

SECTION 1 Pre-operative Considerations

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Thoracic anatomy

DAVID MORRICE

The knowledge of certain aspects of thoracic anatomical arrangements is of great importance to the thoracic anesthetist. It assists in the correct positioning of endobronchial tubes, the identification of diseased lobes, in an understanding of the proposed surgery and also the potential complications that might occur. The availability of computerized tomography (CT) also means that the anesthetist can utilize this to his/her advantage in predicting difficulties in major cases. However, to interpret these, an understanding of normal anatomy is a prerequisite.

Tracheal, bronchial, lobar division

The course and division of the airway is readily seen on flexible and rigid bronchoscopy.

Trachea

This conduit for air and exhaled gases arises from the lower border of the larynx at approximately the level of cervical vertebra C6. The trachea descends in line with the vertebra and moves slightly to the right and posteriorly in doing so. It consists of 16–20 C-shaped cartilaginous rings that provide a semi-rigid structure. Posteriorly the longitudinal trachealis muscle (non-striated) completes the tube

structure. This muscle layer has the appearance of a flowing river and provides an easy landmark to orientation when performing a fiberoptic bronchoscopy. Bifurcation occurs at the level of thoracic vertebra T4. Thus the average length of the trachea in an adult male is 15 cm. The usual anteroposterior diameter is 20 mm.

Throughout its course the esophagus lies directly behind the trachea, with the recurrent laryngeal nerves lying in the groove in between. In the upper (extra thoracic) trachea, the isthmus of the thyroid overlies the trachea anteriorly with the thyroid lobes lying laterally. The relations of the trachea are shown in Figure 1.1. Moving down into the thoracic cavity the thymus overlies the trachea not far below the sternal notch, and below this are arterial vessels arising from the aorta below. These are from right to left, the brachiocephalic artery, left carotid, and below this the ascending aorta arches in front of the trachea, giving in addition to the above the left common carotid and subclavian arteries. During mediastinoscopy the brachiocephalic artery may be compressed by the rigid bronchoscope, affecting the blood supply to the right arm. The medial aspect of the right upper lobe lies against the trachea. The recurrent laryngeal nerves innervate the upper trachea.

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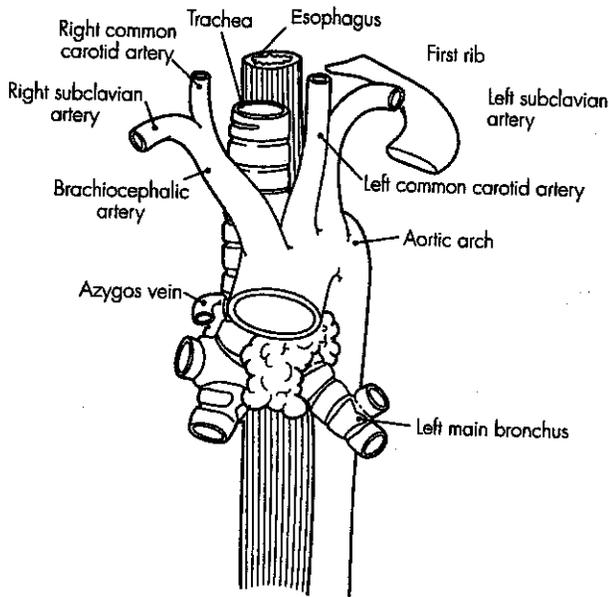


Figure 1.1 The relations of the trachea.

The bronchi and bronchial tree

The bronchial tree with its divisions is illustrated in Figure 1.2.

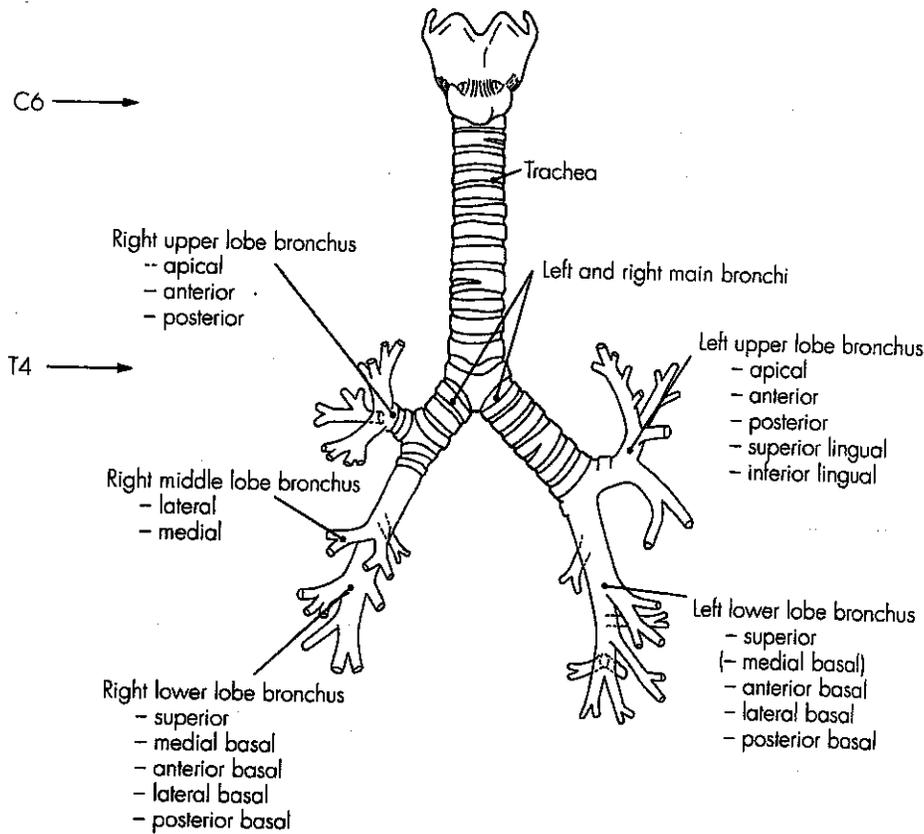


Figure 1.2 The bronchial tree.

The carina and surrounding structures

The last tracheal ring is larger and wider than the others, forming a sharp sagittal ridge called the carina (Latin for the keel of a boat). The carina marks the bifurcation into right and left bronchi. Several important tracheobronchial lymph nodes lie within close proximity and tumor spread to these nodes may blunt the sharp edge of the carina. In addition mediastinoscopy to biopsy these nodes will reveal the intimate relations of the pulmonary artery, aorta and branches and superior vena cava. The pulmonary artery is particularly at risk of injury during this procedure.

Right and left bronchi

The right main bronchus compared to the left is shorter and descends more vertically, i.e. at 25° compared to 45°. This leads to a tendency for endobronchial tubes to favor entry to the right. It ends

when the right upper lobe orifice branches out after 2.5 cm. This lobe consists of three segments (apical, anterior and posterior).

The right upper lobe orifice is directed at 90° from the right main bronchus and may need some bronchoscopic maneuvering to visualize. The apical segment is directed vertically (the other two horizontally); hence on a "slice" of CT it appears as a small well-defined circle. This can be used to aid the localization of tumors within the lungs. Occasionally this lobe may arise higher, even from the trachea.

Following this first division the main bronchus continues as the bronchus intermedius for 3 cm until the middle lobe branches in a direction medially and downwards. Two segments are contained in the medial lobe (lateral and medial) which both project horizontally. This medial lobe is anteriorly placed and wedged between the anterior segment of the upper lobe and the anterior basal segment of the lower lobe.

Thereafter the main bronchus supplies the lower lobe. First to branch off horizontally, opposite the medial lobe orifice, is the apical segment of the lower lobe, worth mentioning because it is directed posteriorly. It is thus prone to collecting secretions in the supine patient. Four further branches are made in a downward direction, all to basal segments of the lower lobe. Thus the right medial lobe and apical portion of the right lower lobe divide at a similar level and appear as a row of three orifices, sometimes being referred to as the secondary carina.

On the left side the bronchial divisions supply the left upper lobe, lingula and lower lobe. At about 5 cm from the carina, the upper lobe bronchus branches to supply both the upper lobe and the lingula. The upper lobe bronchus is 1 cm long and supplies apical, anterior and posterior segments. The upper lobe is difficult to inspect due to its vertical take off and the anterior and posterior segments are also vertically orientated. The lingular orifice

appears more in line with the left main bronchus (i.e. obliquely), and its bronchus is 2–3 cm long. It divides into two segments, superior and inferior, which also head in an oblique direction. In a similar fashion to the right middle lobe, the lingula is anteriorly placed and wedged between anterior segments of the left upper and lower lobes. The left lower lobe bronchus is directed downwards with division firstly into an apical segment arising from the posterior wall and directed horizontally. Below this the bronchus heads vertically downwards and divides into anterior, lateral and posterior basal branches.

Importance of bronchopulmonary segments

There are 20 segments as described above and each can be considered as being discrete physiologically functional units, as each segment has its own separate arterial supply, and venous and lymphatic drainage. The divisions are further illustrated in Figure 1.3. In the case of a lobectomy e.g. for neoplasia the surgeon will have to ensure correct isolation of each of these vessel types to avoid venous congestion or ischemia of other parts of the lung. As a rule each segment is pyramidal shaped and will receive a single branch of the pulmonary artery to perfuse the alveoli. These follow the course of the bronchi, and the division into bronchioles. The bronchus for each segment divides about 15 times into terminal bronchioles. Blood from each segment is drained by intersegmental veins that lie in the connective tissue around the segment, which generally leads to a single branch from each segment. However, the right upper lobe often has additional draining branches of the pulmonary vein. Lymphatics tend to closely follow the course of the bronchi. These drain into tracheobronchial lymph nodes located at the bifurcation of the larger bronchi.

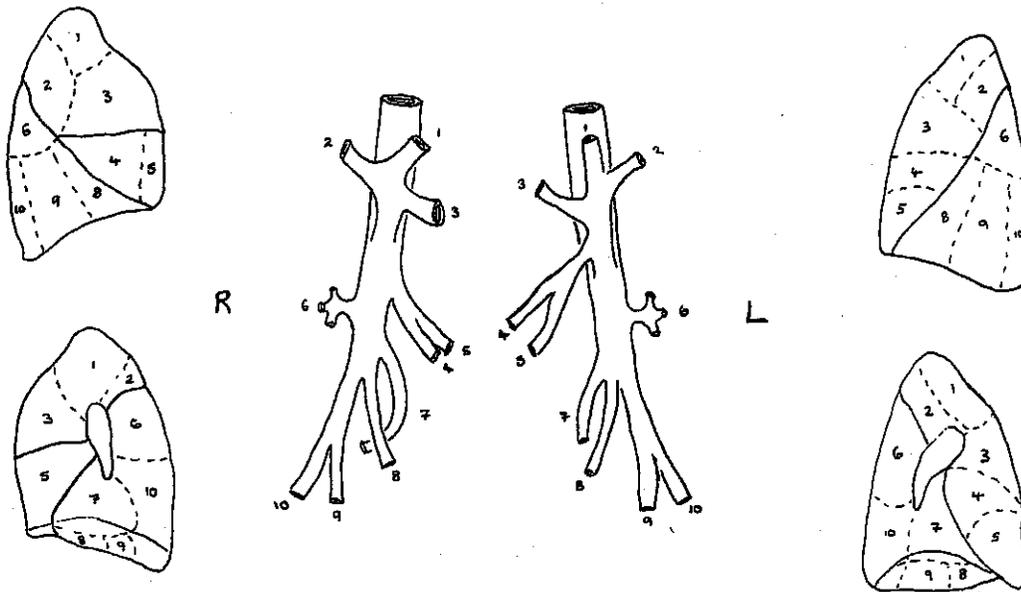


Figure 1.3 The bronchopulmonary segments.

Alveolar units

The terminal bronchioles further divide into respiratory bronchioles from which alveoli arise, finally ending in sacs of alveoli. Deoxygenated blood from the tissues is pumped forward from the right heart via the pulmonary arteries and intrasegmental branches to the fine capillary beds around the alveolar sacs. Here, as described elsewhere, O₂ and CO₂ gas exchange occurs. Thereafter oxygenated blood drains into the pulmonary vein system, which collects in the intersegmental septa and drains ultimately into the left atrium.

Bronchial arteries

These smaller vessels supply the stroma of the lung including the bronchi, pleura and nodes. They follow the posterior aspect of the bronchi as they divide. There are usually two left bronchial arteries arising from the descending thoracic aorta and a right arising variably from the aorta or intercostal arteries. Bronchial veins drain only the more proximal bronchial divisions and may be susceptible to compression by edema as they cross the bronchial wall.

The hilum

The structures that enter the lung are the pulmonary arteries, veins, the primary bronchi, pulmonary nerve plexi and bronchial arteries. Where these structures enter and leave the lung is termed the hilum.

The pleura

Each pleural sac is a closed cavity lined by a serous membrane invaginated by a lung. The outer wall of the chest is lined by the parietal pleura while the visceral pleura cover the lung. The layers of pleura are continuous around the root of the lung. The parietal pleura lines the ribs, costal cartilages and the intercostal spaces, extending superiorly beyond the thoracic inlet to form the cervical dome of pleura. Inferiorly it forms a narrow gutter around the margin of the diaphragm, the costodiaphragmatic recess, and similarly anteriorly in front of the heart, the left costal and mediastinal surfaces are in contact forming the costomediastinal recess. The pleura is supplied by blood from the tissues it covers. The visceral pleura has no pain fibers but the parietal pleura has a rich nerve supply from nerves in adjacent tissues. The lymphatic drainage of the visceral

pleura is to a superficial plexus in the lung and then to hilar nodes, and the parietal pleura drains to parasternal, diaphragmatic and posterior mediastinal nodes.

The paravertebral space

The borders of the thoracic paravertebral space are imprecise. It is a wedge-shaped space with its base being formed by the lateral surface of the vertebral body and intervertebral foramen. It is thought that the prevertebral fascia and anterior longitudinal ligament usually form a barrier to communication to the contralateral paravertebral space, breached only by lymphatic channels. The posterior wall is formed by the inner surface of the vertebral transverse process, the neck of the rib and the attached superior costotransverse ligament. The lateral boundary is formed by the ribs and internal intercostal muscles. The anterior wall is the parietal pleura.

The diaphragm

The diaphragm is a musculotendinous septum separating the thoracic and abdominal cavities. It consists of a peripheral muscular part attached to the edges of a central trilobed tendon (see Figure 1.4).

The peripheral muscular part is divisible into three sections by its attachments:

1. Sternal part – from the back of the xiphoid process by two muscular slips.
2. Costal part – from the inner surfaces of the lower six ribs and costal cartilages, interdigitating with transverse abdominis.
3. Vertebral part – from the sides of the bodies of the upper lumbar vertebrae by two crura and from the medial and lateral arcuate ligaments on each side. The right crus is attached to the bodies of the first three vertebrae and the left crus to the first two. The larger right crus passes forwards and to the right surrounding the esophageal opening. The medial arcuate ligament is the thickened upper edge of the psoas fascia and passes from the body of the first lumbar vertebra to its transverse process. The lateral arcuate ligament is anterior to quadratus lumborum passing from the transverse process of the 1st lumbar vertebra to the 12th rib.

The two halves of the diaphragm are supplied by the corresponding phrenic nerves. The periphery also receives additional sensory branches from

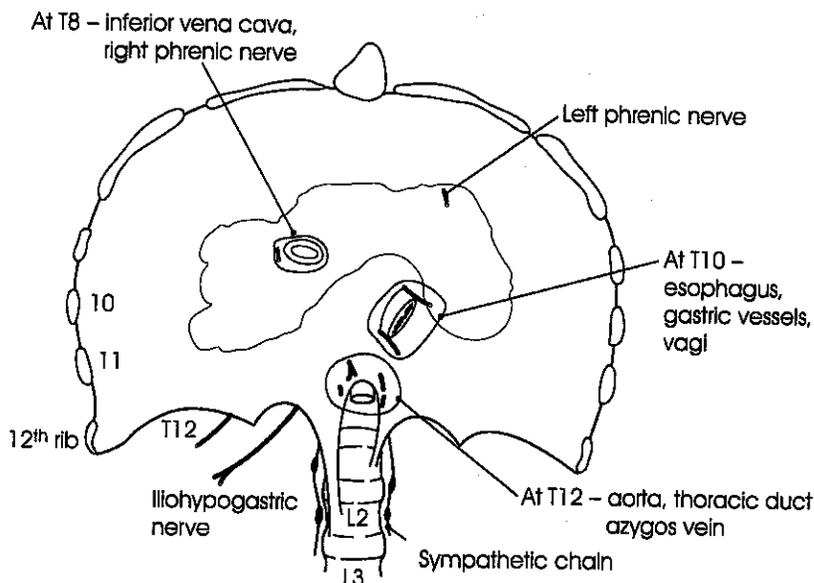


Figure 1.4 The diaphragm.

