases oxygen consumption, P_vO_2 will drop. This is a potent stimulus for HPV. Therefore, the effectiveness of the HPV response varies inversely with cardiac output. A myocardial depressant such as halothane, which decreases cardiac output, reduces blood flow to the collapsed lung, thereby neutralizing any direct depression of HPV during one-lung ventilation.[33]

In clinical practice, the overall effects by inhalational and intravenous anesthetic agents on HPV during thoracotomy are small.[34,35] Intravenous propofol did not increase shunt fraction during one-lung ventilation, whereas shunt did increase threefold in patients receiving isoflurane.[36] However, there is no difference in oxygenation between a total intravenous (propofol–alfentanil) anesthetic which spares the HPV response, and an inhalational anesthetic (isoflurane) which depresses HPV during one-lung ventilation.[37]

Vasoconstrictive drugs [dopamine, epinephrine (adrenaline), phenylephrine] preferentially constrict pulmonary vessels perfusing normoxic or hyperoxic lung segments during one-lung ventilation. This increases pulmonary vascular resistance in the ventilated lung, causing redistribution of blood flow to the collapsed lung, lowering P_aO_2, and possibly increasing the shunt.

Vasodilator drugs (nitroprusside, nitroglycerin) blunt HPV in the atelectactic lung during one-lung ventilation. This increases blood flow to that lung, thereby lowering P_aO_2. Similarly, manipulation of the collapsed lung during surgery causes endogenous thromboxane and prostaglandin I_2-mediated local vasodilation, which may blunt HPV.[38]

[3]

Nitrous oxide (N_2O) expands air-containing spaces (such as a pneumothorax, lung cyst, or an air bleb) and dilutes the amount of oxygen that can be delivered during one-lung ventilation. There is no reason to use N_2O during thoracic surgery. If exposure to high oxygen concentration is undesirable, as in the patient receiving bleomycin,[39] then air should be substituted for N_2O. There may even be a lower incidence of postoperative atelectasis in the ventilated lung after pulmonary surgery when air is substituted for N_2O during one-lung ventilation.[40]

INTRAOPERATIVE MONITORING

Routine monitoring during thoracic operations should include noninvasive blood pressure, pulse oximetry, end-tidal capnography, ECG, and temperature. A urinary catheter should also be placed before long procedures and/or when epidural opioids are planned.

During one-lung ventilation, monitoring oxygenation by oxygen saturation (S_pO_2) is usually adequate.[41] However, in the presence of prolonged hypotension, pulse oximetry may be inaccurate. An indwelling arterial line allows intermittent sampling of arterial blood to monitor oxygenation, ventilation, and acid–base status. It is also useful for beat-to-beat hemodynamic monitoring, especially in situations in which surgical retraction on the heart or great vessels can cause hypotension and arrhythmias.

Continuous intra-arterial blood gas monitoring (CIABG) is now feasible.[42,43] This technology has been used successfully during thoracic surgery,[44] although PO_2 values are relatively inaccurate during one-lung ventilation.[45]

Since intermittent arterial blood gas sampling may fail to detect transient arterial hypoxemia during one-lung ventilation, the application of CIABG as a trend monitor in critical situations such as lung transplantation, lung volume reduction surgery, and pulmonary alveolar lavage seems reasonable.[46] Because of the high cost of the intra-arterial probes, at present there is no role for CIABG during routine thoracotomy or video-assisted thoracoscopic surgery.

Fluid status monitoring with a central venous pressure (CVP) or pulmonary artery (PA) line is not routinely needed during thoracotomy because volume changes are not large. Fluid restriction is indicated for most pulmonary resections.[47] Less than 20 mL kg^{-1} of crystalloid solution over 24 h is recommended, and increasing fluid input to achieve a urine output of 0.5 mL kg^{-1} h^{-1} is not necessary.[48]

Pulmonary edema can occur following any thoracotomy from a variety of causes, including fluid overload, cardiac failure, and aspiration. For procedures with large volume shifts (esophageal resection, pneumonectomy), postoperative volume monitoring with a central catheter is normally indicated.

Open thoracotomy in the lateral decubitus position limits the usefulness of information derived from CVP or PA catheters during

surgery. Normally, the CVP reflects blood volume, venous tone, and right ventricular performance. During thoracotomy, the CVP may be altered by surgical retraction, mediastinal and diaphragmatic shifts, and the application of PEEP, which changes intrathoracic pressure. With advanced pulmonary disease and/or left ventricular dysfunction, the CVP may not reflect left-sided filling pressures.

[4] A PA catheter for monitoring hemodynamic function (preload, afterload, and cardiac output) may be useful in patients with pre-existing myocardial dysfunction, ischemia, and valvular heart disease. A PA catheter can also be used to follow $S_{\bar{v}}O_2$, which is a reflection of oxygen supply and demand. $S_{\bar{v}}O_2$ gives indirect information of end-organ perfusion and may reflect decreases in cardiac output, decreased arterial oxygen content, anemia, or increased oxygen consumption.[49]

When a PA catheter is used, its position should be documented radiographically before surgery since catheters enter the right pulmonary artery 85% of the time. Therefore, during right thoracotomy the catheter tip will be in the nondependent pulmonary artery, and during left thoracotomy the catheter tip will be in the vessel of the nonoperated, dependent lung. In both situations, the data obtained may be inaccurate. Hemodynamic and $S_{\bar{v}}O_2$ measurements are lower during right thoracotomy than during left thoracotomy because both are affected by reduced blood flow to the nonventilated right lung.

Transesophageal echocardiography (TEE) has been used during pulmonary surgery. Clamping the pulmonary artery during pneumonectomy was associated with transient left ventricular dysfunction and mild mitral regurgitation from acute right ventricular dilation.[50] TEE monitoring of high-risk patients with associated cardiac disease during pneumonectomy may help to identify those patients who will go on to develop right ventricular failure and/or hypoxemia in the postoperative period. There were no TEE changes during pulmonary lobectomy. During thoracotomy, TEE can also identify tumor in pulmonary veins, tumor embolism, and tumor invasion into the atrium.[51,52]

ISOLATION OF THE LUNGS

During intrathoracic procedures in which the operated lung is selectively collapsed, hemodynamic stability and oxygenation must be maintained while ventilating only one lung. Isolation of the lungs is essential when it is necessary to protect the dependent lung from contamination while the patient is in the lateral decubitus position.[53,54] Collapsing the operated lung provides optimal operative exposure.

Although selective one-lung ventilation is required for many procedures (e.g. repair of a bronchopleural fistula, lung transplantation, and video-assisted and lung volume reduction surgery), any thoracic surgery will benefit from selective lung collapse. A completely atelectactic lung eliminates the need for vigorous retraction during surgery, so there is less intraoperative lung trauma. Improved exposure shortens the duration of the procedure, therefore total operative costs are also reduced. Isolation and selective collapse of a lung can be achieved by either bronchial blockade or with a double-cuffed, double-lumen endobronchial tube (DLT).

Bronchial blockade

Lung tissue distal to any obstruction will collapse from absorption atelectasis. Historically, bronchial blockade has been achieved with a variety of different devices, including gauze tampons, special cuffed rubber blockers, and with the inflated balloons used for embolectomy, pulmonary artery, and even urinary catheters.[55,56] Bronchial blockade with a Fogarty embolectomy or some other small (4–6Fr) balloon-tipped catheter is the only practical method for lung separation in small children.[57] Larger (8–12Fr) embolectomy catheters are occasionally used for adults.[58,59] In patients with a restricted mouth opening, a nasal intubation with an endotracheal tube followed by bronchial blockade with a Fogarty catheter may be the only practical means of achieving one-lung ventilation.[60] Bronchial blockade should also be considered for patients with a previous laryngectomy and permanent tracheostomy when the stoma is too small to accommodate a DLT.[61]

In adults, the blocker catheter is passed, using a flexible fiberoptic bronchoscope, through an endotracheal tube, whereas in children it is passed alongside the endotracheal tube. Once the catheter is in position in the bronchus, its balloon is inflated until it completely obstructs the airway.

Blockers have several disadvantages. They can be easily displaced when changing the position of the patient or during surgical manipulation. If the blocker slips into the trachea, it may obstruct ventilation to the nonoperated lung and/or fail to isolate the healthy lung from contamination. Another disadvantage is that lung tissue distal to the obstruction cannot be suctioned or re-expanded during the procedure. Continuous positive airway pressure (CPAP) cannot be applied if the patient becomes hypoxemic during one-lung ventilation.

All catheters currently used for bronchial blockade are manufactured for other uses and have low-volume balloons that generate very high pressures when inflated. Tracheobronchial rupture can occur if the balloon is overinflated or if it becomes overdistended with N_2O during the anesthetic.[62] The largest balloon whose catheter will fit the endotracheal tube should be used. The balloon should be inflated with the smallest volume of air that seals the bronchus while under direct observation.

The Univent tube (Fuji Systems, Tokyo, Japan) is designed for bronchial blockade in adults and larger children.[63–65] The tube is a conventional endotracheal tube with an additional small lumen. This lumen contains another smaller tube that can be advanced up to 8 cm past the tip of the larger tube into either bronchus. A balloon located near the tip of the smaller tube, when inflated, serves as the airway blocker while the nonoperated lung is ventilated through the larger endotracheal tube. Bronchoscopy is required for accurate placement of Univent tubes.[66,67]

The operated lung can be suctioned or lavaged, and supplemental oxygen can be insufflated through the lumen of the blocker tube.[68,69] High-frequency jet ventilation can also be used through this lumen.[70] Ventilation to both lungs can be reinstituted at any time by deflating the blocker balloon and withdrawing the blocker back into the body of the main tube. A major advantage of the Univent tube is that the airway need not be reintubated with another endotracheal tube at the completion of surgery if postoperative ventilation is planned.

Although some consider a Univent tube is easier to use than a DLT,[71,72] the time required to position both tubes, the number of tube malpositions, and the surgical exposure provided by each did not differ when the Univent tube was compared with a DLT.[73] Since the blocker tube is so small, its balloon must be inflated with 2–10 times as much air as the bronchial cuff of a DLT in order for it to obstruct the bronchus.[74] This generates high, potentially dangerous pressures that predispose the balloon to carinal herniation or can damage the airway.[75]

Several unique complications have been reported with the Univent tube.[76] A tension pneumothorax can occur when the rigid blocker tube is advanced into the airway wall.[77] Failure to withdraw the blocker completely at the time of airway resection can result in inclusion of the blocker in the staple line.[78] Obstruction of ventilation to both lungs may result if the cuff on the blocker balloon is retracted completely back into the main tube without deflation. This complication is more likely to occur postoperatively in the critical care setting with personnel who are not familiar with the properties of the Univent tube.[79] Removal of the pilot balloon to the blocker will eliminate the risk of this complication.

Double-lumen tubes

The major advantage of a double-lumen tube (DLT) is that either or both lungs can be deflated, re-expanded, or suctioned at any time during the procedure. In addition, CPAP can be administered to the collapsed lung during one-lung ventilation without interrupting ventilation to the nonoperated lung.