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intercostal nerves. There are three large openings in the diaphragm from before forwards: for the inferior vena cava (with the right phrenic nerve) in the central tendon to the right of the midline at the level of the 8th thoracic vertebra; for the esophagus at the level of T10 as above (with branches of the vagus nerve and esophageal branches of the left gastric vessels); and for the aorta, between the crura of the diaphragm in front of T12. This also transmits

the thoracic duct and azygous vein. The left phrenic nerve pierces the left dome of the diaphragm.

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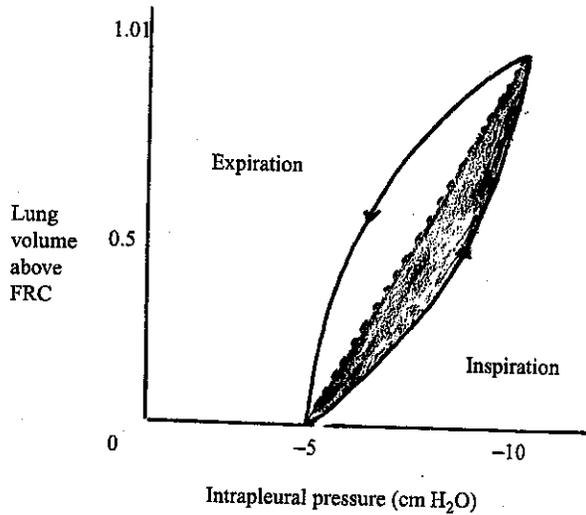


Figure 2.2 Compliance of the lungs.

Dead space

Gas exchange in the respiratory system only occurs in the alveoli. The part of the airway that does not participate in gas exchange is called the dead space. The total dead space consists of the "anatomical" dead space and the "physiological" dead space. The "anatomical" dead space consists of the mouth, nose, pharynx, trachea and main bronchi, and is equivalent to approximately 150 ml. The anatomical dead space functions as a conduit in which the air is filtered of dust particles, humidified and warmed. The functional dead space is normally equivalent to the anatomical dead space. If alveoli are ventilated but no gas exchange is taking place,

Box 2.1 The Bohr equation

$$V_D = \frac{V_T(F_{A\text{CO}_2} - F_{E\text{CO}_2})}{F_{A\text{CO}_2}}$$

Where

V_D is the functional dead space

V_T is the tidal volume

$F_{A\text{CO}_2}$ is the fractional CO_2 concentration in the alveolar gas (can be determined from the terminal portion of air expired)

$F_{E\text{CO}_2}$ is the fractional CO_2 concentration in the expired gas

then these contribute to the functional dead space. The volume of total dead space can be calculated from the CO_2 content of alveolar gas and the tidal volume using the Bohr equation (Box 2.1).

Gas exchange in the lungs

The movement of O_2 and CO_2 in and out of the capillaries both in the lungs and in the peripheral tissues depends on gas diffusion. This in turn is affected by three main factors:

1. The partial pressure gradients of each gas.
2. The diffusion coefficient for each gas.
3. The physical properties of the tissues at the site of exchange (surface area, diffusion distances).

The lungs are well adapted for gas diffusion with a large alveolar surface area and a very thin layer of fluid and tissue separating alveolar gas from pulmonary blood.

Ventilation:perfusion ratio

Normal gas exchange requires both that the alveoli are adequately ventilated and that they are adequately perfused. This relationship is quantified by the alveolar:perfusion ratio, V/Q . V/Q = alveolar ventilation rate/pulmonary blood flow. When this deviates from normal, ventilation-perfusion mismatch occurs. If an area of the lung is inadequately ventilated but adequately perfused, V/Q will be reduced. Blood passing through such areas will be inadequately oxygenated reducing the partial pressure of O_2 in the systemic arterial blood: physiological shunting of blood. This is a major factor contributing to the abnormal blood gases seen in many respiratory diseases.

Pulmonary blood flow

Virtually all the cardiac output passes through the lungs, at arterial pressures of about one-sixth of systemic. The overall pulmonary blood volume is about 500 ml but only 80 ml is in the capillaries. Pulmonary vascular pressures are mainly influenced by gravity. When in the erect position,

Respiratory physiology

CAIT P. SEARL

The primary task of the lungs is respiration. Respiration is the exchange of gases between an organism and its environment with the utilization of O_2 and production of CO_2 . In a multicellular organism such as man, diffusion pathways are too long for the rapid delivery of O_2 and removal of CO_2 . The circulating blood provides a transport system to carry the respiratory gases between the lungs and the distant cells. Oxygen in the inspired air reaches the pulmonary alveoli (ventilation) where it diffuses into the blood whereas CO_2 diffuses in the opposite direction.

Respiratory mechanics

Bulk flow of air in and out of the lungs is achieved by pressure gradients between the mouth and alveoli. These pressure gradients are achieved by movement outward and inwards of the chest creating changes in pleural pressure and hence alveolar pressure changes. When the gas is stationary alveolar and mouth pressures are the same and at atmospheric pressure. Whether the air is flowing or not, the pleural pressure is affected by the inward elastic recoil of the lungs.

Inspiration is an active process: muscular contraction increases the volume of the chest, the lungs expand and the intrapulmonary pressure in the

alveoli falls so that the air flows into the lungs. During expiration the lungs and chest recoil to the positions they occupied at the beginning of inspiration. Expiration is largely passive. During quiet breathing, the diaphragm accounts for around 75% of the lung volume change by its contraction during inspiration and relaxation during expiration. The diaphragm by itself or the scalene and external intercostal muscles alone can maintain adequate ventilation at rest. Expiration is achieved by passive recoil but can be assisted by contraction of the abdominal muscles and the internal intercostal muscles.

Respiratory volumes

The volume in the lungs at maximal inspiration is the total lung capacity (TLC; approximately 6 liters). Its subcomponents are inspiratory reserve volume (IRV), tidal volume, expiratory reserve volume (ERV) and residual volume (RV). The first three comprise the vital capacity (VC) and the latter two comprise the functional residual capacity (FRC) (see Table 2.1 and Figure 2.1). These volumes and capacities increase with body size and are smaller in females. There is a reduction in elastic recoil of the lungs and stiffening of the chest wall with aging. This leads to a gradual increase in RV and FRC and a fall in VC with little change in TLC.

Table 2.1 Lung volumes and capacities.

Ventilatory volumes

Tidal volume (TV)	Amplitude of the oscillation in lung volume during quiet respiration, usually about 400-500 ml
Inspiratory reserve volume (IRV)	Maximum volume of air which can be inspired in excess of normal inspiration
Expiratory reserve volume (ERV)	Maximum volume of air which can be expired in excess of normal expiration
Residual volume (RV)	The volume of air remaining in the lungs after maximal expiration; $RV = FRC - ERV$

Lung capacities

Total lung capacity	Represents the sum of all the ventilatory volumes plus the residual volume
Vital capacity (VC)	The sum of the ventilatory volumes; the volume of gas that is expelled from the lungs from peak inspiration to peak expiration
Functional residual capacity (FRC)	Volume of gas left in the lungs at the end of quiet expiration
Inspiratory capacity	Equals tidal volume plus inspiratory reserve volume

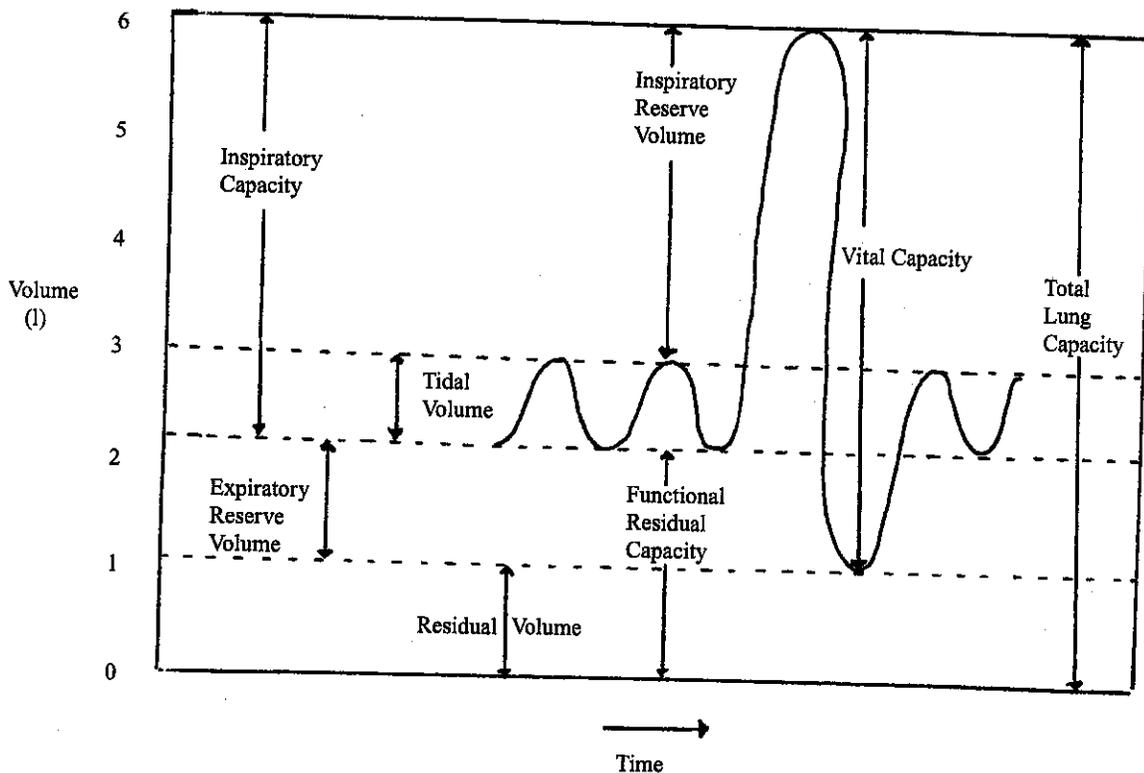


Figure 2.1 Lung volumes and capacities.

Lung compliance

Compliance is a measure of the difficulty of inflation of the lungs. It can be determined from the gradient of a plot of lung volume against distending pressure. This relationship demonstrates hysteresis but an average compliance can be determined using a linear interpolation (see Figure 2.2).

The most important physiological measure is the compliance of the intact respiratory system (i.e. the compliance of the lung and chest wall together). This is usually about 11 kPa^{-1} ($100 \text{ ml/cm H}_2\text{O}$). This may be reduced by diseases of the lung such as pulmonary fibrosis or by abnormalities of the chest wall.

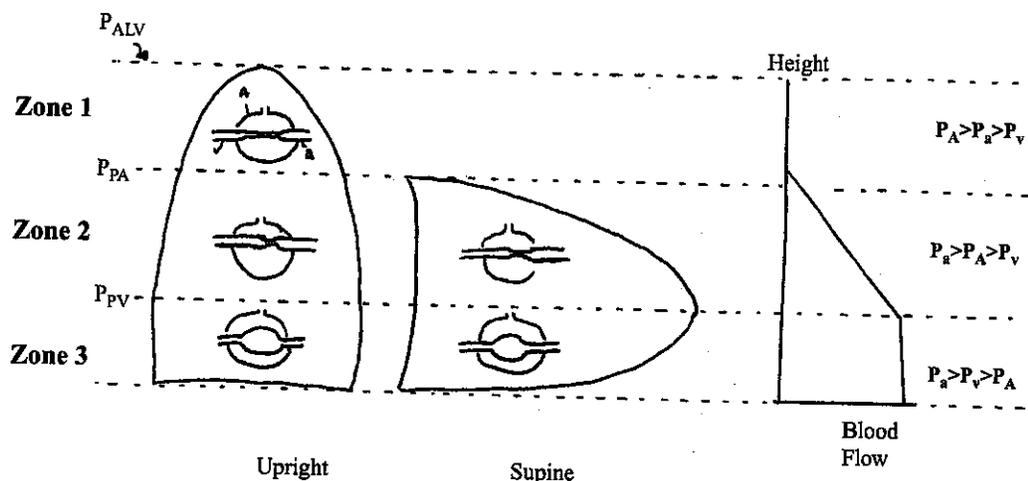


Figure 2.3 The 'West' zones of the lungs showing blood flow through the erect and supine lung and the influence of gravity.

blood flow is nearly zero at the apex and greatest at the bases (see Figure 2.3) as defined by West. This variation is greater than the similar variation in ventilation, resulting in the highest ventilation:perfusion ratio at the top of the lungs. This remains largely true whether the body is supine, prone or in lateral position rather than upright – the zones remaining from uppermost down as depicted. When lying down or in lateral decubitus position zone 1 may not apply as the height is not sufficient.

Hypoxic pulmonary vasoconstriction

Hypoxic pulmonary vasoconstriction continues to attract interest because of persistent mystery about its biochemical mechanism (Box 2.2) and its exact physiological function. Recent work suggests an important role for pulmonary arteriolar smooth muscle cell oxygen-sensitive voltage-dependent potassium channels. Inhibition of these channels by decreased PO_2 inhibits outward potassium current, causing membrane depolarization, and calcium entry through voltage-dependent calcium channels. Endothelium-derived vasoconstricting and vasodilating mediators modulate this intrinsic smooth muscle cell reactivity to hypoxia. Hypoxic pulmonary vasoconstriction seems to decrease with

age, and exhibits marked inter-species and inter-individual differences. The magnitude of HPV in vivo is inversely proportional to lung segment size. The main determinant of HPV is the alveolar partial pressure of O_2 , but mixed venous O_2 contributes to approximately one fifth of the response.

Box 2.2 Hypoxic pulmonary vasoconstriction

HPV is inhibited by

- mediators e.g. Substance P; Calcitonin; atrial natriuretic peptide
- endothelium-derived vasodilators e.g. prostacyclin; nitric oxide
- α -adrenergic blockade
- β -adrenergic stimulation
- increased left atrial pressure
- increased alveolar pressure
- alkalosis
- peripheral chemoreceptor stimulation
- vasodilating drugs e.g. calcium channel blockers; halogenated anesthetics.

HPV is enhanced by

- acidosis
- β -adrenergic blockade
- epidural blockade
- low dose serotonin
- inhibition of cyclo-oxygenase (aspirin, NSAIDs)
- inhibition of NO synthase (L-arginine analogs).

Respiratory pharmacology

CAIT P. SEARL

The twin requirements of respiration (ventilation and perfusion) result in the lungs both being exposed to the external environment and the internal environment through the bloodstream. This is utilized in the uptake, accumulation and metabolism of both environmental and blood-borne "foreign" substances including drugs and environmental pollutants. The importance of the lungs in the pharmacokinetics of many drugs is often forgotten. There are several metabolic pathways known to be present in the endothelial tissues: the cytochrome P-450 monooxygenase enzymes are likely to be particularly important. Many of the drugs that we use as anesthetists are metabolized in the lungs including sympathomimetics, antihistamines, opiates and local anesthetics (Box 3.1).

A thoracic anesthetist needs to be familiar with the drugs that "respiratory" patients are likely to be receiving and also with their potential use in aid of anesthesia and post-operative management. It is also useful to be aware of potential effects of the drugs that we utilize in anesthesia on the respiratory system.