Properties

All DLTs are constructed of two tubes of unequal length. The shorter tube ends in the trachea, whereas the longer tube reaches into a bronchus. A cuff placed above the distal opening of the tracheal tube prevents gas leaks during positive pressure ventilation. Inspired gas can be diverted into either or both lungs by a second cuff on the longer tube in the bronchial lumen. The inflated bronchial cuff isolates and protects each lung. In tubes designed for the right bronchus, the lateral aspect of the bronchial cuff or bronchial lumen itself is fenestrated to allow gas exchange with the right upper-lobe bronchus.

The proximal end of each lumen is fitted to a special connector that allows ventilation to be distributed to either or both lungs. Each tube can be independently opened to the atmosphere, thereby allowing the lung on that side to collapse while ventilation to the other lung can continue. A suction catheter or bronchoscope can be passed down either lumen while ventilating the other lung.

The original rubber Carlens's and White's DLTs had a hook to engage the carina to help position the tube. Carinal hooks make passage through the glottis difficult and often injure the airway. The rubber Robertshaw's DLT has no hook and has larger internal lumens than either the Carlens's or the White's tube. DLTs are now made of polyvinyl-chloride plastic material and resemble Robertshaw's design.

Because of their numerous advantages, disposable plastic DLTs have now almost universally replaced reusable rubber tubes.[80] Plastic DLTs are sold by several manufacturers in a range of sizes (26, 28, 32, 35, 37, 39, and 41Fr). An even smaller, uncuffed, double-lumen tube is available for very small children.[81] Each tube differs slightly in bronchial cuff design[74] and in the length of the endobronchial segment distal to the bronchial cuff.[82] The relatively large lumens in all plastic tubes allow easy passage of a suction catheter or bronchoscope to either lung while presenting less resistance to airflow during one-lung ventilation.[83,84] The transparent plastic makes continuous observation of moisture during ventilation and the presence of secretions or blood in either lumen possible. Each bronchial cuff is dyed blue and is easily visualized during fiberoptic bronchoscopy.

In contrast with the low-volume/high-pressure cuffs of rubber DLTs, the bronchial cuff of plastic tubes have high-volume/low-pressure characteristics. This reduces the danger of ischemic pressure damage to the respiratory mucosa.[85,86] Since cuff shape differs among the different DLTs, each tube exerts a different degree of pressure on the airway wall when its bronchial cuff is inflated. As a general rule, less than 2–3 mL of air should be all that is needed for the bronchial cuff of any adult DLT.[87] As cuffs are extremely fragile, care must be taken when intubating the airway (see below).[88]

Choice of double-lumen tube

A malpositioned DLT will usually obstruct the upper-lobe bronchus. Therefore, the rationale for intubating the bronchus of the operative lung (right or left) is that the intubated lung is always visible during surgery. If the tube is not in a satisfactory position, the upper lobe will either be atelectactic when the chest is opened or the lung will fail to collapse when the appropriate lumen is clamped.[89,90] Bronchoscopy is not needed to confirm tube placement or to aid in repositioning.[91] The surgeon can manually help guide the tube if repositioning does become necessary.[92]

The potential disadvantage of placing the DLT in the bronchus of the operated lung is that there is a risk of displacing the tube with surgical manipulation. A DLT in the operated bronchus may also complicate the resection of that bronchus.[93] A DLT in the nonoperated bronchus (right or left) will stent the airway, thereby decreasing the chance that a sagging mediastinum will obstruct ventilation. However, lung distal to the tube tip can still be compressed.[94]

The human airway is asymmetrical. The average length of the adult left bronchus is about 5.0 cm, whereas the right bronchus is less than 2.0 cm long. Many anesthesiologists prefer a left DLT for both right and left operations in order to reduce the chance of obstructing the upper-lobe bronchus.[95]

A right DLT must be used if there is an intrinsic (tumor, stenosis) or extrinsic (tumor, aortic aneurysm) obstruction of the left bronchus. A right DLT is also used for sleeve resections of the left bronchus and during left single-lung transplants. If a right bronchial intubation is planned, a right rubber Robertshaw's tube may be a better choice. Rubber DLTs have a right upper-lobe ventilation slot that is approximately twice as long as the slot on a right plastic tube. A rubber right DLT therefore has less chance of obstructing the right-upper lobe.[93] The rubber Robersthaw's DLT is also shorter and has a larger external circumference than a plastic tube, so it is unlikely that it will be advanced too far into the right bronchus.

Size selection

The largest DLT with a bronchial lumen that fits the desired bronchus should be used.[96] Many of the problems with DLTs result from using a tube that is too small. Small tubes are often advanced too far into the bronchus, where they are more likely to obstruct the upper-lobe bronchus.[97] Since the bronchial cuff of a smaller tube must be inflated with a larger volume of air to seal the bronchus, the risk of airway injury is increased. During one-lung ventilation, there is more resistance to airflow through the lumen of a smaller tube and this generates higher levels of "auto-PEEP" in patients with COPD.[98]

Direct measurement of bronchial width from a chest radiograph has been tried as a means of selecting DLT size,[96] but the bronchus is not always visible on the chest radiograph. Measurement of the bronchus on the chest computed tomography (CT) scan is accurate, but may be impractical since it requires the assistance of a radiologist.[99,100]

The width of the left bronchus is directly proportional to the size of the trachea.[101] Since the trachea is visible and easily measured from a chest radiograph, tracheal width can be used to predict the size of the left bronchus. This allows selection of the appropriate size left DLT[102] (Table 47.3). The airway of most men will accept a 39Fr or 41Fr DLT, and for most women a 37Fr or 39Fr tube is indicated.[103]

A smaller tube may be needed if there is an intrinsic or extrinsic obstruction of the mainstem bronchus to be intubated.[104] Following a lung transplant, patients may have a bronchus that is significantly smaller than predicted from measurements of the native trachea.[105] For adult patients with very small bronchi relative to tracheal size owing to airway pathology, a bronchial blocker may be indicated.[106]

Placement

An appropriate size left DLT is selected using tracheal width guidelines.[107] Following preoxygenation, general anesthesia is induced and the patient is paralyzed for airway intubation. A Macintosh laryngoscope blade provides the largest area in which to pass a DLT. The improved vision (IV-MAC) laryngoscope blade is particularly useful for DLTs.[108] Extra care must be taken with plastic DLTs since their cuffs are fragile

Table 47.3 *Guidelines for left BronchoCath double-lumen tubes (DLTs)*

Measured tracheal width (mm)	Predicted left bronchus width (mm)	Recommended DLT size	OD (mm) Main body	Left lumen
≥ 18	≥ 12.2	41Fr	14–15	10.6
≥ 16	≥ 10.9	39Fr	13–14	10.1
≥ 15	≥ 10.2	37Fr	13–14	10.0
≥ 14	≥ 9.5	35Fr	12–13	9.5
≥ 12.5	≥ 8.5	32Fr	10–11	8.3
≥ 11	≥ 7.5	28Fr	9.4	7.4

Tracheal width (mm) as measured from the chest radiograph.
Predicted size of left bronchus = Tracheal width (mm) × 0.68
DLT, left-sided BronchoCath (Mallinckrodt Medical, St Louis, MO, USA); OD, outside diameter (mm) for main body and for left lumen of the BronchoCath DLT. Size specifications provided by the manufacturer.

and easily torn by the patient's teeth. The tracheal cuff is usually the cuff that is damaged.[88,109]

The distal tip of the tube is advanced just past the vocal cords. The stylet in the bronchial lumen is then removed. It has been recommended that the stylet be kept in place during the entire placement sequence to increase the success rate of entering the left bronchus.[110] The safety of this maneuver has not been demonstrated.

The tube is rotated 90–120° counterclockwise (towards the left bronchus) and is only then advanced down the trachea. The recommended endpoint of advancing the tube until a moderate resistance is encountered will often result in a DLT that is too deep in the bronchus, especially when a small tube is used. In both men and women, depth of placement is directly proportional to height.[111] For a man or woman of height 170 cm, the DLT should be advanced into the airway 29 cm, and for each ± 10-cm change in height the tube should be advanced or withdrawn ± 1 cm.

Several variations have been described for "blindly" positioning a DLT. The bronchial cuff can be partially inflated while the tube is in the trachea. Then the tube is advanced. When the bronchial lumen enters a bronchus, a marked rise in the tension in the bronchial cuff's pilot balloon will be noted and breath sounds will be heard only over the intubated lung.[112–114] Another method is to intentionally advance the tube deep into the bronchus until resistance is encountered. The bronchial cuff is inflated and the tube is withdrawn until the tension in the bronchial cuff's pilot balloon suddenly drops as the cuff enters the trachea. The tube is then readvanced back into the bronchus.[115] Both these methods involve moving the DLT while its bronchial cuff is inflated. This has the potential for damaging the airway. The following sequence of steps for safe "blind" placement of a left DLT is recommended.[116]

The tube is advanced into the bronchus to a depth based on the patient's height and both cuffs are inflated. When an appropriate size (large) tube is used, only 1–2 mL of air should be needed for the bronchial cuff. The patient is then ventilated through both lumens. Moisture should appear in each, indicating that both lumens are open to the lungs. Both sides of the chest should move with ventilation and bilateral breath sounds should be present.

Initially, the tracheal lumen is clamped. If the tube is in the left bronchus, breath sounds should now be heard only over the intubated left lung. If breath sounds are present bilaterally, the tube is probably not deep enough and should be advanced still further into the bronchus.

If at this point breath sounds are heard only over the right lung, the tube is in the right bronchus. In this situation, both cuffs should be deflated and the tube withdrawn several centimeters until it is in the trachea. Turning the patient's head and neck to the right while bending the head down will help direct the tube into the left bronchus.[117] The tube is rotated to the left and readvanced. Once the tube is in the left bronchus, the left lumen is clamped and the patient is ventilated through the tracheal lumen. Breath sounds should now be heard over the right lung. If the tube is not in a satisfactory position, there will be difficulty ventilating the patient since the inflated tracheal and bronchial cuffs will be obstructing gas flow from the tracheal opening. In this situation, by deflating the bronchial cuff and continuing to ventilate through the tracheal lumen one can accurately determine tube position. If the tube is not deep enough breath sounds will now be present bilaterally. If the tube is too deep, breath sounds will now be present only over the left lung. Both cuffs should be deflated and the tube should be advanced or withdrawn in 0.5-cm increments until it is in a satisfactory position.

Confirmation of double-lumen tube placement

DLT position must always be reconfirmed before surgery since tubes are easily displaced while moving the patient to the lateral decubitus position.[118] The tension in the pilot balloon to the bronchial cuff should be noted after first inflating that cuff.[119] DLTs are often displaced proximally with lateral positioning.[120] Therefore, after turning the patient or at any time during the procedure if the pilot balloon becomes softer the bronchial cuff is no longer completely in the bronchus. The tube should be advanced further into the bronchus until the initial tension in the pilot balloon returns. Confirmation of tube position is accomplished by physical examination of the chest, including auscultation and observation of chest wall movement and measurement of peak inspiratory pressures during independent ventilation of each lung. Water vapor in both lumens usually indicates that both lumens are open to gas-exchanging areas.