Drugs active in the respiratory tract *Drugs used in the treatment of asthma and bronchospasm*

Asthma is clinically characterized by recurrent episodes of coughing, wheezing and dyspnea. It

is characterized by increased responsiveness of the trachea and bronchi to various stimuli and by widespread airway narrowing. The pathology consists of contraction of airway smooth muscle and mucosal thickening from edema and cellular infiltration. Bronchospasm results from a combination of a release of mediators and an exaggerated response to their effects. Therapy is directed at relaxing the airway smooth muscle, reducing bronchial responsiveness and preventing mast cell degranulation.

BRONCHODILATORS

1. **Direct relaxants of respiratory smooth muscle** – methylxanthines such as theophylline; act by reducing the breakdown of cAMP through the inhibition of phosphodiesterases. Methylxanthines are administered orally or intravenously and can have a number of side-effects due to the increased concentration of cAMP in other systems, causing nervousness, tremor, diuretic activity; secretion of gastric acid; and positive chronotropic and inotropic effects. These compounds have a relatively narrow therapeutic range. They are no longer used as first-line therapy.
2. **Selective Beta₂ adrenergic agonists** – e.g. salbutamol, terbutaline. The β_2 agonists act

Box 3.1 Compounds cleared/metabolized by the lungs

Adenosine
Amphetamine
Angiotensin I (converted to angiotensin II)
Atrial natriuretic peptides
Bradykinin
Bupivacaine
Chlorpromazine
Fentanyl
5-Hydroxytryptamine (serotonin)
Imipramine
Isoprenaline
Lidocaine
Metaramine
Methadone
Morphine
Norephedrine
Prostaglandin E₁, E₂ and F_{2a}
Steroids

Compounds released from the lung

Adenosine
Heparin
Histamine
5-Hydroxytryptamine
Leukotriene A₄, B₄, C₄, D₄ and E₄
NO
Plasminogen activator
Prostaglandin I₂, E and F

primarily on airway smooth muscle and are the most effective form of bronchodilator treatment.

3. **Anticholinergic agents** – muscarinic antagonists inhibit the effects of vagal-released acetylcholine at muscarinic receptors in the airways. Atropine is the classic muscarinic antagonist but systemically has no selectivity, limiting its usefulness. Ipratropium delivered by inhaler is poorly absorbed and so has few systemic side-effects. Although the onset can be delayed (up to 45 minutes), the effects are prolonged.

4. **Mast cell stabilizers** – The chromones (sodium chromoglycate and sodium nedocromil) act predominantly by stabilizing mast cells.

LEUKOTRIENE ANTAGONISTS

The cysteinyl leukotrienes cause smooth muscle constriction and proliferation and are important mediators in the inflammatory process. Montelukast and zafirlukast block the effects of cysteinyl leukotrienes in the airways through antagonizing their actions at leukotriene receptors. They are effective when used in asthma both alone and in addition to an inhaled corticosteroid, having an additive effect with the latter.

MAGNESIUM SULPHATE

Intravenous magnesium has been used to supplement the bronchodilatory effects of inhaled B₂ agonists.

GLUCOCORTICOID THERAPY

Inhaled corticosteroids are amongst the most important treatment agents for bronchospasm as they both increase the number of beta₂-adrenergic receptors and their responsiveness to stimulation. They also reduce mucous production and hypersecretion and inhibit the inflammatory response. Systemic glucocorticoid therapy may also be necessary both with acute severe attacks of bronchospasm and episodes of bronchospasm that are failing to respond to inhaled bronchodilators.

Relief of cough

Treatment of cough mainly consists of treatment of the underlying cause. A productive cough in general should not be suppressed as this may result in sputum retention. Cough remedies are categorized into antitussives and expectorants. Antitussive agents may be either centrally or peripherally acting. The centrally acting agents, including dextromethorphan and codeine, work by depressing the medullary cough center or associated higher

centers. Peripherally acting agents may act on either the afferent or efferent side of the reflex pathway and include demulcents, local anesthetics, humidifying aerosols and steam inhalations. Inhalation of water containing volatile substances such as eucalyptus oil may via the deliberate inspiration of warm moist air provide symptomatic relief in bronchitis.

Mucolytics

Mucolytics are prescribed in order to facilitate expectoration by decreasing the viscosity of sputum. They have been shown to benefit some patients with chronic obstructive airways disease and chronic cough with a reduction in exacerbations of the condition. The treatment can be with oral therapy such as carbocysteine and methyl cysteine hydrochloride or by a nebulized route for example dornase alfa. This latter is mainly recommended for use in patients with cystic fibrosis.

Effects of drugs used during anesthesia on the respiratory system

Volatile anesthetics

Volatile anesthetic agents are mainly used for the maintenance of anesthesia during thoracic procedures. They cause a decrease in FRC through decreasing chest wall recoil, bronchodilation, inhibition of HPV and blunting of the ventilatory response to hypoxia. These effects mean post-operatively that any residual volatile anesthesia can cause significant impairment of lung function.

Opiates

Opiate drugs are usually necessary for the treatment of pain in association with thoracic surgery. All these drugs will cause respiratory depression although the magnitude of this effect is variable according to the individual drug and according to the timing of administration, route of administration and patient factors such as comorbidities.

Propofol

Propofol is often used to both induce and maintain anesthesia during thoracic procedures. It has a rapid onset and offset allowing rapid recovery. It has relatively little effect on HPV and no differences in intra-operative PaO₂ were demonstrated when compared with a volatile maintained anesthesia. Other comparisons with volatile anesthesia have shown that propofol is associated with less post-operative lung function impairment.

Other induction agents

Thiopentone remains a popular drug for induction of anesthesia. It is known to release histamine and has been associated with bronchospasm in asthmatic patients. In contrast etomidate rarely causes histamine release and is advocated by some as the drug to use in patients who are at risk of bronchospasm. It is however associated with adrenal suppression, specifically cortisol production from 11-deoxycortisol. Ketamine is not commonly used as an agent for induction in adults, but has bronchodilatory properties and has been used in the treatment of asthma. Benzodiazepines such as midazolam and diazepam will cause decreases in tidal volume although they are associated with an increase in respiratory rate. Both agents will decrease hypoxic ventilatory drive with only partial reversal by the antagonist flumazenil.

Muscle relaxants

Most neuromuscular relaxants have no direct drug effect on the lungs beyond the effects induced by muscle relaxation and paralysis. Some, notably atracurium and mivacurium, cause histamine release predominantly with hemodynamic consequences but also with effects on bronchomotor tone. Rocuronium, vecuronium and cisatracurium have relatively little cardiovascular or respiratory side-effects. The main concerns with usage of a muscle relaxant are ensuring that it is adequately

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reversed with no residual effect. Patients with respiratory disease are likely to be particularly sensitive to small decreases in respiratory muscle function secondary to residual muscle weakness. Reversal of neuromuscular blockade is usually assisted with the administration of neostigmine in combination with an anticholinergic agent such as atropine or glycopyrrolate. Even with the addition of the anticholinergic agent there may be a significant increase in airway resistance secondary to the inhibition of endogenous acetylcholine by neostigmine.

FURTHER READING

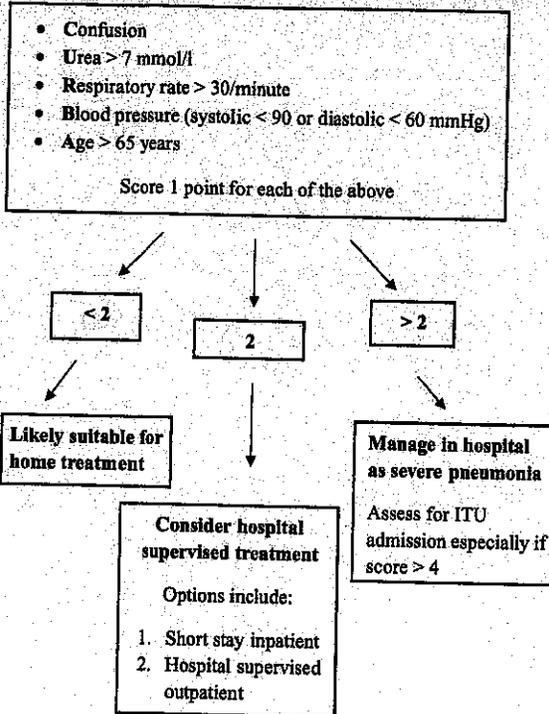
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Box 4.1 Likely causes of pneumonia

- Previously well infant
 - Respiratory syncytial virus
 - Adenovirus
 - Bacterial
- Previously ill infant
 - Staphylococcus*
 - E. coli*
 - Viral agents
- Children
 - Viruses
 - Pneumococcus*
 - Mycoplasma*
- Adults
 - Pneumococcus*
 - Mycoplasma*
 - H. influenzae*
 - Viruses
 - Staphylococcus*
 - Legionella*
- Adults with concurrent respiratory illnesses; the elderly and debilitated
 - Pneumococcus*
 - H. influenzae*
 - Staphylococcus*
 - Klebsiella*
 - Consider tuberculosis; *Legionella*; underlying tumor
- Immunocompromised adults
 - Pneumocystis pneumonia
 - Cytomegalovirus
 - Adenovirus
 - Herpes simplex
 - Bacteria (*Legionella*, *Staphylococcus*, *Pneumococcus*)
 - Opportunistic mycobacteria; tuberculosis
- Hospital-acquired pneumonia in adults
 - Gram-negative bacteria (*Pseudomonas*, *Klebsiella*, *Proteus*)
 - Staphylococcus*
 - Pneumococcus*
 - Anaerobic bacteria; fungi

antibiotic such as clarithromycin. Gram-negative infections such as *Pseudomonas aeruginosa* are commoner as hospital-acquired infections. Guidance from the local microbiologist will guide treatment

Box 4.2 The CURB-65 scoring system



as to likely causes and their antibiotic sensitivities but treatment is often with antibiotics such as cef-tazadime, gentamicin or merepenem.

Tuberculosis

Tuberculosis (TB) is a pulmonary and systemic disease caused by *Mycobacterium tuberculosis* and is spread by airborne droplet transmission. Tuberculosis is a significant worldwide problem with approximately 33% of the human population infected. The human immunodeficiency virus (HIV) epidemic has dramatically altered tuberculous epidemiology. In the UK around 5% of those with HIV are also infected with TB and only about 3% of patients with TB have HIV. However around 98% of patients with HIV are co-infected with TB in developing countries. Tuberculosis represents a complex interaction between *Mycobacterium tuberculosis* and the patient's specific immune response and non-specific resistance

Respiratory diseases

CAIT P. SEARL

In this chapter, common respiratory disease processes will be reviewed. The aim is to give an overview of the disorders that may be met by the thoracic anesthetist and where relevant to consider their anesthetic implications.

Infective disorders

Pneumonia

Pneumonia is defined as infection of the lower respiratory tract parenchyma by infectious agents such as bacteria, viruses and fungi. Pneumonitis is an inflammation of the lung parenchyma caused by non-infectious causes including chemicals, radiation and autoimmune diseases.

The commonest mechanism triggering pneumonia is upper airway colonization with potentially pathogenic organisms that are subsequently aspirated. However the oropharynx may be colonized with such organisms in normal health and the presence of such organisms is not always sufficient to implicate them in the disease process. Pneumonia is the result of a complex interaction between the patient, the infecting organism and the environment. Important factors are the virulence of the causative organism and the vulnerability of the patient. Age and the previous state of health of the patient influence the probability of different causative agents. The likely causes of pneumo-

nia in differing clinical circumstances are shown in Box 4.1.

Patients with pneumonia may present with a history of cough, production of purulent sputum and fever, together with pleuritic chest pain and shortness of breath. The presence of localized chest signs on examination such as crackles or bronchial breathing suggests pneumonia but may not always be present. The severity of pneumonia can be assessed using a scoring system such as the CURB-65 severity score as shown in Box 4.2. The investigations necessary will depend on the severity of the pneumonia: patients with a mild illness responding rapidly to antibiotics do not usually require further investigation whereas more extensive investigations are indicated for patients requiring admission to hospital. A chest X-ray will confirm diagnosis by demonstrating consolidation (Figure 4.1). It may also be helpful in detecting complications such as lung abscess or empyema (see Chapter 17). More specific investigations are aimed at identifying the causative agent – for example, sputum culture +/- a Gram stain; antigen detection tests and serological tests. *Streptococcus pneumoniae* is the most common cause of community-acquired pneumonia, but atypical pathogens such as *Mycoplasma pneumoniae* are also frequent, so treatment is often with a combination of amoxicillin and a macrolide

Control of respiration

Respiration is controlled in the central nervous system with voluntary breathing managed by the cortex and autonomic respiration by structures in the medullopontine region. Efferent output from these two sources is integrated by the spinal cord. Control of automatic breathing is governed by centers in the pons and medulla. These modulate the depth and rate of inspiration. Appropriate blood levels of O_2 , CO_2 and hydrogen ions (pH) are maintained through adjustments of respiration through the medullary center as it responds to afferent inputs from receptors. The medullary center is also important in the maintenance of respiratory rhythm and for the Hering–Breuer reflex, which inhibits respiration when the lungs are stretched. Other inputs to the medullary center include:

- Proprioceptors – coordinating respiration with muscular activity.
- Body temperature.
- Higher CNS centers (cortex, limbic system, hypothalamus) influencing breathing, during, for example, pain, anxiety and sneezing.
- Presso- or baroreceptors – also feeding into the cardioinhibitory area.

Voluntary breath holding inhibits automatic respiration until the break point is reached when the rise in $PaCO_2$ and fall in PaO_2 override the voluntary inhibition. This breakpoint can be delayed by prior hyperventilation.

In patients with chronic CO_2 retention, the medullary center becomes insensitive to changes in $PaCO_2$ so that the PaO_2 is the chief driver of respiration. This is the group of patients in whom breathing 100% O_2 could abolish respiratory drive, eventually causing coma and death. This is relatively uncommon and should not be used as a reason not to administer oxygen.

Non-respiratory functions

Some of the non-respiratory functions are listed below.

- The pulmonary capillary bed acts as a blood filter by removing small clots, detached cells and air bubbles before they reach the systemic system.
- The pulmonary blood vessels act as a reservoir for blood.
- The airways remove airborne particles by a combination of phagocytosis and mucociliary action together with coughing.
- Ventilation of the airways contributes to heat loss and water loss.
- Lung tissue has many metabolic functions, including:
 - Conversion of angiotensin I to angiotensin II;
 - Synthesis and removal of bradykinin and prostaglandins;
 - Storage and release of serotonin and histamine;
 - Inactivation of norepinephrine and epinephrine;
 - Secretion of heparin by mast cells and immunoglobulins in bronchial mucosa;
 - Synthesis of peptides including substance P and opiates.

Effects of anesthesia on respiratory physiology

Under general anesthesia and in the supine position, forced vital capacity (FVC) is reduced by around a fifth in an adult. Obesity and multiple other causes may also further reduce the FVC. A large enough decrease in FVC may bring the end-expiratory volumes below the closing volumes. Closing volumes are the volumes at which small airways begin to close. When the small airways begin to close, areas of low V/Q mismatch develop.

Physiology of spontaneous ventilation in the lateral decubitus position

General anesthesia will reduce the compliance of both upper and lower lungs. This can be returned towards normal by the application of positive

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end-expiratory pressure (PEEP). The weight of the mediastinum and the pressure of the abdominal contents on the diaphragm can impair lower lung expansion and causes decreased FVC. However the greater curvature of the diaphragm will also result in more efficient contraction, with greater expansion matching increased blood flow to the dependent lung. The result is that V/Q remains matched.

During spontaneous ventilation the dependent diaphragm will move towards the head end during expiration, pushing the mediastinum upwards. This produces inefficient ventilation. The resulting mediastinal shift also reduces perfusion because of reduced venous return secondary to sympathetic activation. If the upper chest wall is opened during spontaneous ventilation, paradoxical respiration occurs. During inspiration, gases are drawn out of the upper lung causing it to collapse. During expiration, gases pass from the bottom lung to the upper lung causing it to inflate. This paradoxical respiration causes mediastinal shift generating more work.