Controversy exists as to whether a bronchoscope should[121,122] or should not[91,123,124] be used *routinely* for DLT placement. A bronchoscope is often used to visually confirm DLT position.[125] Looking down the tracheal lumen, there should be an unobstructed view of the blue bronchial cuff below the carina in the appropriate bronchus.[126] Several studies have reported that DLTs placed "blindly," i.e. without bronchoscope confirmation, are frequently not in an "ideal" position.[127–130] A tube is considered to be in an ideal position when the proximal edge of its bronchial cuff is immediately below the carina in the appropriate bronchus.[126] Most "malpositioned" tubes are actually only slightly deeper in the bronchus and will function without problems.[107] Since most tubes will be displaced proximally when turning the patient,[120] bronchoscopic visualization in the supine position is really only useful to determine whether the endobronchial lumen has been placed on the appropriate side.[131] A bronchial cuff 0.5–1.0 cm inside the left bronchus initially will avoid cuff displacement into the trachea when the patient is turned to the decubitus position.

The bronchoscope should also be passed down the bronchial lumen to insure patency of that lumen and to confirm that the upper-lobe orifice is not obstructed. A 4.9-mm-diameter bronchoscope will pass down larger DLTs, but a 3.6-mm-diameter bronchoscope is needed if smaller (35Fr, 37Fr) tubes are used.[132] Very thin pediatric bronchoscopes are needed for smaller (28Fr or 32Fr) tubes. Capnography,[133,134] continu-

ous monitoring by spirometry with flow–volume or pressure–volume loops,[135] and even chest radiographs and fluoroscopy have been used to determine the correct DLT position. If visualization of the glottis by direct laryngoscopy is not possible, a DLT can be advanced over a bronchoscope. Likewise, a bronchoscope in the bronchial lumen can be used as a stylet to advance the DLT into the bronchus if "blind" intubation of that bronchus fails.[136]

If continued ventilatory support is required at the completion of thoracotomy, the DLT may need to be replaced with a conventional endotracheal tube. This can be challenging if the initial intubation was difficult, or even hazardous if airway edema has developed during the procedure. A variety of stylets and tracheal tube exchangers can be used to more safely change from a DLT to an endotracheal tube.[137,138]

Complications

Although DLTs are generally safe and easy to use, complications do occur (Table 47.4). The most frequent problems relate to DLT position. A misplaced DLT can result in airway damage and hypoxemia or compromise any procedure if the operated lung fails to collapse.

DLT position can change at any time during surgery, so constant vigilance is important. Ventilation of the dependent lung, through either the tracheal or bronchial lumen, is never directly visualized. The patient must be monitored by observing changes in the peak inspiratory pressure to the dependent lung, end-tidal CO_2 levels, $S_{p}O_2$, and most importantly mediastinal movement during inspiration. If in doubt, a bronchoscope should be used to confirm tube position.

The airway can be traumatized during intubation and extubation. Most injuries are clinically insignificant – usually asymptomatic ecchymosis of the mucous membranes.[110] More serious problems (arytenoid dislocation, torn vocal cords) occur, but have become relatively uncommon since plastic DLTs replaced the more rigid rubber DLTs.

When the softer plastic DLTs were introduced in the early 1980s, it was thought that they were safer than rubber tubes.[86,139] However, airway rupture has been reported with both rubber[140–145] and plastic DLTs.[146–151] The factors that increase the risk of airway rupture are listed in Table 47.5. Airway damage can present with air leak, subcutaneous surgical emphysema, airway hemorrhage, and cardiovascular instability due to tension pneumothorax. With incomplete laceration, air may dissect into the adventitia, producing an aneurysmal dilation of the membranous wall. If N_2O is used, it will further distend this air collection. The signs of injury may not be evident for many hours after the initial injury, when rupture into the mediastinum or pleural space occurs.[152]

Both cuffs should be deflated when moving the patient or changing the position of the DLT. It is safer to keep the bronchial cuff deflated whenever lung isolation or selective ventilation is not required.

A fiberoptic bronchoscope should be available to inspect the airway if mediastinal emphysema or loss of tidal volume occurs at any time during the surgery. Bronchoscopy should be performed before extubation to identify evidence of trauma if an airway disruption is suspected as this requires immediate surgical intervention for a favorable prognosis.[153] At the completion of every thoracotomy, the integrity of the intubated bronchus should be tested with manual ventilation for leaks with only the tracheal cuff inflated. Table 47.6 lists recommendations to reduce the incidence of major airway injury from a DLT.

A DLT should never be forcibly advanced. One should be even more cautious in patients who have airway pathology such as tracheomalacia or tumor infiltration, and for patients who are immunosuppressed or who are receiving steroids.

Both the bronchial and tracheal cuffs must be inflated with air slowly. It is extremely important not to overinflate the bronchial cuff. A 3-mL syringe can be used for the bronchial cuff as a reminder that 2–3 mL of air is usually the volume needed if an appropriate size (large) tube has been selected.

Table 47.4 *Complications of double-lumen tubes (DLTs)*

Intubation
Carinal hook unable to pass by glottis (Carlens or White DLT)
Unable to advance bronchial limb into bronchus
Tube too large
Airway obstruction (intrinsic or extrinsic)
Trauma
Dental trauma
Airway injury
Laryngitis, mucosal ecchymosis, arytenoid dislocation, tracheobronchial rupture
Rupture thoracic aneurysm
Position
Not far enough into bronchus
Bronchial cuff in carina
Unable to ventilate nonintubated lung with both cuffs inflated or unable to deflate intubated lung
Failure to seal airway – contamination of healthy lung
Down wrong bronchus
Too deep in correct bronchus
Obstruction of upper lobe causing hypoxemia
Failure to collapse upper lobe
Changes during surgery
Surgical manipulation
Movement of patient to decubitus position
Head flexion or extension
Tube inadequately taped or secured
Hypoxemia
Malpositioned DLT
Collapse upper lobe of nonoperated lung
Obstruction by bronchial cuff in trachea
Torsion of tube or bronchial lumen opening against bronchial wall
Carinal hook bent back obstructing tracheal lumen
Miscellaneous
Bronchial lumen interferes with surgical procedures
Pneumonectomy, carinal or sleeve resection, lung transplant
Displacement of mediastinal mass
Bronchial lumen sutured to pulmonary vessel

Nitrous oxide should avoided.[154] When N_2O is used, both cuffs should be inflated with either saline or an O_2/N_2O mixture to prevent cuff distention during the procedure. The bronchial and tracheal cuffs should be deflated periodically to avoid build-up of excessive pressure on the mucosa.

Underinflation of the bronchial cuff can result in a cross-leak with failure to collapse the lung undergoing surgery and contamination of the dependent lung. Overinflation of the bronchial cuff can damage the airway or obstruct the trachea. Several techniques have been described to determine the exact endpoint for cuff inflation.[155,156]

OPTIMIZING OXYGENATION DURING ONE-LUNG VENTILATION

In the lateral decubitus position, approximately 40% of the cardiac output flows to the nondependent lung and 60% goes to the dependent lung. There is normally perfusion of nongas-exchanging areas in each lung, so during two-lung ventilation with the patient in the lateral position approximately 35% of the cardiac output participates in gas exchange in the nondependent lung.

Following selective lung collapse, the lung undergoing surgery continues to be perfused but is not ventilated. Several factors influence the magnitude of this "shunt." Blood flow to the dependent, ventilated lung

Table 47.5 *Risk factors associated with airway rupture*

Direct trauma
Forceful insertion
Frayed tube tip (rubber DLTs only)
Too large DLT – bronchial lumen too large for bronchus
Tube advanced with stylet in place (?)
Movement of tube with cuffs inflated
Carinal hook damages upper airway (Carlens's and White's DLTs)
Cuff overinflation
Too rapid inflation
Too large a volume
Too small DLT (requires larger volume to seal the bronchus)
Overdistention from nitrous oxide
Asymmetric cuff distention – pushes tip into airway wall (rubber DLTs)
Pre-existing airway pathology
Congenital airway wall abnormalities
Airway wall weakness from tumor infiltration or infection
Airway distortion from lymph nodes, intra- and extrabronchial tumors
Patients receiving steroids
Patients with leukemia and lymphoma
Hypotension with hypoperfusion to the airway

DLTs, double-lumen tubes.

Table 47.6 *Recommendations for DLT placement*

1	Select the largest plastic DLT that will safely fit the airway
2	Remove the bronchial stylet as soon as the tube is past the vocal cords
3	Be extra cautious with patients who have tracheobronchial wall pathology, leukemia, steroids, hypoperfusion
4	Advance the DLT into the bronchus to a depth based on the patient's height
5	Inflate both cuffs slowly – never overinflate either cuff
6	Use a 3-mL syringe to inflate bronchial cuff – usually < 3 mL air is adequate if an appropriate size (large) DLT is selected. If more air is needed, reassess tube position by auscultation or by bronchoscopy
7	If you use N_20, inflate both cuffs with saline or an O_2/N_2O mixture rather than air alone – measure cuff pressures intermittently and periodically relieve pressure (if necessary) by partially deflating the cuff to the original inflation volume. Keep bronchial cuff pressure < 30 cmH_2O
8	Deflate both cuffs before moving the patient, deflate the bronchial cuff when lung isolation or selective ventilation is not needed
9	During esophageal procedures, consider partial deflation of the cuff when surgical dissection is near either cuff
10	Test the integrity of the intubated bronchus at the completion of surgery, use fiberoptic bronchoscopy to determine site and extent of injury

DLT, double-lumen tube.

is increased by the effects of gravity and by the increased vascular resistance in the lung undergoing surgery from surgical retraction and the effects of total atelectasis. Other factors that influence the degree of arterial hypoxemia during one-lung ventilation include inadequate inspired oxygen concentration (F_IO_2) and alveolar hypoventilation. Additionally, the right lung has more gas-exchanging areas than the left, and there is a greater drop in P_aO_2 with one-lung ventilation during right-sided operations than during similar procedures on the left side.[157]

Systemic vasopressors (dopamine, epinephrine, phenylephrine) preferentially constrict vessels perfusing normoxic or hyperoxic lung segments, resulting in redistribution of blood flow to the collapsed lung. Vasodilators (nitroprusside, nitroglycerin) relax vascular smooth muscle in the collapsed lung by releasing NO. This increases blood flow to the operated lung, thereby lowering P_aO_2. Drugs that inhibit NO release maintain the HPV response.[158] Surgical manipulation of the lung releases vasoactive substances that blunt HPV.[38] During video-assisted thoracoscopic surgery there is less direct trauma to the lung, so HPV may play a more important role during this type of surgery than during open thoracotomy. Increased pulmonary vascular resistance may be present preoperatively in a chronically diseased lung, so blood flow may already be preferentially diverted to the healthy lung.

When changing from two-lung to one-lung ventilation, tidal volume should be continued unchanged. The ventilator rate should be adjusted to keep P_aCO_2 at 4.8–5.33 kPa (36–40 mmHg). If end-tidal CO_2 measurements are used, it must be realized that, in the lateral decubitus position, these values during one-lung ventilation may be as much as 1.33 kPa (10 mmHg) lower than actual P_aCO_2. Hypocarbia should be avoided because it will increase pulmonary vascular resistance in the ventilated lung and redirect blood flow to the nonventilated lung. Hypercarbia is usually not a problem if minute ventilation is maintained.