Physiology of two-lung ventilation in the lateral decubitus position

Positive pressure ventilation results in most of the ventilation being directed into the upper rather than the lower lung. As perfusion remains greatest in the lower lung, ventilation-perfusion mismatch increases. The result in an anesthetized patient with a closed chest in the lateral decubitus position is a non-dependent lung that is poorly perfused but well ventilated, and a dependent lung that is well perfused but poorly ventilated. Opening the chest wall and pleural space does not change the perfusion distribution but may have a significant effect in terms of ventilation. In the paralyzed, ventilated

anesthetized patient, the non-dependent lung is no longer confined making it easier to ventilate and consequently over-ventilated and under-perfused worsening the mismatch.

Physiology of one-lung ventilation in lateral decubitus position

The blood flow gradient due to gravity favors the dependent lung during one-lung ventilation (OLV). If the non-dependent lung is not ventilated any blood flow to it becomes shunt flow. This will result in a larger alveolar-to-arterial oxygen tension difference with a lower PaO₂ for a given oxygen concentration under identical circumstances, when compared to two-lung ventilation in the same position. In contrast OLV has much less of an effect on PaCO₂ than on the PaO₂. The blood flowing through the relatively under-ventilated alveoli will retain more CO₂ and not take up O₂. As the CO₂ dissociation curve is relatively linear in the physiological range, this favors elimination of CO₂ and the maintenance of relatively normocapnia despite OLV. The O₂ dissociation curve is relatively flat at the top end of the sigmoid-shaped curve and so less of an increase is possible in the uptake of O₂. Hence while the ventilated lung is able to eliminate sufficient CO₂ to compensate for the non-ventilated lung, it is unable to take up sufficient O₂ to compensate in the same way.

FURTHER READING

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- Guyton WF. *Review of Medical Physiology*, 22nd edn. New York: Lange Medical/McGraw-Hill, 2005.

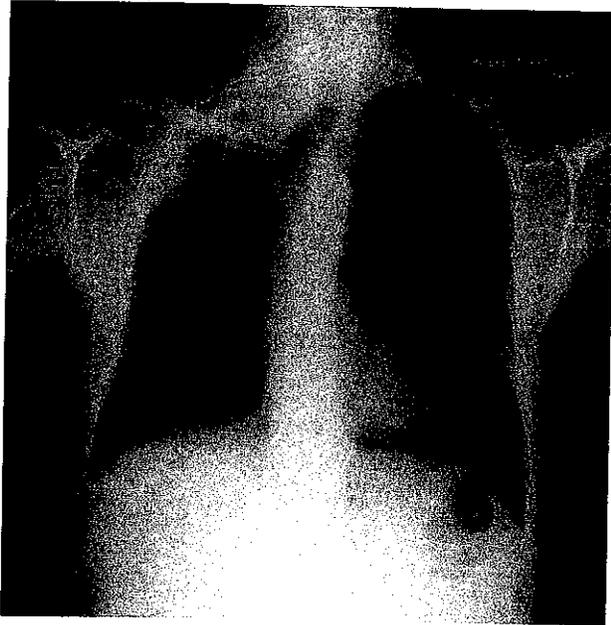


Figure 4.1 Chest X-ray demonstrating pneumonic destruction of the right upper lobe.

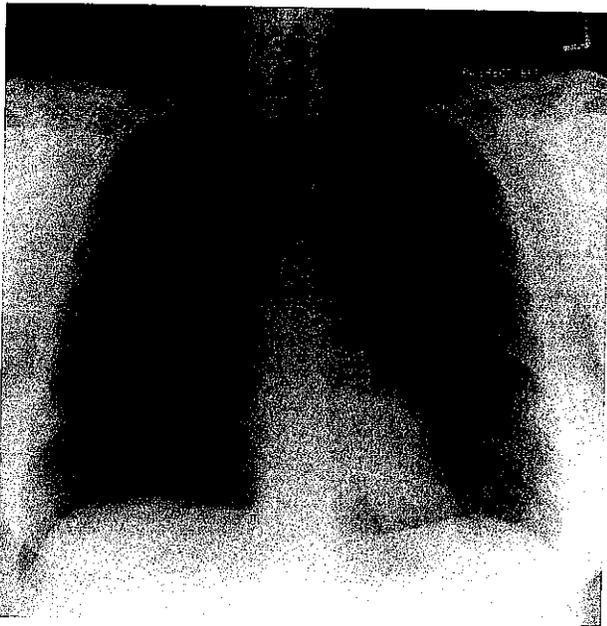


Figure 4.2 Chest X-ray demonstrating an aspergilloma of the right middle lobe.

to infection. Traditionally TB is divided into primary and post-primary TB - these descriptions being based on the characteristic evolution of TB prior to effective chemotherapy. Primary TB is the pattern of disease that is seen in a patient without specific

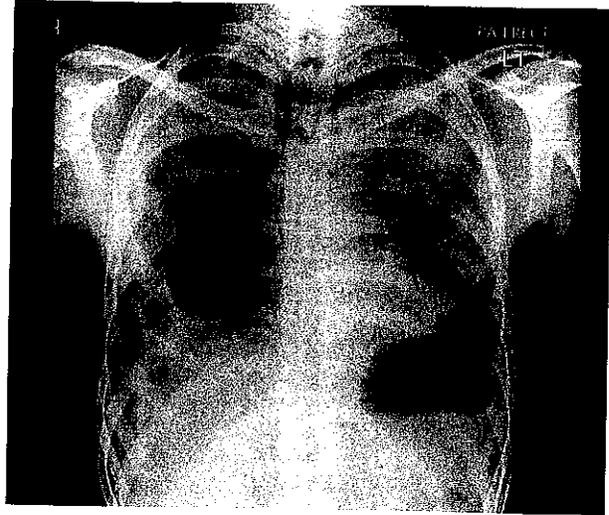


Figure 4.3 Chest X-ray in a patient with silicosis.

immunity to TB. Infection is acquired by inhalation of the organism from an infected individual. The initial lesion develops in the peripheral subpleural region of the lung (Ghon focus) associated with hilar lymphadenopathy. At this stage erythema nodosum may occur. An immune response develops and healing often takes place. This stage is often asymptomatic but may leave calcified nodules on CXR. Active progression of this infection may occur with bronchial spread causing progressive consolidation and cavitation of the lung parenchyma. Lymphatic spread may cause progressive lymph node enlargement, causing bronchial compression with the development of collapse and bronchiectasis. Hematogenous spread causes generalization of the disease and can cause miliary TB and/or TB meningitis. Infection spread during this initial illness can lie dormant in any organ for years and reactivate years later. Post-primary TB is the pattern of the disease seen after the development of specific immunity. It may occur following direct progression of the initial infection or result from an endogenous reactivation of infection or from exogenous re-infection. Reactivation may occur in old age or in circumstances where immunocompetence is impaired (e.g. alcohol dependency;

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immunosuppressive therapy; illness). The disease is most likely to be pulmonary, with the apices of the lungs the most common site. Clinical features tend to be non-specific and for diagnosis to be definitive the organism *Mycobacterium tuberculosis* must be identified. Once the diagnosis is suspected, repeated sputum specimens should be examined for acid- and alcohol-fast bacilli. Chest symptoms include persistent cough, sputum production and hemoptysis. Systemic symptoms may include pyrexia, sweats (particularly at night), anorexia and weight loss. Standard treatment is with 6 months of rifampicin and isoniazid supplemented by pyrazinamide and ethambutol for the first 2 months. At present drug-resistant TB is rare in initial treatments of TB but commoner in patients who have come from Africa or the Indian subcontinent or who have had previous treatment. Multidrug resistance may result from inadequate previous treatment.

Control of TB involves detection and meticulous treatment of cases of active TB, notification of the diagnosis to the public health authorities, contact tracing and targeted vaccination of groups who have a high incidence of TB. Vaccination is with BCG, a live attenuated strain of the bacillus, providing about 75% protection against TB for around 15 years. Routine vaccination of children at the age of 13 in the UK is no longer offered – rather, BCG vaccination is offered to infants in communities with a high incidence of TB and to unvaccinated individuals from countries with a high prevalence of the disease.

In addition to *Mycobacterium tuberculosis*, other mycobacteria can cause pulmonary disease. These are “atypical” or “opportunistic” mycobacteria, the commonest being *Mycoplasma kansasii*, *M. avium-intracellulare*, *M. malmoense* and *M. xenopi*. They are usually low-grade pathogens but can cause infection mainly in patients with impaired immunity or in patients with damaged lungs, e.g. in severe emphysema. They are often associated with chronic symptoms such as cough, sputum produc-

tion, hemoptysis and weight loss. Treatment is often difficult and needs to be prolonged. As these organisms infect only susceptible individuals, there is no need to trace infected individuals’ contacts.

Aspergillus

Aspergillus species are ubiquitous worldwide and are a rare cause of infection in healthy individuals, but in susceptible individuals these fungi can cause significant problems. The fungi are dimorphic, existing as both spores and mycelia. Following inhalation of spores from the environment, a transient saprophytic illness may occur but patients with underlying lung disease can develop persistent colonization. In the saprophytic form the organism can assume a complex pattern of mycelia growth. Depending on the susceptibility of the host, three distinct syndromes can develop:

1. Aspergilloma.
2. Allergic bronchopulmonary aspergillosis.
3. Invasive aspergillosis.

Aspergillomas can present as asymptomatic incidental findings on CXR – see Figure 4.2. They can however be associated with hemoptysis. This is usually minimal although recurrent. Findings are usually of the underlying lung disease rather than associated with the aspergilloma. The treatment is controversial. If associated with frequent or life-threatening hemoptysis, surgical resection is the treatment of choice. However, aspergillomas may spontaneously disappear in around 10% of patients and there is also a variable response to intracavity antifungal treatment. The risk of surgery may be hard to justify in the asymptomatic individual where the underlying lung disease may cause the risk of surgery to be greater than potential risks from the aspergilloma.

Allergic bronchopulmonary aspergillosis is a hypersensitivity lung disease to *Aspergillus* antigens. Patients with atopic asthma are most commonly affected and the disease has its highest incidence in individuals in their 40s. It is commonest in

the UK in the winter months when the air contains the greatest quantity of *Aspergillus* spores. Clinically it presents with bronchospasm. The clinical course is characterized by cough productive of mucoid plugs, hemoptysis, intermittent febrile episodes, chest pain and recurrent pneumonia. Four major findings are suggestive of allergic bronchopulmonary aspergillosis:

1. Recurrent infiltrates on CXR.
2. Blood or sputum eosinophilia.
3. Asthma.
4. Immediate and late (6–8 hours) dermal hypersensitivity to *Aspergillus* antigens.

Treatment includes steroids, bronchodilators and sodium chromoglycate or nedocromil.

Invasive aspergillosis occurs almost exclusively in patients who are already immunocompromised, particularly those with prolonged neutropenia or on high-dose corticosteroids. It involves the lungs in 90% of cases and manifests as a necrotizing bronchopneumonia. Aggressive spread of the disease is its hallmark with metastatic spread causing endocarditis. The clinical manifestations can include dyspnea, non-productive cough, pleuritic chest pain and pyrexia.

Bronchiectasis

Bronchiectasis is usually defined as a persistent and irreversible dilatation and distortion of medium-sized bronchi. It is an acquired disease process that is not a discrete entity but rather the pathological end-stage of a variety of unrelated pulmonary infectious insults and impairment of drainage, airway obstruction or a defect in host defense (see Box 4.3). The morphological changes are usually accompanied by a chronic suppurative lung disease with cough productive of purulent sputum. Bronchiectasis may be confined to a localized area of the lung, where there is a local cause such as bronchial obstruction by a foreign body, or may be diffuse if there is a generalized cause, such as immunoglobulin deficiency.

Box 4.3 The etiology of bronchiectasis

- Severe infection
 - Pertussis (whooping cough)
 - Bacterial pneumonia
 - Recurrent aspiration
 - Tuberculosis
- Bronchial obstruction
 - Foreign body
 - Tumor
 - Hilar lymph node adenopathy
- Cystic fibrosis
- Ciliary dysfunction
 - Primary ciliary dyskinesia
 - Kartagener's syndrome
- Allergic pulmonary aspergillosis
- Associated with systemic disease
 - Rheumatoid arthritis
 - Ulcerative colitis
- Immunodeficient states
 - Hypogammaglobulinemia
- HIV infection
- Idiopathic

The main clinical feature of bronchiectasis is a chronic cough productive of copious purulent sputum. Hemoptysis is common and occasionally severe, requiring therapeutic embolization of hypertrophied bronchial arteries to control. Infective exacerbations may present with pleuritic chest pain and fever. On examination crackles may be audible over affected areas of the lungs and in more severe cases clubbing may be present. A chest X-ray may show features suggestive of bronchiectasis such as parallel tramline shadowing or cystic dilated bronchi, but it may require a high resolution CT scan to confirm diagnosis and to define extent and location of the disease process. Treatment of the specific cause is rarely possible although it is important to identify where possible what the cause is. Chest physiotherapy is the most effective method in preventing accumulation of secretion – particularly postural drainage, percussion and forced expiratory techniques. Antibiotic treatment, guided by the

results of sputum microbiology, is used to suppress chronic infections and to treat exacerbations. Bronchodilator therapy may also be indicated where there is associated reversible airway obstruction. Surgical excision is a potential treatment for individuals who have a localized disease pattern and troublesome symptoms. Lung transplantation may be an option in patients who develop respiratory failure (see Chapter 14).

Lung abscess

A lung abscess is a localized collection of pus within a cavitating necrotic lesion of the lung parenchyma. It presents clinically with a history of cough with expectoration of large amounts of purulent, foul sputum and is often accompanied by hemoptysis, weight loss, pyrexia and general malaise. Drainage of pus from the abscess cavity is the main treatment. This may be achievable through bronchial drainage using postural physiotherapy. It may however be necessary to drain an abscess percutaneously by placing a drain under radiological guidance. Where medical therapy fails, surgical excision of the abscess cavity may be required.

Cystic fibrosis

Cystic fibrosis is the commonest of the potentially lethal inherited diseases in Caucasians, affecting around 1 in 2500 live births in the UK. It is inherited as an autosomal recessive disorder with 1 in 25 of the population being a carrier. Cystic fibrosis results from a mutation to a gene on the long arm of chromosome 7, which codes for a protein named cystic fibrosis transmembrane conductance regulator (CFTR). CFTR functions as a chloride channel in the membrane of epithelial cells causing reduced chloride conductance, most notably in the respiratory, gastrointestinal, pancreatic, hepatobiliary and reproductive tracts (see Box 4.4).

The dysfunction of the CFTR predisposes to severe chronic lung infections via a variety of cel-

Box 4.4 Clinical features of cystic fibrosis

- Upper airways
 - Sinusitis
 - Nasal polyps
- Lungs
 - Infection
 - Bronchiectasis
 - Airway obstruction
 - Pneumothorax
 - Hemoptysis
 - Allergic aspergillus
 - Respiratory failure
- Liver
 - Biliary cirrhosis
 - Hepatosplenomegaly
 - Portal hypertension
 - Gallstones
- Pancreas
 - Malabsorption
 - Malnutrition
 - Diabetes mellitus
- Intestines
 - Meconium ileus
 - Distal intestinal obstruction
 - Rectal prolapse
- Locomotor system
 - Arthropathy
 - Osteoporosis
 - Clubbing
- Other
 - Male infertility
 - Salty sweat

lular mechanisms. In the bronchial mucosa the reduced chloride secretion and increased sodium reabsorption results in abnormally viscous secretions, predisposing to adherence and reduced clearance of bacteria. The high salt concentrations also inactivate the lung epithelial naturally occurring antimicrobial peptides, the defensins. In addition to these defects there are also abnormal mucus glycoproteins which act as binding sites allowing bacteria to adhere to the mucosa and proliferate. As the

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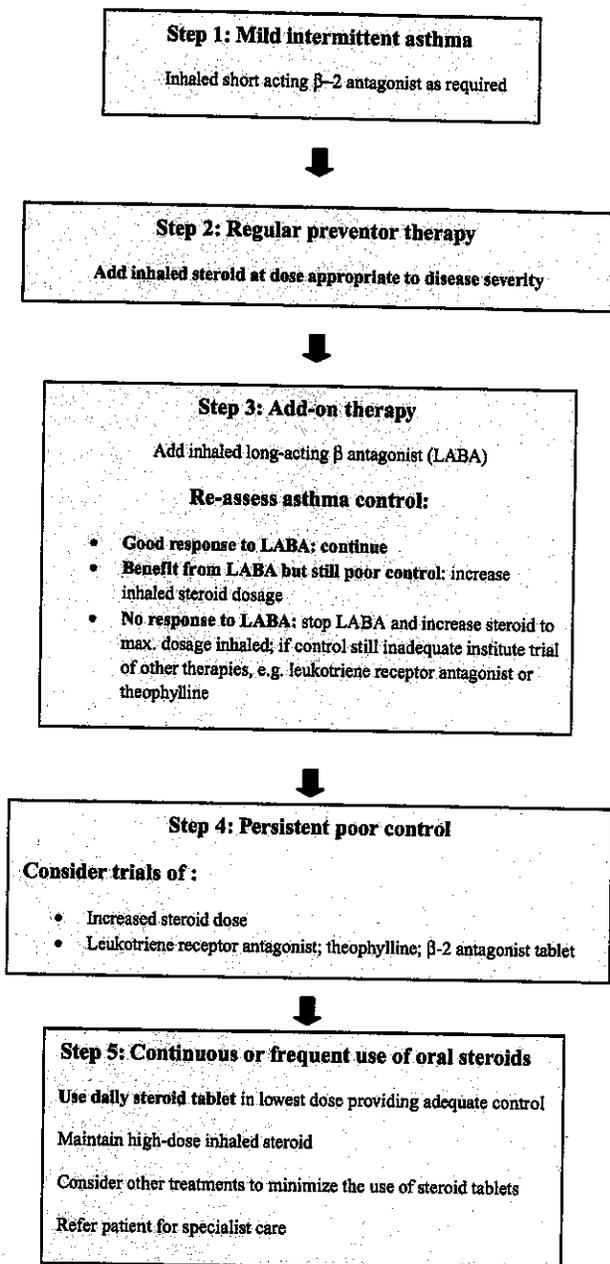


Figure 4.4 Summary of the stepwise management of asthma in adults.

natural histories and therapy options. Whereas in asthma, airway inflammation and hyper-reactivity cause airway obstruction, in COPD alveolar destruction by emphysema causes a loss of elastic recoil and a loss of outward traction in small airways resulting in airway collapse and obstruction with air trapping and hyperinflation. COPD also encom-

passes chronic bronchitis due to mucus hypersecretion in the central airways and defined as cough and sputum production for 3 months of 2 successive years in a patient in whom other causes of chronic cough have been excluded. Tobacco smoking is the main cause of COPD, although only about 15% of smokers develop the disease suggesting additional factors including genetics may be important in the pathogenesis. Emphysema is thought to develop as a consequence of lung destruction by proteolytic digestion due in part to an imbalance of proteases and anti-proteases. In patients with a genetic deficiency of α_1 -anti-trypsin severe emphysema develops at a young age.

ANESTHETIC CONSIDERATIONS IN COPD

Pre-oxygenation may need to be for an extended period to achieve denitrogenation. Induction and maintenance of anesthesia utilizing short-acting drugs may be preferable. The aim is to allow the patient to breathe spontaneously as soon as possible following the procedure to decrease the risks of air leakage secondary to positive pressure ventilation. Anesthesia with total intravenous technique using propofol or using a volatile such as sevoflurane may be preferred to using longer-acting agents. For muscle paralysis, drugs such as vecuronium, rocuronium or cisatracurium might be preferred for both their shorter duration of action and their absence of histamine release.

Difficulty in ventilation in COPD may occur due to airtrapping. The presence of large cystic and bullous areas may induce progressive air trapping and "pulmonary tamponade" physiology resulting in catastrophic reduction of venous return in addition to asphyxia. During ventilation it is important to monitor the volumes being delivered to and exhaled from the lungs to ensure that air is not becoming trapped.

Management of patients with emphysema is further discussed in Chapter 13.

Interstitial lung disease

Interstitial lung disease is an imprecise term referring to a diverse range of diseases which affect the alveoli and septal interstitium of the lung and which can progress to diffuse lung fibrosis. Presentation is usually with progressive dyspnea, a dry cough, lung crackles and diffuse infiltrates on chest X-ray. Lung function tests show a restrictive pattern with reduced vital capacity but a normal FEV₁/FVC ratio; a reduced transfer factor indicating impaired gas diffusion; and hypoxemia that may be accompanied initially by a relative hypocapnia. The next key investigation is usually a high-resolution CT scan as this can give an indication of the extent and pattern of the disease. Investigation should also be aimed at moving from essentially a diagnostic label of pulmonary fibrosis to a more specific diagnosis of a disease process to enable best treatment management: see Box 4.5.

Occupational lung disease

Occupational lung diseases result from the inhalation of dusts, gases, fumes or vapors encountered in the work environment. The effects of these inhaled substances vary depending on their particle size; solubility; toxicity; the duration and intensity of exposure; and the patient's susceptibility. Some substances are generally irritant in a non-specific manner or are toxic to the airways (e.g. ammonia) with all individuals exposed being similarly affected. Other substances include hypersensitivity or allergic reactions in susceptible individuals giving rise to asthma or extrinsic allergic alveolitis (see above). Asthma is the commonest form of occupational lung disease and avoidance of exposure to the inducing agent is the main form of treatment. Other substances promote fibrosis in the lung parenchyma – examples include coal dust causing coalminer's pneumoconiosis; asbestos (see Box 4.6); and silica causing silicosis (see Figure 4.3).

Patients who have suffered disability as a result of occupational lung disease will have a right to compensation from governmental agencies and they may also wish to pursue litigation against their employers. An accurate diagnosis is therefore required and these patients may present for lung biopsy. The death of a patient with a suspected occupational disease must be reported to the Coroner.

Diseases involving the pulmonary circulation

Thromboembolic disease

Pulmonary embolism usually occurs as a complication and consequence of deep vein thrombosis. These typically develop in the deep veins of the legs and then travel to the lungs causing obstruction of the pulmonary vasculature. Factors predisposing to venous thrombosis were described by Virchow as a triad of venous stasis, damage to the vein wall and hypercoagulable states. The clinical features of pulmonary embolism depend upon the size and severity of the embolism and are summarized in Box 4.7. When the diagnosis of pulmonary embolus is suspected, patients should be assessed for compatible clinical features and potential risk factors, with the exclusion of alternative diagnoses. An assay of D-dimer levels may be useful and if there remains a high clinical suspicion then definitive imaging should be performed, either with computed tomography, pulmonary angiography or ventilation/perfusion scanning. Heparin is used to achieve rapid anticoagulation and this is maintained with the use of warfarin. Where there is circulatory compromise from a massive pulmonary embolus thrombolytic therapy may be attempted. Management of chronic thromboembolic pulmonary hypertension is discussed further in Chapter 15.

Unusual forms of embolism may occur including fat (following long bone fractures); amniotic fluid peripartum; and air (usually iatrogenic).

Box 4.5 The differential diagnosis in pulmonary fibrosis

Idiopathic pulmonary fibrosis (IPF) (cryptogenic fibrosing alveolitis)

Commoner in men (M:F 2:1) and in older age groups.

Presents with progressive dyspnea, dry cough, crackles, a restrictive defect in lung function and reticulonodular infiltrates on chest X-ray.

Lung biopsy shows a typical appearance of "Usual interstitial pneumonia" with areas of interstitial fibrosis, inflammation and honeycombing.

A combination of azathioprine and steroid treatment is used but response is often poor and lung transplantation may be necessary.

Idiopathic interstitial pneumonias

- **Non-specific interstitial pneumonia**
Has uniform inflammatory changes and less fibrosis on lung biopsy with ground glass opacification on CT scan.
Responds to corticosteroids.
Has a better prognosis than idiopathic pulmonary fibrosis.
- **Cryptogenic organizing pneumonia**
Seems to be a pattern of response to various insults including amiodarone, connective tissue diseases or ulcerative colitis, but often has no identifiable cause.
Patients often present with cough, malaise, pyrexia and dyspnea with an elevated ESR.
Often responds dramatically to corticosteroids
- **Desquamative interstitial pneumonia**
Relatively rare form of interstitial lung disease affecting smokers with the particular feature of alveolar macrophage desquamation
Responds to cessation of smoking together with corticosteroids

Systemic diseases

- The typical features of IPF can occur in association with a connective tissue disease.
- These diseases may also have a number of other lung complications.
- Examples include rheumatoid arthritis; systemic sclerosis; systemic lupus erythematosus and sarcoidosis.

Inorganic dusts causing e.g. Coalminer's pneumoconiosis, silicosis and asbestosis

Organic dusts causing extrinsic allergic alveolitis

- Extrinsic allergic alveolitis is an immunologically mediated disease in which a hypersensitivity reaction occurs in a sensitized individual to an inhaled antigen. A complex immune response occurs involving antibody reactions, immune-complex formation, complement activation and cellular responses.
- Complete cessation of exposure to the provoking antigen is the main treatment.
- Examples include farmer's lung and bird-fancier's lung.

Inhaled toxins and drugs

- Paraquat.
- Amiodarone.
- Bleomycin.
- Nitrofurantoin.

Box 4.6 Disease conditions related to asbestos exposure

- **Asbestosis**
A pneumoconiosis with diffuse parenchymal lung fibrosis resulting from prolonged heavy exposure to asbestos. Presents 10–25 years after exposure with cough, progressive dyspnea, basal crackles, clubbing and a restrictive ventilator defect with impaired gas diffusion.
Chest X-ray shows bilateral reticulonodular shadowing.
- **Pleural plaques**
Incidental finding on chest X-ray on workers exposed to asbestos.
Do not give rise to any impairment of lung function.
- **Asbestos pleurisy and pleural effusions**
Many years after the initial exposure to asbestos, patients develop episodes of pleurisy with pleuritic pain and pleural effusions.
Pleural fluid is an exudate which is often blood-stained.
Usually spontaneously resolve but are often recurrent episodes.
- **Pleural thickening**
When extensive causes dyspnea and a restrictive defect.
- **Asbestos-related lung cancer**
Increased risk of lung cancer particularly in smokers.
- **Mesothelioma**
Malignant disease of the pleura associated with a history of asbestos exposure in at least 90% of cases.
Average lag period of 20–40 years between exposure and development of mesothelioma.
Presents with pain, dyspnea, weight loss and lethargy.
VAT pleural biopsy may be needed to provide a definitive histopathological diagnosis.
Pleurodesis may control the effusion.
As the tumor progresses it encases the lungs and may involve the pericardium and peritoneum. Blood-borne metastases may occur.
Radical surgery can be attempted but has a poor success rate. Radiotherapy may shrink the tumor and often relieves symptoms of pain. Chemotherapy may also result in tumor shrinkage.
Poor prognosis with the majority of patients dying within 2 years.

Pulmonary hypertension

Pulmonary hypertension is a diagnosis encompassing several distinct disease processes affecting the cardiopulmonary system (see Box 4.8). It is defined as a mean pulmonary artery pressure greater than 25 mmHg at rest. The commonest setting for pulmonary hypertension in thoracic anesthesia is probably in patients who have hypoxemia secondary to chronic lung disease: WHO Class III. In this setting it may be referred to as *cor pulmonale*. This essentially is right ventricular hypertrophy and failure secondary to pulmonary hypertension that has arisen from secondary chronic pulmonary vasoconstriction secondary to hypoxemia.

ANESTHETIC IMPLICATIONS OF PULMONARY ARTERIAL HYPERTENSION

The pulmonary circulation is highly responsive to vasoconstrictive stimuli including vasoconstrictive drugs (e.g. metaramine, noradrenaline); hypercarbia; acidosis; agitation or pain. Increases in pulmonary arterial pressure may cause acute failure of the right ventricle with right ventricular dilatation and septal shift. This causes an acute decrease in left ventricular filling and left ventricular contractility and leads to left ventricular failure. This leads to hypotension, which in turn may lead to decreased coronary perfusion. In patients with pulmonary hypertension and right

Box 4.7 Presentations of pulmonary embolism

Massive pulmonary embolism

- Acute.
 - > 50% occlusion of circulation.
 - Sudden circulatory collapse with cyanosis, chest pain, hyperventilation and engorged neck veins.
 - ECG may show S1, Q3, T3 pattern.
 - Chest X-ray is usually unhelpful.
 - Angiography shows filling defects and poor perfusion.
- Sub-acute.
 - > 50% occlusion of circulation.
 - Progressive severe dyspnea with no obvious cause, dyspnea even at rest.
 - Raised jugular venous pressure, loud P2 on auscultation of heart.
 - ECG may show RV strain pattern.
 - Chest X-ray may show infarcts.
 - Angiography and scan show severe perfusion defects.

Acute minor pulmonary embolism

- With infarction.
 - Pleural pain, hemoptysis, effusion, fever, hyperventilation.
 - Chest X-ray segmental collapse/consolidation.
- Without infarction.
 - May be "silent".
 - Chest X-ray and ECG may be normal.
 - Angiography may show obstruction if early.
 - Scan will show perfusion defects.

Chronic thromboembolic pulmonary hypertension (WHO Class IV)

- Progressive dyspnea and hyperventilation.
- May get effort syncope.
- Clinical features of pulmonary hypertension.
- ECG shows right ventricular hypertrophy and axis deviation.
- Chest X-ray may show a prominent pulmonary artery.
- Angiography may be normal or show slow circulation or peripheral pruning.
- Scan expected to show patchy irregularity.
- See Chapter 15.

Box 4.8 WHO revised classification of pulmonary arterial hypertension

Group I: Pulmonary arterial hypertension

Previously known as primary pulmonary hypertension. Now known as idiopathic pulmonary hypertension. Rare disease particularly affecting young women.

Treatment is specialist and may include prostacyclins such as epoprostenol and iloprost; endothelial receptor antagonists (e.g. bosentan); and selective phosphodiesterase-5-inhibitors (e.g. sildenafil). Surgical options may include atrial septostomy to decompress the failing right heart, and transplantation.

Group II: Pulmonary arterial hypertension associated with left heart disease

E.g. with chronic mitral valve disease.

Group III: Pulmonary arterial hypertension associated with lung disease and/or hypoxemia

Commonly associated with right ventricular hypertrophy and subsequent failure, known as cor pulmonale.

Group IV: Pulmonary hypertension associated with chronic thromboembolic disease

Discussed in Chapter 15.

Group V: Pulmonary arterial hypertension due to miscellaneous causes

Pulmonary hypertension can occur as a complication of collagen vascular diseases such as systemic sclerosis and systemic lupus erythematosus.

Pulmonary hypertension can also arise as a complication of drug therapy such as fenfluramine (an appetite suppressant agent).

Useful reference: Proceedings of the 3rd World symposium on pulmonary arterial hypertension, Venice, Italy. June 23-25 2003. *J Am Coll Cardiol* 2004; **43**(Suppl.12): 51-90.

ventricular hypertrophy blood flow to the right coronary ventricle assumes the diastolic phase flow dependency whereas in normal individuals the flow is both during systole and diastole. This increases susceptibility to hypotension. The response of the right ventricle is dependent on the general status of the patient – those with chronically elevated pulmonary arterial pressures have a “trained” ventricle and can often cope better with increases in pulmonary vascular resistance than those with previously normal pulmonary circulations.

Principles of management of patients with pulmonary hypertension include: identification of those at risk; invasive monitoring; maintenance of intravascular volume and myocardial contractility; and careful use of vasoactive agents. One of the most dangerous periods for these patients is at induction of anesthesia and institution of mechanical ventilation. Both the systemic vasodilation secondary to induction agents and the increase in afterload to the right heart may acutely cause decompensation of the right ventricle. Aims for anesthesia include avoiding systemic hypotension and myocardial depression; and to avoid hypoxia, hypercarbia and metabolic acidosis. The anesthetist should consider monitoring right heart function with central venous pressure and/or pulmonary arterial pressure monitoring and possibly utilize transesophageal echocardiography. Right ventricular function can be improved with inotrope and pulmonary vasodilator therapy. Management of heart failure is further discussed in Chapter 22.