If hypoxemia occurs, the position of the DLT or bronchial blocker should be immediately reconfirmed. Other mechanical problems (tube obstruction, bronchospasm) should be considered. Clinically significant hypoxemia usually does not occur immediately after initiation of one-lung ventilation, but after 10–15 min because it takes that long for the nonventilated lung to completely collapse and for any remaining O_2 in that lung to be absorbed.

The largest proportion of the cardiac output is directed to the ventilated lung, therefore matching ventilation to perfusion is important. In the lateral decubitus position, the intra-abdominal contents shift the diaphragm cephalad, thus reducing the FRC of the dependent lung. General anesthesia further decreases FRC. Therefore, during lateral thoracotomy, the dependent lung may have areas of low ventilation to perfusion ratios and areas that are completely atelectactic. To maximize P_aO_2 the dependent lung should be ventilated with 100% O_2 with a large tidal volume.[159]

Relatively large tidal volumes (10–14 mL kg^{-1}) are needed to recruit dependent lung alveoli. Tidal volumes of less than 8 mL kg^{-1} result in a further decrease in FRC, which leads to increased areas of dependent lung atelectasis. Larger tidal volumes (> 15 mL kg^{-1}) overdistend the alveoli and increase pulmonary vascular resistance, thereby causing redistribution of blood flow to the nondependent lung.

Even with a shunt of 25% to the nonventilated lung, an F_IO_2 of 1.0 and large tidal volume ventilation will usually produce a $P_aO_2 > 20$ kPa (150 mmHg).[160] At this oxygen tension, arterial hemoglobin is 100% saturated. A high F_IO_2 causes vasodilation of the vessels in the dependent lung, increasing perfusion of that lung and further decreasing shunt.

A theoretical concern is that an F_IO_2 of 1.0 can lead to absorption atelectasis of the dependent lung. Although the addition of 10–20% nitrogen to the inspired gas mixture will decrease the propensity for the dependent lung to collapse, clinically this maneuver is unnecessary. Concerns about pulmonary damage from hyperoxia are only relevant for patients with histories of certain drugs (bleomycin, nitrofurantoin, amiodarone, mitomycin C) or acute inflammatory lung diseases. These have been associated with postoperative pulmonary oxygen toxicity.[161–163]

During one-lung ventilation, peak inspiratory pressure will be high because the dependent lung will have decreased compliance while being inflated with a relatively large tidal volume.[164] In addition, airway resistance will be increased through the single lumen of the DLT. Since airway pressure changes may be great, continuous monitoring of ventilatory mechanics during one-lung ventilation has been recommended.[165] Special ventilators employing instantaneous breath-to-breath analysis to adjust ventilatory patterns (adaptive lung ventilation) have been used during one-lung ventilation.[166]

The traditional practice of using large tidal ventilation during one-lung ventilation is now questioned. Barotrauma, presumably from high

peak pressures during one-lung ventilation, has been associated with pulmonary complications following thoracotomy. Pressure-controlled ventilation has been suggested as an alternative to volume-controlled ventilation during one-lung ventilation. Pressure-controlled ventilation is associated with lower peak airway pressures, lower shunt, and higher oxygen levels than conventional volume-controlled ventilation.[167]

If hypoxemia occurs during one-lung ventilation, PEEP can be applied to the ventilated lung. Many factors cause a reduction in FRC during one-lung ventilation, including the effects of general anesthesia, the pressure of the abdominal contents on the diaphragm, the weight of the sagging mediastinum, and incorrect positioning of the patient on the operating room table. In the presence of decreased FRC, PEEP (10 cmH_2O) will recruit collapsed and underinflated alveoli and improve oxygenation. However, with normal or increased FRC, PEEP will decrease cardiac output, increase alveolar airway pressure, and increase dependent lung pulmonary vascular resistance, which in turn will divert blood flow to the nonventilated lung, thereby increasing hypoxemia.[168]

During one-lung ventilation, if alveolar pressure fails to fall to atmospheric pressure at the end of expiration, "intrinsic" or auto-PEEP will be generated.[169,170] The degree of auto-PEEP that develops is determined by several factors, including the extent of preoperative pulmonary disease, the time allowed for expiration, and airflow resistance through the single lumen of the DLT. Auto-PEEP also increases with age, perhaps because of loss of lung elastic recoil.

Although some patients do benefit from the addition of external PEEP during one-lung ventilation, at present it is not possible to predict who those patients are.[171] External PEEP may be therapeutic during one-lung ventilation in patients who do not generate significant levels of auto-PEEP.[172]

The collapsed lung can be partially re-expanded to treat severe hypoxemia. A single breath to the operated lung will temporarily correct the ventilation–perfusion imbalance.[173] The lung will slowly recollapse, so it must be re-expanded about every 5 min. This maneuver cannot be used during video-assisted thoracoscopic surgery as it will interfere with operative exposure.

Insufflation with 100% O_2 to the nonventilated lung is inadequate treatment for hypoxemia because the O_2 will fail to reach and recruit collapsed alveoli. However, insufflation by CPAP with 100% O_2 to the nonventilated lung is a very effective means of correcting hypoxemia.[174–176]

CPAP maintains the patency of the operated alveoli with O_2, so unsaturated mixed venous blood perfusing that lung becomes oxygenated. Any increased airway pressure from the CPAP may further increase pulmonary vascular resistance, which will divert blood flow to the dependent, ventilated lung. The nonventilated lung remains partially distended but "quiet" with CPAP.

Initially, relatively high pressures are required when applying CPAP to an atelectatic lung in order to open the collapsed alveoli. CPAP should be applied during the deflation phase of a full breath to the nondependent lung.[177] Once distended, relatively small levels of CPAP (5–10 cmH_2O) are all that is required to maintain satisfactory oxygenation. Higher levels of CPAP overdistend the lung and interfere with surgical exposure.

CPAP is effective only when there are no major disruptions in the airway because a nonintact bronchus will not allow distending pressures to be maintained. CPAP is therefore not helpful with a bronchopleural fistula, during sleeve resection of the airway, or during massive pulmonary hemorrhage or bronchopleural lavage. Any airway obstruction by mucus, blood, or tumor may not permit adequate airway pressures to reach the lung. CPAP is not normally used for treating hypoxemia during video-assisted thoracoscopic surgery since any distention of the lung undergoing surgery will compromise the surgical exposure.

The combination of PEEP (5–10 cmH_2O) applied to the ventilated lung and CPAP (5–10 cmH_2O) to the nonventilated lung has been used to treat severe hypoxemia, but is seldom necessary when upper-lung CPAP is used alone.[178]

In animal models, inflation of a nonocclusive pulmonary artery catheter balloon located in the main pulmonary artery of the lung undergoing surgery can redistribute blood flow to the lung not undergoing surgery. However, if the pulmonary artery catheter were to shift to the dependent lung, inflation of the balloon could potentially increase hypoxemia by redirecting blood to the upper lung.

During a pneumonectomy, ligation of the pulmonary artery completely eliminates shunt, thus maximizing the relation between ventilation and perfusion. Clamping the lobar pulmonary artery during lobectomy should increase vascular resistance in that lung segment, directing more blood to the ventilated lung. Occlusion of the main pulmonary artery during limited pulmonary resection is not advised as this maneuver will alter the activity of the alveolar lining material (surfactant) and will deleteriously affect lung re-expansion following surgery, i.e. it will potentially induce an ischemia–reperfusion injury.

Pharmacologic manipulation of the pulmonary blood flow is another approach to matching perfusion with ventilation. It has been suggested that, since some endogenous prostaglandins inhibit HPV, prostaglandin inhibitors might potentiate the HPV response and improve oxygenation during one-lung ventilation. In an *in vitro* study, ibuprofen, a cyclo-oxygenase inhibitor, did reverse the depression of HPV by halothane.[179]

Direct infusion of prostaglandin F_{2a}, a potent pulmonary vasoconstrictor, into the pulmonary artery of the nonventilated lung during one-lung ventilation results in a significant decrease in shunt and an increase in $P_{a}O_2$.[180] The continuous infusion of a vasodilator, prostaglandin E_1, into the pulmonary artery of the ventilated lung improved both arterial oxygenation and venous admixture during one-lung ventilation.[181,182]

Nitric oxide has an important physiologic role in the modulation of vascular tone. The vasodilator properties of inhaled NO (NO_i) are restricted to the pulmonary circulation.[183] NO_i (5–80 p.p.m.) decreases pulmonary vascular resistance in animal models.[184] NO_i also decreased pulmonary vascular resistance during one-lung ventilation in supine patients.[185] However, during one-lung ventilation in the lateral position, NO_i (40 p.p.m.) did not decrease mean pulmonary artery pressure in patients with normal pulmonary vascular resistance and the shunt remained unchanged.[186]

The use of NO_i to improve oxygenation during one-lung ventilation has been disappointing. This may be because the effects of NO_i are directly proportional to the degree of pulmonary vascular resistance present in the ventilated lung before NO administration and most patients undergoing pulmonary resection have normal or only slightly elevated pulmonary artery pressures.

During one-lung ventilation, the combination of NO_i to the ventilated lung and a potent pulmonary vasoconstrictor (almitrine) decreased perfusion to lung undergoing surgery and markedly improved oxygenation.[187] NO_i and other pulmonary vasoconstrictors may prove helpful for hypoxemia during video-assisted thoracoscopic surgery when application of CPAP is not practical.[188]

VENTILATION AT THE COMPLETION OF THORACOTOMY

Prior to reinflating the collapsed lung, both lumens of the DLT should be suctioned to remove any mucus, blood, or debris from each lung. Both lungs must be fully re-expanded and the mediastinum must be midline at the completion of one-lung ventilation. Following total collapse, the operated lung will re-expand unevenly during inflation. Lung volume increases as collapsed alveoli are recruited. Alterations in pulmonary surfactant occur during one-lung ventilation and this necessitates the application of high-sustained pressures in order to reopen the atelectac-

tic lung. Once the lung has been fully reinflated and deflates to residual volume, subsequent inflation will require lower pressures consistent with normal surfactant activity.

Following any pulmonary resection, the integrity of the bronchial repair or the bronchial stump must be tested before the chest is closed. The surgeon pours warm saline into the pleural cavity while the anesthesiologist applies increasing levels of inflation pressures (up to 30–40 cmH_2O) by manually compressing the reservoir bag. This maneuver demonstrates air leaks and, when repeated several times, allows the previously collapsed lung to fully re-expand. Although a lung that has been collapsed for several days can develop unilateral ("re-expansion") pulmonary edema if inflated too rapidly, this is extremely uncommon following one-lung ventilation.

Immediately after most thoracic procedures (including pneumonectomy), patients should be extubated and allowed to breathe spontaneously. After any pulmonary resection, positive pressure ventilation places stress on the bronchial suture line and exacerbates air leakage and formation of a bronchopleural fistula.

POST-THORACOTOMY PAIN MANAGEMENT

The decrease in FRC and the ability to cough and breathe deeply following thoracotomy are influenced by pain. The patient who experiences pain may be unable or unwilling to clear secretions. This in turn can lead to atelectasis and eventually pneumonia. Besides improved pulmonary function, comfortable patients have a lower incidence of cardiac complications after thoracic procedures.[189]

Analgesic regimens associated with the best post-thoracotomy pulmonary function are generally felt to be superior. In addition to spirometry and arterial blood gas values, post-thoracotomy pain is also assessed by visual analog pain scores (at rest and with cough and movement), the amount of additional opioid requested, the length of hospital stay, and overall patient satisfaction.

The lateral thoracotomy incision is very painful.[190] The site and extent of the incision influences the degree of pain. Disruption of intercostal nerves and inflammation of the chest wall and pleura all contribute to the discomfort. The number and the sites of the chest drainage tubes also influence pain. Some patients also experience shoulder pain of unknown etiology following thoracotomy.[191]

Systemic opioids

Systemic opioids have a very narrow therapeutic window. With inadequate pain relief, the patient will be unwilling to cough and clear secretions while the overmedicated patient will be oversedated and unable to cough and breathe deeply.

Intermittent opioid administration may be associated with a significant time lag between the request for medication, the actual administration of the medication, and the onset of action. When systemic opioids are used, patient-controlled analgesia (PCA) or continuous intravenous infusion are preferred.[192,193]

Neuraxial opioids

Neuraxial (epidural, intrathecal) analgesia can reduce pain and improve pulmonary function following thoracotomy.[194]

The current practice is to combine epidural local anesthetics with epidural opioids.[195] However, the addition of a local anesthetic may not significantly improve the analgesia achieved by epidural opioids alone.[196,197] The addition of bupivacaine (0.1%) does not affect epidural meperidine (pethidine) requirements, but does reduce pruritus after thoracotomy.[198] Hypotension or motor blockade from the local anesthetic may limit the patient's ability to ambulate. The addition of epinephrine, even in concentrations as low as 1:300 000, does decrease the amount of opioid needed for effective analgesia.[199]

Post-thoracotomy opioid neuraxial analgesia, when compared with parenterally administered opioids, improves pulmonary function and oxygenation and reduces postoperative complications.[200–203] This has been demonstrated with every opioid that has been given either intrathecally or epidurally following thoracic surgery.[204–206]

Unlike local anesthetics, neuraxial opioids are "selective." Other than potentiation of analgesia, they have no known hemodynamic or motor effects. Opioids work by binding to opiate receptors in the spinal cord. There is some evidence that pre-emptive administration of epidural opioids before surgical incision can reduce pain following thoracotomy.[207] Single-shot intrathecal morphine produces analgesia for up to 24 h, but the intrathecal route is limited by the need for repeated injections unless a catheter is placed.[208]

The epidural route is generally preferred since the incidence of respiratory depression is less than with intrathecal opioids and the presence of a catheter allows for continuous, prolonged drug administration in the postoperative period. Patient-controlled epidural opioid analgesia is also possible if a catheter is present.[209] Depending on the agent selected, a catheter placed at either the lumbar or the thoracic epidural level is effective for post-thoracotomy analgesia.[210]

Lipophilic agents rapidly diffuse across the dura into the cerebral spinal fluid, bind to the spinal opiate receptors, and produce a rapid onset of action. The more lipid soluble the agent, the more potent it is. Lipophilic agents such as fentanyl and sufentanil are better suited for epidural administration at the thoracic level.[211] These drugs provide satisfactory analgesia following thoracotomy, but because of their relatively short duration of action they should be administered by constant infusion.

Lipophilic opioids are rapidly absorbed into the systemic and cerebral circulation, and severe acute postoperative respiratory depression may occur with even small doses of epidural sufentanil.[212] Hydrophilic agents (morphine, hydromorphone) diffuse more slowly into the cerebral spinal fluid.[213] Their onset of action is delayed, but the length of action is longer than with the lipophilic agents. These agents are preferred when a continuous infusion is not possible.

Hydrophilic opioids can also be safely administered by constant infusion. Their low lipid solubility allows these drugs to be given either at the thoracic or at the lumbar level for post-thoracotomy pain relief without any difference in analgesic efficacy.

The complications of epidural opioids administered during surgery occur in the postoperative period. All epidural opioids decrease the ventilatory response to CO_2, but significant respiratory depression and apnea are rare. The lipophilic drugs cause *acute* respiratory depression owing to central effects from systemic absorption. The hydrophilic opioids are associated with *delayed* respiratory depression occurring hours after administration, presumably as a result of the rostral spread of the drug in the cerebrospinal fluid to the brain.

The factors predisposing to delayed respiratory depression include: concomitant administration of opioids or sedatives by other routes prior to or during surgery; high doses; the hydrophilicity of the agent; advanced age; and the site of administration (intrathecal > thoracic epidural > lumbar epidural). With appropriate dosing and selection of patients, serious respiratory depression is uncommon. The frequency and occurrence of minor complications (pruritus, urinary retention, nausea and vomiting) varies with the specific opioid used and should not be a contraindication for their use following thoracotomy.

Adjuncts

Cryoanalgesia, the freezing of intercostal nerves, can reduce incisional pain following thoracotomy. The nerve at the incision space and the two intercostal nerves above and below are frozen. The loss of sensory and motor function that follows cryoanalgesia usually lasts 1–6 months. However, many patients experience painful neuralgias at the treatment sites.[214] Cryoanalgesia may reduce, but does not eliminate, post-thoracotomy pain and is useful only as a supplement to other forms of pain control.[215]

Electrodes can be placed on either side of the thoracic incision and electrical stimulation applied (transcutaneous electric nerve stimulation; TENS). TENS does not reduce opioid requirements or improve pulmonary function following thoracotomy, but is effective in treating pain after video-assisted thoracoscopic surgery.[216]

Intercostal nerve blocks can be performed directly intraoperatively with the lung collapsed or percutaneously following surgery. These blocks reduce opioid requirements following thoracotomy. Continuous blocks are more practical than repeated individual blocks owing to the relatively short action of the local anesthetics.[217,218] Continuous intercostal blockade can be achieved by placing several catheters in the intercostal grooves during surgery. Systemic toxicity from intravascular absorption or from direct injection and unrecognized dural puncture resulting in marked hypotension are major complications of intercostal nerve blocks.

Interpleural blockade can be performed by injecting local anesthetics into the thoracic cavity between the visceral and parietal pleura.[219] One approach is to place an epidural catheter directly through the chest wall. Local anesthetics can also be given directly into the chest drainage tube after the lung has been re-expanded. Because a significant loss of local anesthetic can occur with an open chest drainage tube, clamping the chest tube following anesthetic administration increases the success rate.[220] Pain relief following thoracotomy is incomplete when the interpleural technique is used alone.[221,222] Local anesthetic on the diaphragm may actually impair respiratory function.[223] Interpleural analgesia is useful for analgesia following video-assisted thoracoscopic surgery.[224]

Ipsilateral stellate ganglion block is also effective for treating shoulder pain after thoracotomy.[225] Nonsteroidal anti-inflammatory drugs (NSAIDs) are also effective for shoulder pain resistant to epidural analgesia. When used in combination with other analgesics, NSAIDs improve pain relief and postoperative pulmonary function following thoracotomy.[226,227] Ketorolac supplementation of hydromorphone epidural analgesia reduced total opioid requirement and was associated with better postoperative pulmonary function than the combination of epidural hydromorphone and bupivacaine.[228]

NSAIDs have a peripheral site of action, have anti-inflammatory and antipyretic activity, and can be conveniently administered intravenously or by rectal suppository. One advantage after thoracotomy is that they are not respiratory depressants. Side-effects, including increased bleeding time, gastrointestinal and central nervous system symptoms, and a deterioration of renal function, may limit their use in some at-risk patients.

Low-dose intramuscular or intravenous ketamine is another useful adjunct for post-thoracotomy analgesia.[229,230] Intravenous tramadol, an analgesic with mixed μ-opioid and nonopioid activity, is also effective in the treatment of post-thoracotomy pain.[231]

POST-THORACOTOMY COMPLICATIONS

Atelectasis

The most common pulmonary complication following thoracotomy is atelectasis. Significant atelectasis will cause a mismatch of ventilation with perfusion and result in hypoxemia.

Atelectasis may develop from pulmonary injury during surgery, incomplete lung re-expansion following one-lung ventilation, or from bronchial obstruction by mucus as a result of inadequate clearance of secretions. Deep breathing, coughing, pulmonary toilet with suctioning, and clearance of secretions with incentive spirometry, aerosolized bronchodilators, and early postoperative ambulation are important. Effective pain relief is essential.

For lobar or whole lung atelectasis due to mucus plugging, the patient should be placed in the lateral position with the healthy lung dependent. This position improves ventilation/perfusion and promotes mucus drainage from the operated lung. If oxygenation does not improve with lateral positioning and chest physiotherapy, fiberoptic or rigid bronchoscopy and lavage may be required.

Unresolved atelectasis can proceed to pneumonia, a potentially fatal condition following pneumonectomy and in patients with borderline pulmonary reserve. Associated respiratory failure may require reintubation of the airway and mechanical ventilation.[232] This increases the risk of stump disruption and formation of a bronchopleural fistula. Empyema (pus in the pleural space) may result from pneumonia.

Airway trauma

Injury to the airway can occur at any time during surgery and may go unrecognized intraoperatively. A damaged airway can present postoperatively with respiratory distress, an air leak, subcutaneous emphysema, hemorrhage, or cardiovascular instability owing to tension pneumothorax.

Immediate surgical intervention is essential. Any positive pressure ventilation, even mask-assisted ventilation, will cause gas to enter either subcutaneous tissue or the chest, further exacerbating the situation. An awake bronchoscope-guided intubation of the airway is recommended, followed by spontaneous ventilation with an inhalational anesthetic agent. For a tracheal injury, the endotracheal tube should be carefully advanced past the defect. For a bronchial injury, the tube should be placed in the intact bronchus and the injured lung isolated.

Pneumothorax

In the presence of a bronchopleural communication, the volume of air in the pleural space will increase. A tension pneumothorax will develop if air continues to enter the chest and is not effectively decompressed. The risk of pneumothorax following pulmonary resection is reduced by placement of a pleural drainage tube. However, a pneumothorax can still develop if the chest tube or suction apparatus malfunctions, or if the tube is occluded. The tube must have access to the air leak. Positive pressure ventilation, especially with PEEP, increases the risk of developing a pneumothorax.

If the pneumothorax continues to enlarge it will displace mediastinal structures and compress the great vessels, leading to cardiovascular collapse. Signs of a tension pneumothorax include decreased chest movement and diminished breath sounds, a unilateral wheeze, and hyper-resonance to percussion on the affected side. The peak inspiratory pressure during mechanical ventilation will increase and may reach very high levels. A chest radiograph is diagnostic. The patient may

experience increasing respiratory distress, tachypnea, tachycardia, and hypotension. A decrease in S_pO_2 may occur before other signs become obvious. Immediate decompression with a large intravenous catheter or new chest tube may be lifesaving.

Cardiac herniation

Cardiac herniation can occur after pneumonectomy if the adjacent pericardium has been disrupted.[233] If the pericardial defect is repaired intraoperatively, the risk of herniation is low. Herniation is more common following right pneumonectomy. After left pneumonectomy, the great vessels and mediastinal structures provide more of a barrier to herniation. Factors that contribute to cardiac herniation include suction applied to the side of pneumonectomy, positioning the patient with the operated side dependent, positive pressure ventilation, coughing, and vomiting.