Malignancy

Lung cancer

Lung cancer remains the commonest cause of cancer death worldwide. In the UK it kills about 34 000 people each year. For clinical purposes the disease is classified into two groups.

1. **Small cell carcinoma:** comprising 20% of lung cancers; also known as oat-cell carcinoma, this cancer arises from neuroendocrine cells as is

evidenced by the occasional ectopic hormone production. It is highly malignant, growing rapidly and metastasizing early. Usually by the time of diagnosis it will be widely disseminated such that systemic chemotherapy is the most appropriate therapy.

2. **Non-small cell carcinoma:** comprising the remaining 80% and further divisible into **squamous-cell carcinoma** (45%); **adenocarcinoma** (20%) and **large-cell carcinoma** (15%). Adenocarcinoma has a higher incidence in patients with either localized (e.g. following TB or localized irradiation) or generalized lung fibrosis, so-called “scar carcinomas”. With non-small cell carcinoma, surgical resection can offer the best opportunity for “cure” but only 10–20% of patients prove suitable for surgery. These patients require careful pre-operative assessment and staging of their disease as is discussed in Chapter 5.

Other thoracic neoplasms

Alveolar cell carcinoma

This rare tumor arises in the alveoli of the lungs and spreads along the alveolar and bronchiolar epithelium. It may be associated with the production of large amounts of mucin and copious sputum production. A transbronchial biopsy may provide diagnosis. Surgical resection is the treatment of choice if the tumor is confined to one lobe.

Carcinoid tumor

This tumor is commoner in younger patients and its occurrence is not related to smoking. It tends to be less malignant than bronchial carcinomas and is often slow growing, if locally invasive. Most arise in the main bronchi and present with hemoptysis and wheeze. Most can be cured by surgical resection. Rarely carcinoid tumor from the lung can metastasize to the liver. This may result in the secretion of substances including 5-hydroxyindolacetic

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acid and hence the classic "carcinoid syndrome" of flushing, wheeze and diarrhea.

Mesothelioma

This is nearly always related to asbestos exposure – see Box 4.6.

FURTHER READING

- *British Thoracic Society Guidelines for the Management of Community Acquired Pneumonia in Adults. Update 2004.* Accessed at www.brit-thoracic.org.uk
- *Tuberculosis: Clinical Diagnosis and Management of Tuberculosis and Measures for its Prevention.* Accessed at www.brit-thoracic.org.uk
- *Cystic Fibrosis Trust (UK).* www.cftrust.org.uk
- *British Guideline on the Management of Asthma.* British Thoracic Society/Scottish Intercollegiate Guidelines Network. Accessed at www.brit-thoracic.org.uk
- King TE. Clinical advances in the diagnosis and therapy of interstitial lung diseases. *Am J Respir Crit Care Med* 2005; 172: 268–79.
- *The Diagnosis and Treatment of Lung Cancer: Clinical Guideline 24.* National Institute for Clinical Excellence 2005. Accessed at www.nice.org.uk.

Pre-operative assessment of the thoracic surgical patient

SION BARNARD AND DOUGLAS AITCHISON

Thoracic surgery ranges from small low-risk procedures to major surgery, and for malignant and non-malignant disease. All may run into problems post-operatively. Assessment of the thoracic patient for surgery comprises two distinct areas. The first is the resectability of the lesion if malignant (or, more appropriately, correctability if benign) and the second is the fitness to withstand the morbidity it inevitably involves, referred to as operability by most surgeons. The role of pre-operative assessment in this second area is to assess the comorbidity in what is generally a relatively elderly and unfit population in order to gauge whether they will withstand the degree of surgery planned. Thorough clinical assessment coupled with appropriate further investigation is required. In assessment of resectability, radiological imaging techniques are the most commonly used methods.

When assessing malignancy, one of the major indications for thoracic surgery, it is crucial to follow the usual oncological principles of assessment. Disease must be assessed in terms of local, regional and systemic disease, with appropriate techniques used for each based on prior probabilities of disease and appropriate regional and national guidelines.

Clinical history and examination

A standard history is taken to elicit symptoms and to enable fitness for surgery (and anesthesia) to be assessed. Symptoms may include shortness of breath, chest pain and weight loss. Exercise tolerance should be assessed. A physical examination should pay particular attention to the respiratory system but also to factors that might suggest additional problems peri-operatively, for example a kyphosis. Watching a patient walk allows an informal functional assessment to be made. More formally this is part of lung function testing.

Lung function tests

Spirometry

Spirometry measures the inspiratory and expiratory volumes and more complex techniques using plethysmography also allow estimation of total lung volume. Total lung capacity (TLC) represents the lung volume at maximal inspiration. Forced vital capacity (FVC) represents the maximal exhaled volume. The forced expiratory volume (FEV) is the volume exhaled over a particular time-period. FEV over 6 seconds (FEV₆) is taken to indicate the true FVC during assessment. The forced expiratory volume over the first second (FEV₁) is a reliable

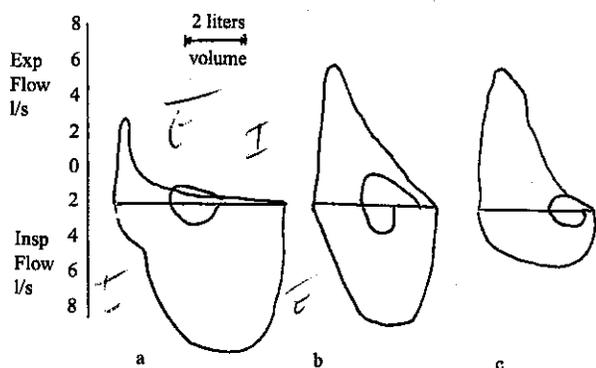


Figure 5.1 Flow volume loops. Loop b is normal while a to the left shows small airways disease and c illustrates tracheal obstruction.

indicator of ventilatory function and is closely associated with post-operative respiratory function, discussed below. The difference between the TLC and the FVC is the residual volume (RV). This is increased in COPD and asthma.

Peak expiratory flow rate is a more reliable indicator of expiratory airflow limitation (e.g. severity of asthma for monitoring purposes) than spirometry and is measured during maximal forced expiration. Minute ventilatory volume (MVV) is the maximal volume that can be breathed over a minute. Spirometry using electronic equipment can also calculate flow-volume loops, useful for interpretation of both the inspiratory and expiratory phases. Stridor, or inspiratory obstruction, is characteristic of large airways disease i.e. of the trachea or bronchus. This is displayed by a very flattened inspiratory limb of the loop. Small airways disease, e.g. asthma, COPD, is more characteristically expressed as a flattened expiratory curve with a long tail and the appearance of an exponential type curve. These are shown in Figure 5.1.

Most of the spirometric values are normally related to height, sex and age, and equations derived from clinical series are used to predict individualized normal data. For surgical use, these are often expressed as percentage of normal predicted values to allow size-independent expression. These are important for calculations of likely post-operative

respiratory function, discussed below under the BTS guidelines for lung surgery.

Gas transfer capacity

Diffusion capacity is usually measured by gas transfer factor using carbon monoxide (CO). Its high blood solubility and hemoglobin uptake is 230 times that of oxygen and practically zero concentration in atmospheric air or blood (at least in non-smokers) enable CO transfer factor (T_LCO) to be easily measured and independent of perfusion. Techniques rely on reasonable tidal volumes (i.e. >0.75 liters) and may be inaccurate if the patient is unable to hold a single breath. Adjustments are made for hemoglobin concentration and alveolar volume to enable normalization of values. Reference ranges for the local center are usually expressed and values may also be expressed as percentage predicted. Like spirometry, these values are important for assessment of fitness for surgery and are discussed below in the BTS guidelines for lung surgery.

Functional tests

These tests are not specific for lung function but enable a reasonably accurate assessment of the performance of combined cardiorespiratory function during exercise, provided there is no limiting musculoskeletal problem. Walking the patient down the corridor to the clinic or office is the simplest. Another is climbing flights of stairs: inability to climb more than a single flight is a very high risk indicator with any surgery; two flights without stopping is adequate for esophageal surgery; three flights as an indicator of good outcome following lobectomy; and five for pneumonectomy. Measured 6-minute walk test or numbers of shuttle runs of 10 meters between cones in 12 minutes or until stopping, are others that are more accurately measured and have been extensively validated. Measurement of the maximal oxygen uptake during exercise on a cycle or treadmill, VO_{2max} expressed as liters per minute or ml/kg per min for

inflammatory response is unable to clear the infection, a continuous cycle of infection and inflammation ensues. This results in progressive lung damage and bronchiectasis and ultimately respiratory failure and death. Clinical features of the respiratory component include persistent cough and purulent sputum production typical of bronchiectasis, development of digital clubbing and progressive airway obstruction with associated wheeze. Serial measurements of FEV₁ allow some monitoring of the severity and progression of disease process. Culture of sputum may initially isolate *Staphylococcus*, *Haemophilus influenzae* and *Streptococcus pneumoniae*, but in older children and teenagers, isolates are likely to include mucoid strains of *Pseudomonas aeruginosa*, sometimes with pan-resistance to antibiotic therapy. As the cycle of lung damage progresses with destruction of the lung parenchyma, increasing airway obstruction and increasing impairment of gas exchange, patients develop hypoxemia, hypercapnia and right-sided heart failure with cor pulmonale. Hemoptysis is common as the persistent inflammatory response provokes hypertrophy of the bronchial arteries. Pneumothoraces may occur, particularly in advanced disease, and may require pleurodesis if recurrent. As respiratory failure intervenes, pulmonary transplantation may be required (see Chapter 14).

Cystic fibrosis is a complex disease involving all the body's systems and therefore a multidisciplinary approach is required to the treatment of patients with the disorder. The optimal treatment is probably achieved through management at regional specialist centres. The basic elements of treatment comprise clearance of the bronchial secretions through physiotherapy; treatment of pulmonary infection by antibiotic therapy (guided by microbiological advice); correction of nutritional deficits by the use of pancreatic supplementation and dietary support; and psychological/social support for both the patient and their family.

Airway obstruction disorders

Asthma

Asthma is a disease characterized by chronic airway inflammation with increased airways responsiveness and airways obstruction. Symptoms include wheeze, dyspnea and cough. Airway obstruction is variable and may be reversible with therapy. Asthma represents a clinical syndrome that is diverse in presentation and in effects. It is multifactorial in origin but appears to arise from a combination of environmental factors and genetic susceptibility. There is a general consensus that asthma is increasing in prevalence and that this is likely to be due to environmental factors. The British Thoracic Society recommends a stepwise approach to managing asthma according to its severity (see Figure 4.4). The aim of management is to abolish symptoms, to restore normal airway function and to reduce the risk of severe life-threatening attacks. For the majority of patients the disease is controlled by a combination of a regular inhaled steroid and using an inhaled bronchodilator drug as required for symptomatic relief. The drugs used in the treatment of asthma are discussed further in Chapter 3.

ANESTHETIC CONSIDERATIONS IN ASTHMA

It is generally advisable to use an anesthetic technique that is unlikely to provoke bronchospasm. This is particularly important in thoracic anesthesia as the added airway manipulation from bronchoscopy, placement of DLT or bronchial blocker may also provoke bronchoconstriction.

Chronic obstructive pulmonary disease

Chronic obstructive pulmonary disease (COPD) is defined as a chronic slowly progressive disorder characterized by airflow obstruction. Although there is some overlap with asthma, they are different disorders with different etiologies, pathologies,

body-weight adjusted values, is the most complex but also the most closely related to fitness for surgery. A very close correlation between outcome following surgery and need for heart or lung transplantation has been shown with this test. Values below 10 ml/kg per min are insufficient for most surgical procedures. Values above 15 ml/kg per min are satisfactory for lobectomy, with several series reporting no increase in post-operative risk for any procedure with VO_2max above 20 ml/kg per min.

Arterial blood gas analysis

Measurements of arterial blood gases (ABG) allow an understanding of the carriage and delivery of oxygen, carbon dioxide and with a co-oximeter, levels of carbon monoxide or levels of methemoglobin containing iron in its ferric (Fe^{3+}) form that is incapable of oxygen uptake. Absolute values of pre-operative arterial carbon dioxide pressure are not directly related to outcome following surgery, with a resting hypercapnia not a contraindication to surgery, but resting hypoxia with saturations below 90% or significant desaturation on exercise are both indicators of increased operative risk.

Tremor or peripheral venous dilatation often represent hypercapnia and merit ABG analysis. Increased respiratory rate and accessory muscle use indicate underlying impairment to airflow. Arterial blood gases should also be performed on any patient cyanosed at rest, with peripheral oxygen saturation 94% or below or on oxygen therapy pre-operatively.

Radiological investigations

Initial imaging techniques involve the plain chest film, traditionally involving PA and lateral images. Sensitivity for detecting small lesions is reasonable but not as high as CT scans but the radiation exposure is very low and the investigation is widely available. Usually, localization of the lesion and approximate size estimation are possible, along with assessment of the rest of the lung fields, cardiac

silhouette and the bones of the thoracic cage. More subtle signs such as presence of emphysematous bullae, pulmonary fibrosis, right-sided paratracheal lymphadenopathy (by increased tissue density adjacent to the trachea and right main bronchus which is normally adjacent to lung tissue) and loss of volume may also be observed. However, investigation is rarely limited to plain films as further information is invariably helpful for even non-surgical management.

Computerized tomography (CT)

The standard imaging technique for further chest imaging is the CT scan, usually performed to a lung cancer protocol with IV contrast and imaging at 5 mm slices from the root of the neck including the glottis down to the mid or lower abdomen to include the whole liver and spleen. An alternative technique, often used for patients with chest pain is the PE protocol in which contrast administration is timed and the scan often continued down to the lower calf. The former is more suited for thoracic surgery but the presence of tumor or thrombus within the pulmonary arteries is clearer on the latter if relevant. Computerized tomography scanning equipment is improving in sophistication and ability all the time and the newest scanners use helical multi-array sources and detectors to enable scanning of the entire chest within a single breath-hold.

The CT scan allows staging of any lung tumor, assessment for emphysema and bullous disease, shows presence of small or loculated effusions, pleural thickening and the presence of most lymph nodes above 5 mm in diameter. In addition, the tissue density often allows for a degree of diagnostic information, such as that the heavily calcified apical rounded nodule in a young patient previously exposed to TB is probably the benign remnant of a Ghon focus. Likewise, an irregularly bordered spiculated lesion with areas of differing intensity is much more likely to be malignant. Intrabronchial

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Table 5.1 Lung cancer staging: 1 - TNM classification.

Tumor	Definition
T1	Tumor < 3 cm maximal dimension, surrounded by lung, or endobronchial distal to lobar bronchus
T2	Tumor > 3 cm maximal dimension, or reaching visceral pleura, or obstructive atelectasis of less than one lung, or lobar endobronchial tumor or main bronchial tumor > 2 cm from the carina
T3	Tumor involving the apex of the chest, main bronchial tumor within 2 cm of the carina, atelectasis of the entire lung, or tumor of any size invading the chest wall, parietal pleura of the mediastinum (including the phrenic nerve) or pericardium or the diaphragm
T4	Macroscopic or histological invasion of the mediastinal structures including the heart, great vessels, trachea, esophagus, carina, vertebral bodies, recurrent nerve, malignant pleural effusion, distant pleural metastases or multiple neoplastic nodules within the same lung lobe
Nodes	Definition
N0	No lymph node involvement (pN0 on pathological lymph node sampling)
N1	Metastases to hilar, interlobar, lobar or segmental nodes
N2	Metastases to ipsilateral mediastinal or subcarinal nodes
N3	Metastases to scalene or supraclavicular or contralateral nodes
Metastases	Definition
M0	No metastases
M1	Presence of metastases

lesions may also be seen on careful inspection of the lumen on several contiguous slices.

CT - tumor staging

Staging of presumed or confirmed lung cancer is performed according to strict criteria agreed internationally, as shown in Table 5.1. Tumor (T), nodal (N) and metastatic (M) levels are all assessed and classified numerically. An overall stage associated with 5-year survival can easily be read from Table 5.2.