The signs of cardiac herniation occur following surgery and may include radiographic abnormalities, atrial and ventricular dysrhythmias, sudden hypotension, and superior vena caval syndrome.[234] Cardiovascular collapse occurs as a result of acute angulation of the heart and great vessels.

Hemodynamic stability can only return following surgical replacement of the heart to its normal position. If not immediately diagnosed and promptly treated, cardiac herniation is almost uniformly fatal. Even with aggressive treatment the mortality rate is 50%.[235] Until the chest is reopened, the patient should be positioned on the side with the remaining lung dependent.

Hemorrhage

The clinical signs of major hemorrhage (tachycardia, hypotension, oliguria) are usually obvious in hypovolemic patients. Excessive chest tube drainage and a falling hematocrit are indications for surgical re-exploration.

Structural injuries

Structural injuries following thoracotomy occur from intraoperative surgical trauma or from malpositioning. The phrenic nerve is often injured, especially during extensive mediastinal dissection or if the nerve is embedded in tumor. The patient may experience some distress during spontaneous breathing. For the patient with limited pulmonary reserve, this complication can be devastating. The chest radiograph will reveal an elevated hemidiaphragm.

The left recurrent laryngeal nerve can be injured during hilar dissection. A unilateral injury usually results in hoarseness, but is otherwise asymptomatic.

Damage to the thoracic duct can occur from surgery or from central venous cannulation on the left side. Surgical intervention may be necessary to stop the resulting chylothorax.[236]

Dysrhythmias

Supraventricular dysrhythmias (atrial tachycardia, atrial flutter, and atrial fibrillation) occur in as many as 20% of patients following pneumonectomy.[237] Arrhythmias can occur after any thoracotomy or video-assisted thoracoscopic procedure.[238] Advanced age and pre-existing cardiac disease are important risk factors.

Any resulting impaired cardiac output may require fluid loading to enhance preload, which in turn can adversely affect pulmonary function. Prophylactic digitalization has long been recommended, especially following pneumonectomy, but recent studies have reported either no difference[239] or a higher incidence[189] of arrhythmias in thoracic surgical patients receiving digoxin. Prophylactic metoprolol can decrease the incidence of atrial fibrillation after pulmonary resection.[240]

Arrhythmias associated with profound hypotension require immediate cardioversion. Verapamil has been reported to be effective for atrial flutter or multifocal atrial tachycardia after lung resection.[241] Verapamil can cause hypotension from vasodilatory and negative inotropic effects and significant bradycardia occurs in as many as 25% of patients.[242] The administration of phenylephrine will support cardiac output. Beta-blockade may be necessary for rapid supraventricular dysrhythmias associated with hypertension and myocardial ischemia, but beta-blockers may cause bronchospasm. Esmolol is effective but short acting, so a continuous intravenous infusion may be needed.

Postpneumonectomy pulmonary edema

Pulmonary edema after pneumonectomy, an often fatal complication, is relatively common. Postpneumonectomy pulmonary edema occurs in as many as 5% of patients.[243] Some studies have associated positive fluid balance,[244,245] while others find no clear-cut relation between intraoperative fluid load and the development of pulmonary edema.[246] Postpneumonectomy pulmonary edema may be the result of increased pulmonary endothelial permeability after pneumonectomy[247] or impaired lymphatic drainage following radical lymph node dissection.

Cardiac output and right-heart filling pressures should be monitored following pneumonectomy if there is evidence of tissue hypoperfusion, especially when epidural-administered local anesthesia has caused vasodilation and hypotension. In most situations, small amounts of a vasopressor (ephedrine or neosynephrine) rather than intravenous fluid should be used to improve cardiac performance.

Right-heart failure

Extensive lung resection decreases pulmonary vascular cross-sectional area, resulting in increased pulmonary vascular resistance, which may lead to acute right-heart failure with or without pulmonary edema. Prior to pneumonectomy, if temporary unilateral occlusion of the main pulmonary artery to the operated lung by a PA catheter increases mean PA pressure more than 4–5.33 kPa (30–40 mmHg), the risk for postoperative right-heart failure is high. Even when predicted right-heart function is adequate, postoperative hypoxemia can precipitate right-heart failure.

Clinical signs of right-heart failure include supraventricular dysrhythmias, distended neck veins, hepatomegaly, and peripheral edema. The electrocardiogram may demonstrate left and right ventricular strain, and a chest radiograph may reveal right atrial and ventricular enlargement. Echocardiography will confirm the diagnosis.

The therapeutic goal is to support right ventricular preload and decrease pulmonary vascular resistance without lowering systemic blood pressure. Ventilatory support may be needed to correct reversible causes of increased pulmonary vascular resistance (hypoxemia, hypercarbia, and respiratory acidosis). Volume loading supports right ventricular preload. Pharmacologic treatment includes dobutamine, an inotrope whose vasodilatory action decreases pulmonary vascular resistance. Amrinone, which has less chronotropic effect than dobutamine, is an alternate choice, especially in the presence of myocardial ischemia, but systemic hypotension may limit its usefulness.

For pulmonary vasodilation and right ventricular afterload reduction, nitroglycerin is superior to nitroprusside. Both agents cause systemic hypotension and blunt HPV. The potential use of inhaled NO during and following pulmonary resection has been investigated.[248]

SPECIAL PROCEDURES

Fiberoptic bronchoscopy

Fiberoptic bronchoscopy can be performed under local or general anesthesia. If local anesthesia is chosen, sedatives should be used with caution since they may not be well tolerated by patients with pulmonary disease, particularly those that retain CO_2.

An anticholinergic agent is given to dry the airway and to blunt irritative reflexes. Glycopyrrolate is preferred to atropine or scopolamine. The upper airway is first anesthetized with local anesthetic solution delivered by nebulizer or with viscous lidocaine (lignocaine) gargle. Tracheal anesthesia is achieved by transtracheal injection of local anesthetic.[249] Superior laryngeal and glossopharyngeal nerve blocks can also be performed. These blocks depress airway reflexes so the patient should be told to take nothing by mouth for several hours following bronchoscopy.

During awake bronchoscopy, the patient should receive supplemental O_2. This is achieved using mouth-held nasal prongs or with a special face mask (Patil-Syracuse mask) which has a diaphragm through which the bronchoscope is passed.

For fiberoptic bronchoscopy, any general anesthetic (inhalational or intravenous) technique is acceptable, but N_2O is usually avoided to allow a higher F_IO_2.[250] A bronchoscope passed through an endotracheal tube produces a significant PEEP effect. Barotrauma can result if ventilation is attempted through a small tube obstructed by the bronchoscope. An endotracheal tube with an internal diameter of 8.0 mm or larger should be used. Suctioning through the bronchoscope decreases alveolar O_2, reduces FRC, and can cause significant hypoxemia.

The laryngeal mask airway (LMA) may be used instead of an endotracheal tube for therapeutic or diagnostic bronchoscopy under local or general anesthesia.[251] There is a larger internal diameter for easy passage of the bronchoscope. There may be less discomfort and trauma with an LMA, and capnographic and volume monitoring of respiration is also possible during the procedure. The endoscopist will also have access to the vocal cords and upper trachea

Rigid bronchoscopy

During rigid bronchoscopy, the patient should be paralyzed and ventilation controlled in order to minimize the risk of trauma from movement. Patients with suspected malignancy may have an increased sensitivity to nondepolarizing muscle relaxants (myasthenic or Eaton–Lambert syndrome), so short-acting relaxants should be given in small incremental doses or a continuous intravenous infusion of succinylcholine (suxamethonium) used and effects should be titrated using a blockade monitor.

A variety of ventilatory techniques can be used during rigid bronchoscopy.[252] Following hyperventilation with 100% O_2 to denitrogenate the lungs and to lower P_aCO_2, O_2 can be delivered by insufflation at a high flow (10–15 L min^{-1}) ("apneic oxygenation") without actually ventilating the patient. Although satisfactory oxygenation can be achieved for long periods, apnea should not extend beyond 5 min because of CO_2 accumulation.

Oxygen and anesthetic gases can also be delivered through the side arm of the bronchoscope by intermittent ventilation. Ventilation is possible as long as the eyepiece is in place, but must be interrupted whenever biopsy or suctioning is performed. An intravenous anesthetic technique with a short-acting agent such as propofol, alfentanil, or remifentanil allows continuous administration of anesthetic, which may not be possible with an inhalational anesthetic technique.[253]

During long procedures, CO_2 accumulates, thereby predisposing the patient to dysrhythmias, particularly in the presence of light anesthesia. Intermittent hyperventilation lowers P_aCO_2 and deepens the anesthetic. High fresh gas flows are needed to compensate for the leak around the bronchoscope. Oxygen can also be delivered by a Sanders system, which uses the Venturi principle to deliver O_2 by jet ventilation. The presence of an eyepiece is not necessary with this technique.

The complications of bronchoscopy include damage to the teeth, hemorrhage, broncho- and laryngospasm, subglottic edema, and airway perforation. A chest radiograph should be obtained following bronchoscopy to exclude the presence of pneumothorax or mediastinal emphysema from barotrauma.

Mediastinoscopy

Transcervical mediastinoscopy is usually performed on the right side of the chest with the patient in the reverse Trendelenburg position. For left mediastinal pathology, an open Chamberlain procedure is performed. The mediastinoscope is advanced following the anterior aspect of the trachea, passing close to the innominate vessels and the aortic arch.

Mediastinoscopy can be performed under local anesthesia. However, with the patient breathing spontaneously, the risk of venous air embolism and mediastinal injury from movement are greatly increased. General anesthesia, muscle paralysis, and controlled positive pressure ventilation is preferred. An anticholinergic agent should be given to blunt the autonomic reflexes (bradycardia) that result from compression or stretching of the trachea, vagus nerve, or great vessels.

The mediastinoscope may compress the innominate artery, causing a loss of the right radial pulse in the presence of a normal ECG. This may be misinterpreted as "hypotension" and lead to inappropriate aggressive treatment. During mediastinoscopy, blood pressure measurements should be obtained from the left arm, and the right radial pulse should be continuously monitored by plethysmography or by pulse oximetry. A decrease in the right radial pulse with normal left arm blood pressure indicates that the mediastinoscope needs to be repositioned. This is especially important if the patient has a history of impaired cerebral vascular circulation since carotid artery perfusion can be compromised by vascular compression by the mediastinoscope.

The major complications of mediastinoscopy include pneumothorax, recurrent laryngeal nerve damage, air embolism, and hemorrhage. If a large mediastinal blood vessel is torn, fluids given through an intravenous line in the arm may enter the mediastinum through the tear in the vein. In this situation, a large-bore catheter should be placed in a lower extremity vein. Although blood loss is usually quite small, blood should always be available since an emergency thoracotomy or sternotomy may be required to control bleeding. Other complications of mediastinoscopy include acute tracheal collapse, tension pneumomediastinum, hemothorax, and chylothorax. All patients must have a chest radiograph in the immediate postoperative period.