CT - lymph nodal assessment

Lymph nodal assessment by CT must be regarded as anatomical rather than histological and is therefore not completely accurate. The absence of nodal disease is N0, presence of involved (enlarged) nodes within the lung is N1 and those within the mediastinum is N2. Contralateral, scalene or abdominal node involvement is classed as N3. In

Table 5.2 Lung cancer staging: 2 - overall staging. TNM staging presented on y and x axes, with M1 read as any T or N level within each. Approximate 5-year survivals with treatment are listed in percentages for each stage and subgroup.

TNM	N0	N1	N2	N3	M1
T1	IA 60%	IIA 34%	IIIA 13%	IIIB 3%	IV 1%
T2	IB 38%	IIB 24%	IIIA 13%	IIIB 3%	IV 1%
T3	IIB 22%	IIIA 9%	IIIA 13%	IIIB 3%	IV 1%
T4	IIIB 7%	IIIB 7%	IIIB 7%	IIIB 3%	IV 1%
M1	IV 1%	IV 1%	IV 1%	IV 1%	

the setting of lung cancer, sensitivity and specificity for involved lymph nodes range between 70-80%. Lymph nodes vary in size within the chest

dependent upon age, sex and location and are generally oval in shape. Lymph node staging must be used for all enlarged nodes in patients who are potentially operable and the primary tumor resectable. Likewise, for all patients with borderline nodal size criteria or multiple small nodes in whom the primary is very advanced and/or the operability borderline, it is prudent to perform other nodal staging to prevent unnecessary non-curative surgery at greater operative risk. Further methods of lymph nodal staging include operative (mediastinoscopy and others) and functional imaging techniques such as positron emission tomography (PET), as discussed below.

CT – metastatic assessment

A computerized tomography scan is sensitive for the detection of adrenal and liver metastases from lung cancer, along with para-aortic lymphadenopathy. Ultrasound scan or PET may also be required to quantify these and differentiate from benign cysts. The presence of benign coincidental adenomas is a relatively common finding in the older patient and must be carefully diagnosed as it should not preclude curative lung surgery.

Although not generally recommended in the absence of neurological signs or symptoms, CT scan of the head with and without contrast is an important staging procedure in those with advanced lung tumors e.g. chest wall invasion, a possibility of lung secondaries, or with a difficulty in assessing baseline cerebral function. Presence of most cerebral metastases would prevent surgery to resect lung cancer; there is little more unfortunate than performing radical lung surgery and reconstruction only to see the patient succumb to untreatable cerebral malignancy.

Magnetic resonance imaging

Magnetic resonance imaging (MRI) scanning involves computerized reconstruction of tissue volumes from the detection of radiowaves emitted

from the changes in alignment of protons within the patient lying in an intense magnetic field. There are several techniques which may be used, including the T1 and T2 relaxation times and the proton density, which all show different tissue quantification and are particularly useful for certain features. In general, however, the resolution of the MRI scan is not superior to the CT scan and it is no better at confirming the presence or absence of invasion than CT scanning. However, there are two areas where MRI scanning is routinely used in the radiological assessment of the thorax. These are both areas adjacent to bones, where CT resolution is difficult due to streak artefact from the slice reconstruction techniques used, and include assessment of Pancoast-type tumors involving the apex of the chest and brachial plexus and the neurogenic tumors of the paravertebral sulcus to assess whether there is invasion of the neural foramina or extension into the vertebral canal.

Positron emission tomography

Positron emission tomography (PET) scanning has revolutionized the imaging of the body by allowing localized measurement of tissue metabolic activity. Briefly, a radioactive isotope of fluorine is bound to deoxyglucose and administered intravenously. It is taken up as a glucose analog by metabolically active cells and only partially metabolized. It then remains stuck cytoplasmically until alternative pathways can break it down. Meanwhile the unstable fluorine isotope decays by the emission of a positron, which annihilates a nearby electron on collision with the release of two identical energy photons in opposite directions and identical constant wavelength. These are detected by a special detector array, in the newest scanners located within a CT scanner for co-localization. It is important to be aware that PET may have false negatives when metastases are present in tissues with a high underlying metabolic rate, especially brain. For this reason, scans are very rarely shown above the neck.

Table 5.3 Risks of thoracic procedures.

Procedure	Death	Bleeding	Others
Mediastinoscopy	0.3%	Sternotomy 0.1%	Pneumothorax 1% Recurrent nerve injury 1%
VAT pleural procedure	1.5%	1%	Prolonged airleak 1%
Lobectomy	2–4%	1%	Prolonged airleak 5–10%
Pneumonectomy	5–10%	1%	Bronchopleural fistula 2–10%
Lung volume reduction	5–10%	1%	Prolonged airleak 40%

Results of PET scanning are more accurate than CT scanning for nodal and metastatic assessment. A PET negative mediastinal scan is specific enough at 90–95% to rule out nodal disease in most lung cancer patients although it cannot detect micrometastases. Likewise, a positive nodal scan is indicative of increased uptake although unable to differentiate between infection or tumor metastases. Therefore, PET positive mediastinal nodes would still need further surgical assessment prior to lung resection. Despite this potential limitation, it is felt that PET scanning reduces the need for preliminary mediastinoscopy in many patients if negative and prevents a futile thoracotomy in 1 in 16 patients assessed as node-negative on CT assessment. PET scanning is proving very useful in the workup of difficult cases such as those with borderline operability, multiple metastases or extended resection, where the likelihood of disease outside the chest is higher, allowing reduction in the chance of futile surgical resection.

Operability

As discussed above, operability is generally taken to mean the ability of the patient to recover from the morbidity of the surgery. Thoracic surgical procedures range from small such as mediastinoscopy, to pneumonectomy or trauma thoracotomy which may have much higher risks. Approximate risks of death and significant complications are shown in Table 5.3.

As well as an understanding of the risks of death or major morbidity with the procedure, it is important to understand that a thoracotomy generally reduces the effective FEV₁ by around 10–15% due to pain, splinting and the mechanical effects of the incision on the chest wall musculature and ribs. The expected benefit of the surgery is also important in this context.

British Thoracic Society – Guidelines

National guidelines exist in many countries regarding the management of patients considered for lung cancer surgery. There are differences between them based mainly on the availability of advanced techniques for investigation, treatment and the precise nature of primary care, referral practice and funding. The British Thoracic Society (BTS) guidelines, published in 2001, underlie the operative management of lung cancer in the UK and a brief outline follows.

- **Age.** Risk of mortality and morbidity increases with increasing age, although acceptable rates of survival are seen in carefully selected older patients. Increased age is a significant factor increasing mortality for pneumonectomy or chest wall resection and should be considered a relative contraindication.
- **Pre-operative lung function.** Evidence from many published series shows that a pre-operative FEV₁ of over 2 liters is an indicator of low risk for pneumonectomy and

Table 5.4 Anatomical segments.

Right lung		Left lung	
Upper	3	Upper	4 (proper)
Middle	2		(lingula) 2
Lower	5	Lower	5
Total	10	Total	9

pre-operative FEV₁ of over 1.5 liters is an indicator of low risk for lobectomy; in these cases, BTS guidelines do not recommend further respiratory functional testing in the absence of breathlessness.

- **Post-operative predicted lung function**, based on extrapolation from pre-operative spirometry and gas transfer, is an important predictor of surgical risk. Approximate lung function may be predicted using the normal anatomical 19 segments and the estimated number to be resected, using the following formula:

$$\text{ppoFEV}_1 = \text{FEV}_1 * (19 - R)/19$$

where R is the proposed number of segments in the resection and ppoFEV₁ is the post-operative predicted FEV₁.

The same formula may be used to calculate the predicted gas transfer value.

The numbers of segments in each lobe are listed in Table 5.4. Evidence from many published series shows that a post-operative predicted FEV₁ > 40% and post-operative predicted T_LCO > 40% of normally predicted with no desaturation is sufficient for acceptable operative risk. An absolute lower limit of FEV₁ of 800 ml is recommended. If there is significant pathology affecting the lung to be resected, it may be that the above formula will underestimate the predicted post-operative lung function. In the setting of segmental or lobar collapse of the affected lung, the number of affected segments should be subtracted from

the numerator and the denominator of the above equation. If the results are borderline or there is doubt about the function following resection, the differential perfusion from radionuclide studies is invaluable. If patients have both post-operative predicted FEV₁ and T_LCO below 40%, the patients are high-risk and should be considered for radiotherapy, chemotherapy or very limited (e.g. wedge) resection. All other patients should be considered intermediate risk and should undergo exercise testing with shuttle runs or VO₂max. Fewer than 25 shuttles or below 15 ml/kg per min oxygen uptake are indicators of very high surgical risk. Above these values, risks may be acceptable.

- **Cardiac assessment.** All patients undergoing lung resection should have resting 12-lead electrocardiography and echocardiography in the presence of cardiac murmur or known valvular or structural heart disease. No patient should undergo surgery within 6 weeks of myocardial infarction (MI) and all patients within 6 months of MI should have a cardiology opinion. Previous coronary artery bypass grafts should not limit surgery if the patient has adequate functional capacity and is asymptomatic. Patients with a single risk factor for coronary disease but no symptoms are at low risk and do not need further investigation. Those with previous disease but adequate functional capacity, e.g. able to climb a single flight of stairs comfortably likewise do not need further investigation. Those at high risk should undergo exercise or other stress testing and consideration of percutaneous or surgical intervention prior to lung surgery as appropriate.
- **Assessment of other organ systems.** Patients should have adequate nutritional status: weight more than 10% below ideal or low serum

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albumen levels should be taken as indicators of increased or high surgical risk. Liver function testing should also be performed to highlight evidence of dysfunction or indication of metastatic disease.

- **Renal function.** Renal impairment is an indicator of increased risk for major surgery but should not necessarily contraindicate potentially curative surgery. Renal dialysis should prompt careful consideration of the expected survival with and without malignancy but likewise should not contraindicate lower risk potentially curative procedures.
- **Brain.** In patients of intermediate or higher risks with surgery, extended staging may include head CT scanning to exclude metastasis (as mentioned in the imaging section). Careful consideration of those with progressive cerebral disease such as dementia, Parkinson's or ischemic strokes should be made as both increased peri-operative risk and reduced long-term survival will result.
- **Blood.** Blood testing should exclude the presence of untreated anemia. Polycythemia is common in heavy smokers and those with

lung disease and is an increased risk factor for thrombo-embolic complications peri-operatively. Presence of myelodysplastic syndromes has similar effects along with an increased risk of hemorrhage and is associated with poorer long-term survival although some patients with low-grade forms may have a reasonable prognosis. The presence of hyponatremia suggests the syndrome of inappropriate ADH secretion (SIADH).

FURTHER READING

- British Thoracic Society and Society of Cardiothoracic Surgeons of Great Britain and Ireland Working Party. Guidelines on the selection of patients with lung cancer for surgery. *Thorax* 2001; 56: 89–108.
- Mountain CF, Libstitz HI, Hermes KE. *A Handbook for Staging, Imaging and Lymph Node Classification*. Charles P. Young, 2003.
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Lung isolation

DAVID PLACE

With the developments in thoracoscopic and minimally invasive cardiac surgery, the absolute indications for lung isolation have expanded. These developments have coincided with the availability of new bronchial blocker devices. These devices may have advantages over the traditional double lumen endobronchial tube (DLT) in specific circumstances.

This chapter aims to give an overview of the indications and techniques available for lung isolation in adult patients, using devices currently available in the UK.

History

Before the development of the Carlens left-sided DLT in 1950, a tube that was designed for differential lung spirometry, anesthetists used either single lumen tubes or bronchial blockers to produce lung isolation. The major developments in devices are listed in Table 6.1.

Current single-use plastic DLTs are based on the Robertshaw design; a red rubber tube, oval in cross-section with two D-shaped lumens placed laterally to each other to maximize internal luminal size and reduce gas flow resistance. The right-sided version has a right upper lobe ventilation slot in the bronchial portion of the tube. The bronchial and tracheal cuffs of plastic DLTs have high volume,

low pressure cuffs, in order to reduce the risk of mucosal trauma.

In the 1980s, the Univent tube, a combined single lumen tube with incorporated bronchial blocker, became available. This was further revised recently to become the Univent torque control blocker (TCB) (Vitaaid Ltd.), the changes enabling improved control of the bronchial blocker.

Today, the availability of the fiberoptic bronchoscope (FOB) has encouraged the use and development of bronchial blockers which would previously have required rigid bronchoscopy for placement. Over the past decade several independent bronchial blockers have been developed which can be positioned under FOB guidance, and used in conjunction with standard single lumen tracheal tubes. Other balloon-tipped catheters, such as Fogarty embolectomy catheters, have also been used as bronchial blockers.

Indications for lung isolation

Two broad categories encompass the main reasons to perform lung isolation:

1. To prevent contamination of normal lung with blood or pus.
2. To control the distribution of ventilation. This can be to facilitate surgical exposure or to enable ventilation in cases of airway disruption,

Table 6.1 Major developments in lung isolation devices.

Date	Name	Device type
1932	Gale & Waters	Endobronchial tube with carinal cuff
1936	Magill	Right and left endobronchial tubes with short cuffs
1936	Magill	Red rubber endobronchial blocker
1950	Carlens	Left-sided DLT, carinal hook. Oval in horizontal plane
1955	Macintosh & Leatherdale	Left endobronchial tube, tracheal and bronchial cuffs, tracheal suction channel
1955	Macintosh & Leatherdale	Cuffed tracheal tube with left bronchus blocker and bronchial suction channel, Curved to enable blind insertion
1957	Green & Gordon	Right endobronchial tube, carinal hook and upper lobe ventilation slot. Bronchial and tracheal cuffs
1960	Bryce-Smith & Salt (left-sided version 1959 Bryce-Smith)	Right DLT, oval in anterior-posterior plane, slit in endobronchial cuff for upper lobe ventilation
1962	Robertshaw	Right and left sided DLTs Oval cross-section in lateral plane, no carinal hooks, right upper lobe ventilation slot. Increased inner luminal diameters
1982	Univent tube (Vitaid Ltd.)	Univent tube; combined single lumen tube and retractable bronchial blocker
1999	Arndt blocker (Cook)	Wire guided bronchial blocker. High volume low pressure cuff. Coupled with fiberoptic bronchoscope via a monofilament loop during insertion
2003	Cohen Flexitip Endobronchial blocker (Cook)	Flexitip bronchial blocker. Tip flexed under control from wheel at hub. Positioned under FOB guidance. Used with standard tracheal tube

(Modified from Pappin JC. The current practice of endobronchial intubation. *Anaesth* 1979; **34**: 57-64.)

Table 6.2 Absolute indications for lung isolation.

- A** To prevent contamination of normal lung
Major pulmonary hemorrhage
Bronchopleural fistula with empyema
Lung abscess
Whole lung lavage for alveolar proteinosis (rare)
- B** To control the distribution of ventilation
Bronchopleural fistula
Traumatic airway disruption
Giant bullae at risk of rupture
- C** Allow surgical access
Thoracoscopic surgery
Minimally invasive cardiac surgery

airway fistulae, giant bullae or other severe unilateral lung disease. Table 6.2 lists examples of absolute indications for lung isolation.

In current clinical practice, most surgeons would expect lung isolation to facilitate surgical access for the majority of procedures requiring thoracotomy. It is an absolute requirement for thoracoscopic surgery and many procedures are now performed using video-assisted thoracoscopy (VATS).

Other procedures that usually require lung isolation include esophageal surgery, surgery of descending thoracic aorta, spinal surgery via thoracotomy and chest wall resection.

Methods and choice of technique

The DLT, bronchial blocker and an appropriately sized single lumen tracheal tube can all produce lung isolation, if placed in a main stem bronchus. No one technique has overarching advantages for all situations, but each lends itself to certain clinical

applications. The choice will be influenced by the anesthetist's experience, certain patient factors such as abnormal airway anatomy, and surgical requirements.

The double lumen endobronchial tube

Double lumen endobronchial tubes in common use are plastic, oval in cross-section and have an antero-posterior curve corresponding to the oropharyngeal curve and a right or left lateral



Figure 6.1 Mallinckrodt bronchocath double lumen endobronchial tube (right) (DLT).