Mediastinal mass

Anterior mediastinal masses may compress the superior vena cava, the major airways, and even the heart. Complete or partial airway obstruction from an anterior mediastinal mass can occur following patient positioning and/or with relaxation of the muscles maintaining airway patency.[254] Preoperatively, it is important to ask whether the patient has experienced dyspnea in the supine position. A chest CT scan and other radiologic studies should be obtained to determine the extent of the tumor mass and any involvement with surrounding structures. Flow–volume loops are needed in both the upright and supine positions. A marked decrease in FEV_1 and peak expiratory flow rate in the supine position suggests the potential for airway obstruction with

anesthesia. In this situation, it is appropriate to consider radiotherapy to the mass prior to surgery.

A rigid bronchoscope may be needed to bypass an obstruction occurring in the trachea during induction of anesthesia and to allow the patient to be ventilated. The patient's position may need to be changed from supine to lateral or even prone to relieve the compression.

If biopsy of the mediastinal mass cannot be performed under local anesthesia, an awake fiberoptic bronchoscopy-assisted airway intubation followed by induction of general anesthesia is usually recommended. Spontaneous ventilation helps to maintain airway patency. The Trendelenburg position and positive pressure ventilation should be avoided if superior vena caval obstruction is present.

Bronchial obstruction

Secretions, blood, anatomic distortion, tumor, or foreign bodies can each cause bronchial obstruction. Retention of secretions can occur distal to the obstruction. A preoperative chest radiograph or CT scan may reveal consolidation or an abscess cavity beyond the airway obstruction.

Isolation of the lungs is critically important since pus can flood the airway as the obstruction is relieved. Manipulation of the lung or simply turning the patient to the lateral decubitus position can dislodge a foreign body or secretions into the trachea or dependent lung. Therefore, the lungs should be isolated before turning the patient. A DLT is preferred to a bronchial blocker as this provides protection to the dependent lung while allowing the involved lung to be suctioned. Only the dependent lung is ventilated while the lumen to the nondependent lung is left open for drainage or suction.

In some instances, the obstruction can behave as a ball valve. Positive pressure ventilation distends the bronchus and allows gas to pass the obstruction during inflation, but then the gas is trapped during deflation. A progressive rise in peak inspiratory pressure and a decrease in expired volume compared with inspired volume may indicate gas trapping. Ventilation of only the dependent lung is extremely important in this situation.

Nd-YAG laser

The neodymium–yttrium–aluminum–garnet (Nd-YAG) laser is used to debulk tumors and to tunnel through or widen obstructed airways.[255] The Nd-YAG laser beam can be transmitted through a flexible quartz monofilament, so it can be used with either rigid or flexible bronchoscopes. Although the procedure can be performed under local anesthesia, general anesthesia with muscle relaxants is preferred as any movement could result in a misfired laser causing damage to healthy tissue. With general anesthesia, there is improved airway control, and debris and blood are more easily removed so that aspiration is avoided.

The patient should not be premedicated with a sedative or opioid. Atropine or glycopyrrolate can be used as a drying agent. After intravenous induction and muscle relaxation, an inhalational anesthetic agent is used. Patients should be ventilated with an $F_{I}O_{2}$ of 0.4 or less and 60% or more nitrogen (from inspired air) to reduce the chance of an airway fire. Nitrous oxide, which is combustible, should be avoided. No special endotracheal tube is needed since the laser is only fired beyond the tip of the tube. However, if misfired, tube ignition is possible. The laser should only be fired deep in the airway, but damage to the anesthesiologist's eyes is possible from a misfired laser, so goggles or glasses are recommended.

In an alternative technique, with total intravenous anesthesia (TIVA) with propofol or remifentanil, laser surgery may be performed through a rigid bronchoscope.[256] This technique requires a high concentration of O_2 delivered through jet system, so combustion is a possibility.

Only the surface of the treated tissue is visible. Any underlying damage and edema will not be apparent, and airway obstruction and hemorrhage can develop as late as 48 h after the procedure. Hypoxemia, perforation of the tracheobronchial tree, and airway hemorrhage are major complications of Nd-YAG procedures. The surgeon may need to apply epinephrine-soaked pledgets directly if bleeding is profuse.

Airway stenting

Placement of a tracheal or bronchial stent will maintain patency of airways with stenosis and malacia. Stenting is particularly useful following pulmonary transplantation. Either silicone stents or self-expanding stainless-steel stents are used.

For general anesthesia, a fiberoptic bronchoscope-assisted airway intubation of an awake, cooperative, spontaneously breathing patient allows visualization while minimizing the risk of airway obstruction. An inhalational anesthetic induction is an alternative choice. Once the trachea is intubated, there is still the possibility that the airway distal to the endotracheal tube can collapse so a rigid bronchoscope should be available to re-establish a patent airway if necessary.

The stenting procedure requires an immobile field for accurate airway length measurements and for positioning of the stents, therefore muscle relaxants may be needed. Associated bronchial balloon dilation of the airway can be extremely stimulating, so a rapid-acting intravenous anesthetic agent should be available and given if the patient moves.

All inhalational anesthetic agents have bronchodilatory effects which are useful because of the frequent presence of reactive airway disease in patients undergoing stenting procedures. Intravenous agents such as propofol or remifentanil can maintain anesthesia during the periods of bronchial dilation and stenting when ventilation with an inhalational agent is not possible.

Stenting procedures are usually performed in the confined, unfamiliar, environment of the radiology suite. One must always be prepared for complications. Common problems can include marked resistance to ventilation from a misplaced or dislodged stent or even airway rupture. Emergence from anesthesia can also be challenging. The anesthesiologist must have all the necessary airway equipment available in order to re-establish a patent airway, including tracheostomy.

Pulmonary hemorrhage

Protecting the noninvolved airway is the first priority in the management of massive pulmonary hemorrhage. Death usually results from drowning in blood, rather than from hypovolemia or exsanguination. The methods used for isolating the lung during pulmonary hemorrhage include a DLT, bronchial blockade with the Univent tube, or placement of a single-lumen endotracheal tube into the bronchus of the nonbleeding lung. If bleeding is massive and visualization of the larynx is difficult, awake laryngoscopy and intubation is recommended.

Advancing an uncut endotracheal tube to its full length will usually intubate the right main bronchus. If bleeding is coming from the right lung, the tube may have to be passed over a fiberoptic bronchoscope into the left lung. If the left lung is bleeding, an endotracheal tube inserted into the right main bronchus will probably obstruct the right upper lobe as well as isolating the entire left lung, so hypoxemia may occur.[257] Confirmation of lung isolation by bronchoscopy is necessary before the patient is turned to the lateral decubitus position.

Once the airway is established and the contralateral lung is protected, attention is directed toward detecting and controlling the site of bleeding while maintaining adequate gas exchange. The site of hemorrhage is determined by bronchoscopy. The larger lumen of a rigid bronchoscope makes the suctioning of greater volumes of blood easier, but fiberoptic bronchoscopy through a tube in the airway is safer.

Video-assisted thoracoscopic surgery

Insertion of an endoscope into the thoracic cavity is an accurate, safe, and reliable alternative to open thoracotomy for a broad spectrum of intrathoracic procedures. Video-assisted thoracoscopic surgery is used to obtain pulmonary and pleural biopsies and for limited lung resection, laser treatment of tumors, lung volume reduction surgery (LVRS), treating spontaneous and traumatic pneumothoraces, sympathectomies, chest wall resections, and limited cardiac, orthopedic, and many other intrathoracic operations.[258,259]

Patients undergoing video-assisted thoracoscopic surgery procedures range from healthy, low-risk (e.g. bilateral sympathectomies) patients to those with very severe pulmonary disease (e.g. LVRS).[260] Therefore, each anesthetic will vary depending on the patient, and the choice of monitors will depend on the overall status of the patient and not necessarily on the procedure alone. The benefits of video-assisted thoracoscopic surgery include less pain, better postoperative pulmonary function, and more rapid recovery with a shortened hospital stay.

Video-assisted thoracoscopic surgery can be performed under local, regional, or general anesthesia, the choice depending on the duration and extent of the operative procedure.

When the incision is made, air enters the pleural cavity, causing a partial pneumothorax. If the procedure is performed using local anesthetics with the patient awake, the pneumothorax is usually well tolerated because the skin and chest wall form a seal around the thoracoscope, thus limiting the degree of atelectasis.

Several rules apply for all video-assisted thoracoscopic procedures. When general anesthesia is indicated, a DLT or bronchial blocker must be used because collapse of the operated lung is essential. DLTs offer the advantage of allowing passage of a fiberoptic bronchoscope or suction catheter to aid lung collapse and for pulmonary toilet.

Ventilation to the lung undergoing surgery is discontinued by opening the lumen of the DLT to air. Collapse of the lung undergoing surgery occurs when air enters the pleural cavity. Intermittent suction down the lumen of the DLT may help to hasten collapse. To augment selective collapse, CO_2 has been insufflated into the pleural space. This maneuver should be avoided because it can cause marked hemodynamic instability owing to a response similar to that of a tension pneumothorax.[261,262]

If the patient undergoing video-assisted thoracoscopic surgery becomes hypoxemic, the application of CPAP is not practical as even a modest distention of the lung undergoing surgery will compromise surgical exposure. The combined use of NO and a pulmonary vasoconstrictor to correct hypoxemia during video-assisted thoracoscopic surgery may be beneficial.[188]

Pain after video-assisted thoracoscopic surgery is unpredictable and analgesic requirements vary greatly between individual patients. Systemic opioids remain the analgesic regimen of choice. NSAIDs, intercostal nerve blocks, and even epidural opioids have been used. TENS has been used with some success in patients undergoing video-assisted thoracoscopic surgery.[216]

Local anesthesia (0.25% bupivacaine plus 0.5 mL kg^{-1} epinephrine) administered through the chest drainage tube at the completion of surgery after the lung has been reinflated also works well. The local anesthetic solution must be given before the drainage tube is placed on suction.

Bronchopleural fistula

A bronchopleural fistula is a pathologic communication between the airway and the pleural cavity. The fistula may extend to the skin (bronchopleural–cutaneous fistula). Bronchopleural fistulae occur following pulmonary resections (most common after pneumonectomy), from rupture of a bullae, bleb or cyst into the pleural space, from erosion of a carcinoma, and from trauma.

If an empyema is present, it should be drained prior to surgery on the fistula. The safest means is to drain the empyema under local anesthesia with the patient sitting up and leaning toward the affected side. A drain, connected to an underwater seal, should be left in the cavity. The empyema may be loculated, so complete drainage of the empyema is not always possible.

The most important anesthetic concerns when managing a bronchopleural fistula are separation and isolation of the lungs to avoid contamination and selective ventilation of the healthy lung. These goals are best achieved with a DLT.

Prior to intubation, the chest drainage tube should be left unclamped for continued drainage of pus and to avoid a tension pneumothorax. Intubation can be performed in an awake patient or under general anesthesia provided the patient breathes spontaneously. Controlled ventilation must be avoided until the lungs are completely separated, i.e. until the DLT is in place, to prevent a tension pneumothorax.

The bronchus of the healthy lung should be intubated. Because a tube "blindly" advanced can pass through the fistula, a fiberoptic bronchoscope is used to direct the DLT into the noninvolved bronchus. Once in position, the bronchial cuff should be inflated immediately to separate the lungs. Only the healthy lung is ventilated. In the presence of an empyema, there may be a massive outpouring of pus through the tracheal lumen immediately after intubation.

Bronchopleural fistulae can be treated without surgery using a DLT to independently ventilate each lung ("split-lung" ventilation). The healthy lung is ventilated with a normal tidal volume while the affected lung is ventilated with a smaller volume or is left unventilated. CPAP can also be applied at pressures below the opening pressure of the fistula. The critical opening pressure is found by adding small increments of CPAP to the affected bronchus until continuous bubbling appears in the underwater sealed chest drain. High-frequency jet ventilation is ineffective and may make the bronchopleural fistula worse.[263]

Lung cysts and bullae

Thin-walled, air-filled cavities in the lungs (cysts and bullae) may represent end-stage emphysematous lung changes or may be congenital and isolated findings. Repeated pneumothoraces from spontaneous rupture, infection, and/or dyspnea are the usual indications for surgery. The degree of functional impairment depends on the condition of the remaining lung, the size of the airspace, and the amount of compression of the surrounding healthy lung tissue by the cyst.

Patients with spontaneous pneumothoraces can be divided into two clinical groups. The first have apical blebs and are usually young with excellent pulmonary reserve. The second group have emphysematous blebs, are older, and have advanced COPD often with CO_2 retention. These latter patients would benefit by avoiding thoracotomy. Video-assisted thoracoscopic resection of bullae and blebs is usually attempted in both groups, although those in the emphysematous group frequently requires open thoracotomy.

If the bulla is very compliant, during controlled ventilation a large portion of the applied tidal volume will be wasted in this additional dead space. Positive pressure ventilation should be used with caution in case the bulla ruptures, leading to a tension pneumothorax. Nitrous oxide must always be avoided. Once the chest is opened, the risk of a tension pneumothorax disappears.

Intubation with a DLT in a spontaneously breathing awake or anesthetized patient is recommended. Positive pressure ventilation with rapid small tidal volumes at pressures less than 15 cmH_2O can be used during induction and maintenance of anesthesia. Once the DLT is in place, the nonaffected lung (if free of bullae) is ventilated with a normal

tidal volume. After resection of each bulla, the operated lung should be re-expanded and checked for air leaks and for the presence of additional bullae. Most patients can be extubated at the completion of surgery, but if postoperative controlled ventilation is needed, low positive pressures should be used to reduce the chance of developing a tension lung cyst or pneumothorax.

Bronchopleural lavage

The whole lung may be irrigated in the treatment of alveolar proteinosis, bronchiectasis, refractory asthma, inhalation of radioactive dust, and cystic fibrosis.[264] When bilateral lung disease is present, ventilation–perfusion scans should be obtained preoperatively so that lavage can be performed first on the more severely affected lung.

With the patient under general anesthesia, a DLT is used to isolate the lungs. The bronchial lumen should enter the bronchus on the side to be lavaged. An F_IO_2 of 1.0 is used throughout the procedure. The cuff of the bronchial lumen should maintain separation of the lungs at pressures as high as 50 cmH_2O in order to prevent the leak of lavage fluid into the healthy, ventilated lung. Some anesthesiologists prefer the lavaged lung to be dependent so that the risk of leakage to the healthy (nondependent) lung is reduced. Others prefer the lavaged lung to be nondependent as in this position perfusion will more closely match ventilation in the dependent lung. As a compromise, lavage may be performed in the supine position.

Warm, heparinized, isotonic saline is infused by gravity down the bronchial lumen from a height of 30–40 cm above the chest while the patient is in a head-up position. When the lavage fluid ceases to flow, the patient is placed in a head-down position, and chest percussion and vibration are applied for 1 min to loosen material. The infused fluid is then allowed to drain passively in to a collecting system. As many as 20 repetitions of this procedure may be required before the drained lavage fluid becomes clear. Accurate measurement of inflow and outflow volumes is important. Following the last passive drainage, the lung is suctioned and then re-expanded using large tidal volumes and high pressures. Most patients can be extubated in the operating room.

The most serious complication of this procedure is aspiration of the lavage fluid into the ventilated lung. This may result in a marked decrease in oxygenation requiring immediate reinstitution of two-lung ventilation with PEEP.

Lung volume reduction surgery

Lung volume reduction is the only surgical treatment for emphysema. It is performed using either video-assisted thoracoscopic surgery with laser ablation of the lung[265] or sternotomy with surgical excision of lung tissue.[266] Not all patients with advanced emphysema are candidates for lung volume reduction surgery (LVRS).[267] Table 47.7 lists the selection criteria.

The aim of either LVRS operation is to reduce the lung volume by 20–30%. The mechanism of benefit by reducing nonfunctioning lung tissue is to enhance elastic recoil of the remaining lung and to improve the efficiency of the diaphragm.[268] This in turn leads to better matching of ventilation to perfusion and improved right ventricular filling.

The lungs must be isolated and ventilated with a DLT or bronchial blocker. Higher levels of potentially dangerous auto-PEEP are produced if a small DLT is used in patients with advanced COPD.[98] Therefore, it is very important to use a large DLT. Because of significant obstruction to expiratory flow, the ventilator tidal volume, rate, and inspiratory–expiratory ratio must be adjusted for the individual patient to avoid air trapping. Unlike the usual practice of ventilating the nonoperated lung with large tidal volumes, smaller tidal volumes are used because of the presence of bullous emphysema in the ventilated lung. The ventilator must be adjusted to provide a long expiratory–inspiratory ratio (1:4 or 1:5), and maximum peak inspiratory pressure during one-lung ventilation should be limited to 35 cmH_2O.

A contralateral tension pneumothorax should be considered if S_pO_2 drops and peak inspiratory pressure rises during one-lung ventilation. Air trapping with hyperinflation of the ventilated lung is another potential cause of increased airway pressures. Transient hypoxemia can result from mucus plugging, so frequent suctioning for pulmonary toilet is indicated. Significant parenchymal air leaks are present in almost every patient following LVRS.

An arterial line is mandatory for continuous pressure monitoring and for frequent blood gas analysis. A pulmonary artery catheter may be helpful for patients with pulmonary hypertension especially in the presence of right ventricular hypertrophy.[269]

Permissive hypercapnia must also be accepted since these patients are intentionally hypoventilated. End-tidal CO_2 levels may not reflect the higher arterial P_aCO_2 because of significant dead space wasted ventilation. Despite very high P_aCO_2 levels at the completion of surgery, most patients have their airway extubated in the operating room. After surgery, the patient must be continuously monitored and the anesthesiologist must be available.

Assisted ventilation by mask may be needed in the immediate postextubation period. Equipment must be available should reintubation and mechanical ventilatory support become necessary.

The key to the anesthetic management of patients undergoing LVRS, whether performed through a sternotomy incision or by video-assisted thoracoscopic surgery, is satisfactory postoperative analgesia. Epidural analgesia with smaller amounts of opioids should be used even when LVRS is performed by the video-assisted thoracoscopic surgery approach. If the epidural ceases to function, it should be replaced. Parenteral opioid analgesia should be avoided if possible.

Lung transplantation

Indications for lung transplantation include infectious (cystic fibrosis, bronchiectasis), obstructive (COPD, α_1-antitrypsin deficiency), restrictive (idiopathic pulmonary fibrosis), or severe pulmonary hypertension.

Table 47.7 *Selection criteria for lung volume reduction surgery (LVRS) patients*

Medical history
Severe COPD (emphysema rather than chronic bronchitis)
Age < 75 years
No cigarette smoking for 6 months
"Lowest" effective prednisone dose
No previous chest surgery
Pulmonary function
FEV_1 > 15% but < 30–35% of predicted
TLC > 120% of predicted
P_aCO_2 < 50 mmHg
Cardiac function
If suspect PA hypertension, measure PA pressure (< 35 mmHg)
No evidence of LV dysfunction (normal dobutamine stress test)
Radiographic
$\dot{V}/\dot{Q}$ scan with predominantly upper lobe disease – decreased upper lobe perfusion
CT scan with heterogeneous distribution of air trapping – emphysema with upper lobe predominance
Hyperinflation, flattened diaphragm (chest radiograph)

COPD, chronic obstructive pulmonary disease; FEV, forced expiratory volume; TLC, ; PA, pulmonary artery; LV, left ventricular; CT, computed tomography.

In some instances, patients with COPD may undergo LVRS as a "bridge" or as an alternative to transplantation. Patients with cystic fibrosis, bullous emphysema, or vascular diseases may require double-lung transplantation, whereas patients with Eisenmenger syndrome may require combined heart–lung transplantation.

Patients are usually dyspneic at rest. Many are hypoxemic, and CO_2 retention is not uncommon. Most procedures are performed on an emergency basis to minimize ischemia time, but donor lobectomy for lung transplantation in children can be scheduled on an elective basis.

Transplant recipients are usually started on cyclosporine (cyclosporin) preoperatively, and intravenous azathioprine is also administered before the start of anesthesia. Premedication with opioids or sedatives is avoided because of the tenuous respiratory status of these patients.

Many patients do not tolerate the supine position and must have their backs elevated during induction. Cricoid pressure is needed for those at risk for aspiration. Induction usually consists of etomidate, ketamine, and/or high doses of opioid to avoid hypotension.

Hypoxemia and hypercarbia must be avoided to prevent further increases in pulmonary artery pressure. Hypotension is treated with vasopressors. Large fluid boluses are avoided. An anesthetic technique combining opioids with an inhalational agent and 100% O_2 and muscle relaxants is recommended. Nitrous oxide is not used since it exacerbates pulmonary hypertension, reduces the concentration of inspired O_2, and expands intravascular air bubbles. Arterial blood gases are followed so that acid–base balance can be maintained. A pulmonary artery catheter is also required, but may have to be withdrawn into its sterile sheath if the pulmonary artery on the side of transplantation has been catheterized.

A DLT, Univent tube, or bronchial blocker is required for single-lung transplantation through a lateral thoracotomy incision. Cardiopulmonary bypass should also be available. Hypoxemia during lung collapse or marked pulmonary hypertension when the pulmonary artery is clamped may necessitate the need for cardiopulmonary bypass. Prostaglandin E_1, amrinone or milrinone, nitroglycerin, and dobutamine may be required to control pulmonary hypertension and prevent right ventricular failure. More recently, NO has been used to treat pulmonary hypertension during pulmonary transplantation.[270,271]

Most patients require ventilatory support for 24–48 h after transplantation. If cardiopulmonary bypass is not used, or once coagulation studies return to normal, a thoracic or lumbar epidural catheter should be placed for administration of neuraxial opioids.

Cyclosporine may exacerbate renal dysfunction. Mannitol and furosemide may be required postoperatively to induce diuresis. Inotropic support with dopamine, isoproterenol, and epinephrine may be necessary, especially if pulmonary hypertension and right ventricular failure are present. TEE is helpful in differentiating between right ventricular and left ventricular dysfunction and for evaluating pulmonary blood flow after transplantation.

Transplantation disrupts neural innervation of the transplanted lung. Although the respiratory pattern is not affected, the cough reflex is abolished below the carina and there is bronchial hyperactivity in some patients. Loss of lymphatic drainage increases extravascular lung water, predisposing the transplanted lung to pulmonary edema. Intraoperative fluid replacement should be kept to a minimum.

Atelectasis and retained secretions require frequent postoperative bronchoscopy. Other surgical complications include damage to the phrenic, vagus, and recurrent laryngeal nerves.

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