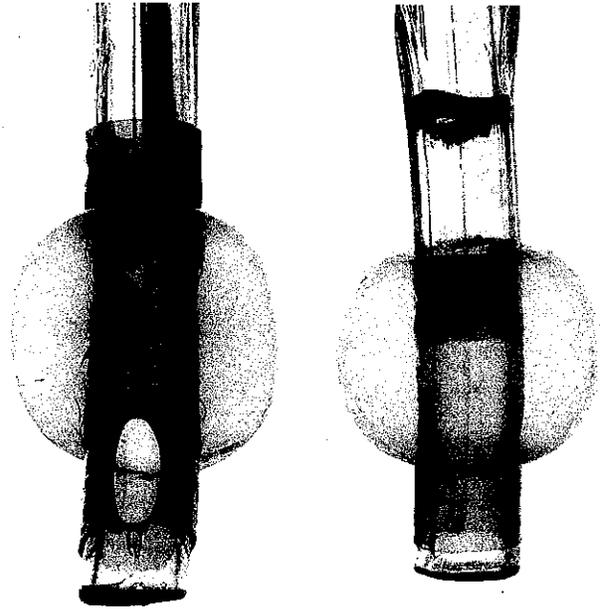


Figure 6.2 A comparison of the endobronchial components of right and left Mallinckrodt bronchocaths.

curve corresponding to tracheobronchial anatomy (Figure 6.1). One lumen opens in the trachea below the tracheal cuff and the other terminates at the tip of the endobronchial portion. On right-sided tubes there is an endobronchial ventilation slot to allow ventilation of the right upper lobe (RUL) (Figure 6.2). The arrangement of the cuff and slot varies depending on tube manufacturer. The endobronchial cuff is colored blue to assist with identification during bronchoscopy.

The main advantages of the DLT over other methods are:

1. Rapid inflation and deflation of either lung without repositioning.
2. Suction and fiberoptic inspection to either lung is immediately possible.

Due to their larger size, DLTs are more difficult to insert than single lumen tubes, particularly if laryngoscopy is difficult. Table 6.3 compares the internal and external dimensions of DLTs and single lumen tubes. They are unsuitable for prolonged weaning from ventilation due to higher airflow resistance and patient discomfort from the bulkier tube.

Table 6.3 A comparison of the approximate outer diameters (OD) of single lumen and double lumen tubes of different sizes (actual sizes vary between manufacturers).

Single lumen tube		Double lumen tube	
Inner diameter (mm)	OD (mm)	size (Ch)	OD (mm)
7	9.5	32	10.7
7.5	10.2	35	11.7
8	10.8	37	12.3
8.5	11.4	39	13
9	12.1	41	13.7

When using the double lumen tube, there are two important considerations:

1. Choice of tube side; right or left?
2. Which size of tube to use?

Right or left-sided tube?

The right main bronchus is much shorter than the left; 2 cm rather than approximately 5 cm on the left. The position of the right upper lobe take off is also variable and can even arise from the trachea. For these reasons it may be difficult, or impossible, to simultaneously isolate the right lung and ventilate the right upper lobe using a right-sided DLT. Even with correct initial placement, minimal movement of the tube may result in failure to ventilate the right upper lobe. This can result in hypoxia during one-lung ventilation and may predispose to post-operative right upper lobe collapse.

As the left main bronchus is longer, it does not present the same problems. Left endobronchial intubation usually provides a safe and stable tube position, and is the preferred technique, unless there is an absolute indication for a right DLT (Table 6.4). The greater angulation of the left main bronchus may occasionally create technical difficulties with endobronchial intubation.

Tube sizing

Plastic DLTs are available in Charriere (Ch) gauge (or French gauge (F)) sizes 26, 28, 32, 35, 37, 39 and

Table 6.4 The indications for a right-sided double lumen endobronchial tube.

- Left pneumonectomy*
- Left lung transplantation
- Intraluminal tumor or stent in left main bronchus
- External compression of left main bronchus
- Left tracheobronchial disruption
- Acute angulation of left main bronchus

* Left-sided tube can be used with surgical cooperation.

41 although not all manufacturers produce their tubes in all of these sizes. Most anesthetists choose the size of tube based on the patient's height and sex (Table 6.5), although a more accurate approach may be to take measurements of tracheobronchial dimensions from scans and X-rays. A correctly sized tube should pass easily through the glottis and the endobronchial component should enter the bronchus without resistance. If the tube is undersized it may be advanced too far into the distal airways, resulting in obstruction of lobar bronchi and risk of barotrauma and volutrauma to ventilated segments.

It has been shown by Brodsky *et al.* that tracheal width, measured at the level of the clavicles on PA chest X-ray, could predict left DLT size (Table 6.6). This has not been successfully applied in all patient populations.

Table 6.5 A guide to left double lumen endobronchial tube (DLT) size based on patients height and sex.

Women		Men	
Height (m)	Left DLT size (Ch)	Height (m)	Left DLT size (Ch)
< 1.5	32	< 1.6	37
1.5–1.6	35	1.6–1.7	39
> 1.6	37	> 1.7	41

Table 6.6 Suggested left double lumen endobronchial tube (DLT) size according to tracheal width measured on PA chest X-ray at level of clavicles.

Measured tracheal width (mm)	Left DLT size (Ch)
= 18	41
= 16	39
= 15	37
= 14	35
= 12.5	32

(From Brodsky JB, Macario A, Mark JBD. Tracheal diameter predicts double-lumen tube size: a method for selecting left double-lumen tubes. *Anesth Analg* 1996; **82**: 861–4.)

Pre-operative chest X-rays and scans should be carefully examined to assess tracheobronchial anatomy. Distortion, angulation or compression of major airways may influence choice of lung isolation technique.

Insertion of the DLT

The tube is supplied with a wire stylet placed in the bronchial lumen to increase rigidity. This should be in place prior to insertion. Laryngoscopy is performed in the usual way and the tip of the tube is passed through the vocal cords with the endobronchial tip pointing anteriorly. After passage through the cords the wire stylet should be removed before further tube advancement. The tube is then advanced and rotated 90° either to the left or right

depending on which bronchus is to be intubated. Advancement of the tube is stopped on sensation of slight resistance. Alternatively, the tube can be inserted to a predetermined depth using the guide for left-sided tubes; 29 cm at the incisor teeth for a patient 170 cm tall, ± 1 cm for every ± 10 cm of height.

The FOB can also be used at the outset to position the tube by advancing the tube over the FOB into the appropriate bronchus. This may be particularly useful in patients of short stature where the trachea will be short, or where difficult positioning is predicted due to tracheobronchial anatomy.

The tracheal cuff is then inflated and ventilation is commenced through both lumens. Care should be taken with these initial inflations as it is possible to deliver the entire tidal volume to a single lobe if an undersized tube has passed beyond a bronchial division during blind insertion.

Checking DLT position

Traditionally tube position has been assessed using auscultation (Table 6.7), but with the advent of fiberoptic bronchoscopy, high rates of malposition were discovered despite satisfactory findings at auscultation. Blind placement of right-sided disposable plastic tubes has a high incidence of right upper lobe obstruction and so FOB assessment of right DLTs is now considered mandatory. An important landmark is the trifurcation of the RUL bronchus which should be visible using the FOB through the ventilation slot of a correctly positioned tube

Table 6.7 A method to check double lumen endobronchial tube position using chest auscultation.

1. Ventilate through both lumens whilst inflating tracheal cuff to abolish air leak, and ensure equal air entry in all lung fields
2. Clamp connector to tracheal lumen and open tracheal lumen to air
3. Ventilate through bronchial lumen whilst slowly inflating bronchial cuff until air leak from open tracheal lumen abolished*
4. Auscultate chest to ensure the intended lung is isolated
5. Clamp bronchial connector and open bronchial lumen to air
6. Ventilate through tracheal lumen only and auscultate to ensure air entry to non-intubated lung only

* Should only require small volume of air (< 3 ml) to seal endobronchial cuff with optimal tube size.

(Figure 6.3). The FOB should be passed through both lumens of the DLT to ensure that lobar bronchi are patent and that the position at the carina is correct. The proximal edge of the bronchial cuff should be just visible beyond the carina and not herniating above it.

The endobronchial cuff should be inflated with the minimal amount of air to produce a seal, in order to avoid high cuff pressures which may lead to mucosal damage. Tube position should be rechecked with any change in patient position. Moving from supine to lateral decubitus frequently results in movement of the tube, usually in a cephalad direction.

Complications of DLT insertion

Minor upper airway trauma, dental damage and failed intubation are potential complications occurring with the use of any device used to intubate the trachea. Malposition resulting in failure to produce lung isolation, airway obstruction, hypoxemia and

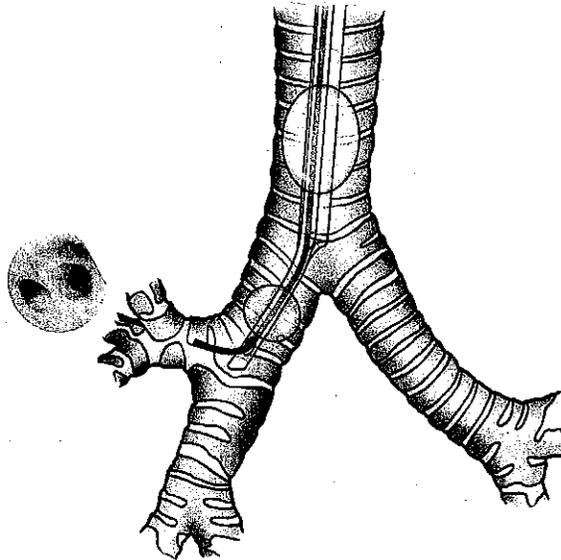


Figure 6.3 A diagram showing a correctly positioned right double lumen endobronchial tube (DLT). The fiberoptic bronchoscope view of the trifurcation of the right upper lobe is shown.

atelectasis can occur with the use of the DLT. Rarer complications include tracheal or bronchial rupture and even incorporation of the device into a surgical staple line. Care during insertion, removal of the wire stylet before tube advancement and caution with endobronchial cuff inflation may reduce the risk of serious complications.

Bronchial blockers

There are two main configurations of bronchial blockers.

1. The blocker is incorporated into a channel in the wall of a tracheal tube as in Univent TCB (Vitaid Ltd.) (Figures 6.4 and 6.5).
2. Independent catheter as in Arndt Blocker (Cook) (Figure 6.6), Cohen Flextip Endobronchial Blocker (Cook), Uniblocker (Vitaid Ltd.), Coopdech blocker (Smiths Medical).

All of the specifically designed bronchial blockers are balloon-tipped catheters with a central channel to allow suction, lung deflation and insufflation of

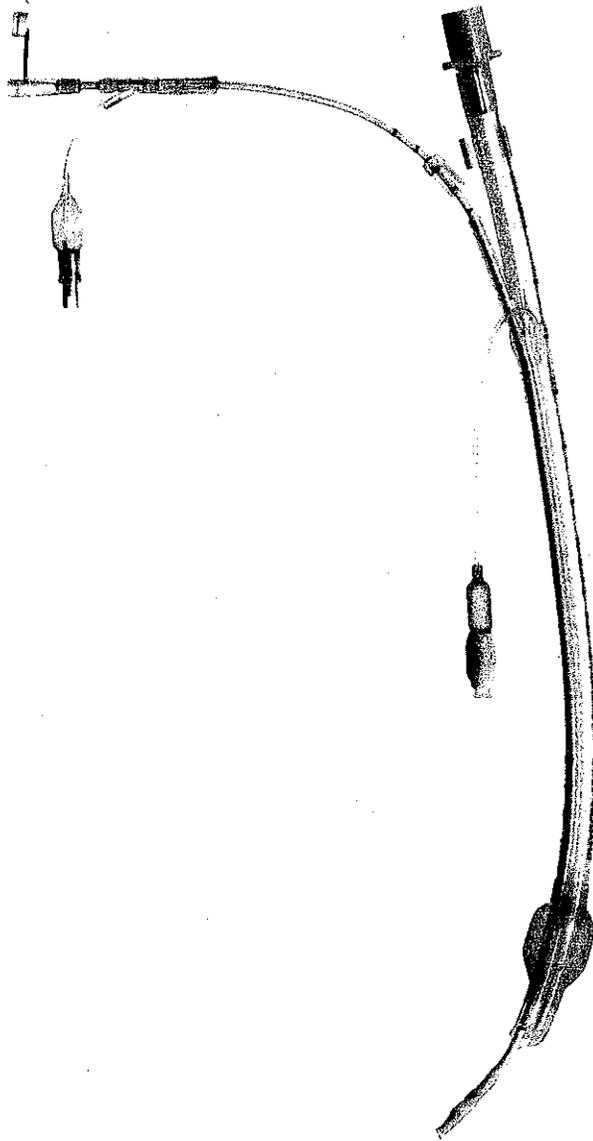


Figure 6.4 Univent TCB (Vitaid).

oxygen. They differ in their method of manipulation of the catheter tip during placement.

The Univent TCB is unique in that it combines a single lumen silicone tracheal tube with a retractable 2 mm diameter endobronchial blocker incorporated into the anterior wall of the tube. The cuff of the blocker is low pressure high volume and requires approximately 2–8 ml of air to seal, depending on the size of the airway to be occluded. An increasing range of sizes are available, including pediatric, but it is important to realize that the exter-

nal diameter is larger than standard single lumen tracheal tubes with the same internal diameter. The tube can be used as a standard single lumen tracheal tube until lung isolation is required, at which time the blocker is advanced into the required position in either main bronchus under vision via FOB. To assist with placement, the tracheal tube can be rotated towards either left or right main bronchus as appropriate.

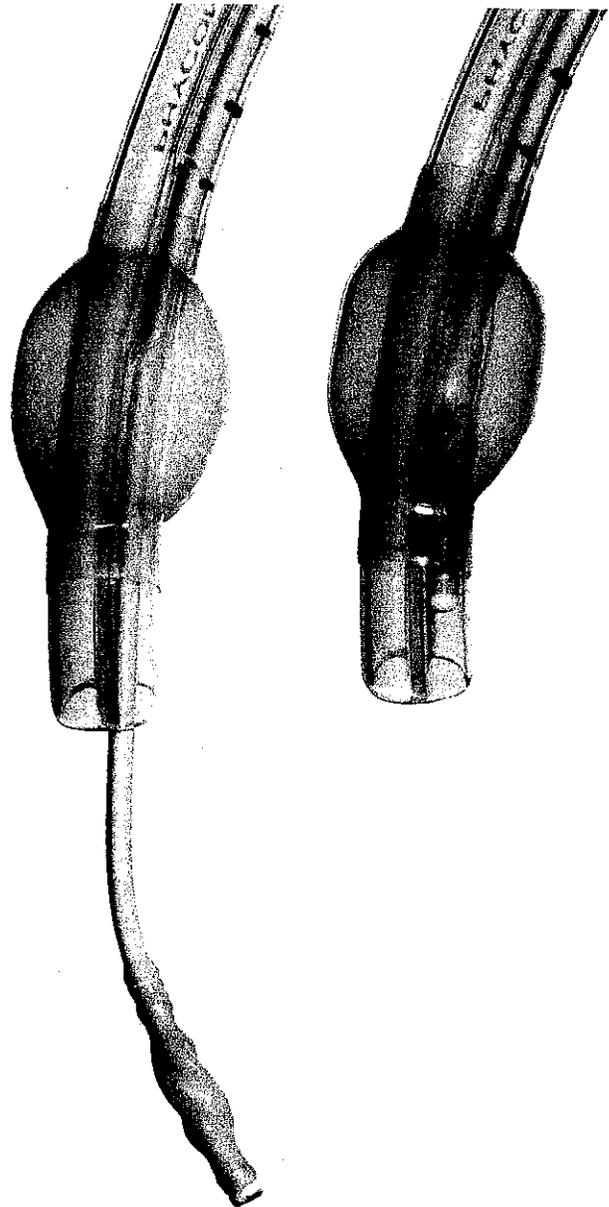


Figure 6.5 Tip of Univent TCB (Vitaid) showing tracheal cuff and blocker extended and retracted.

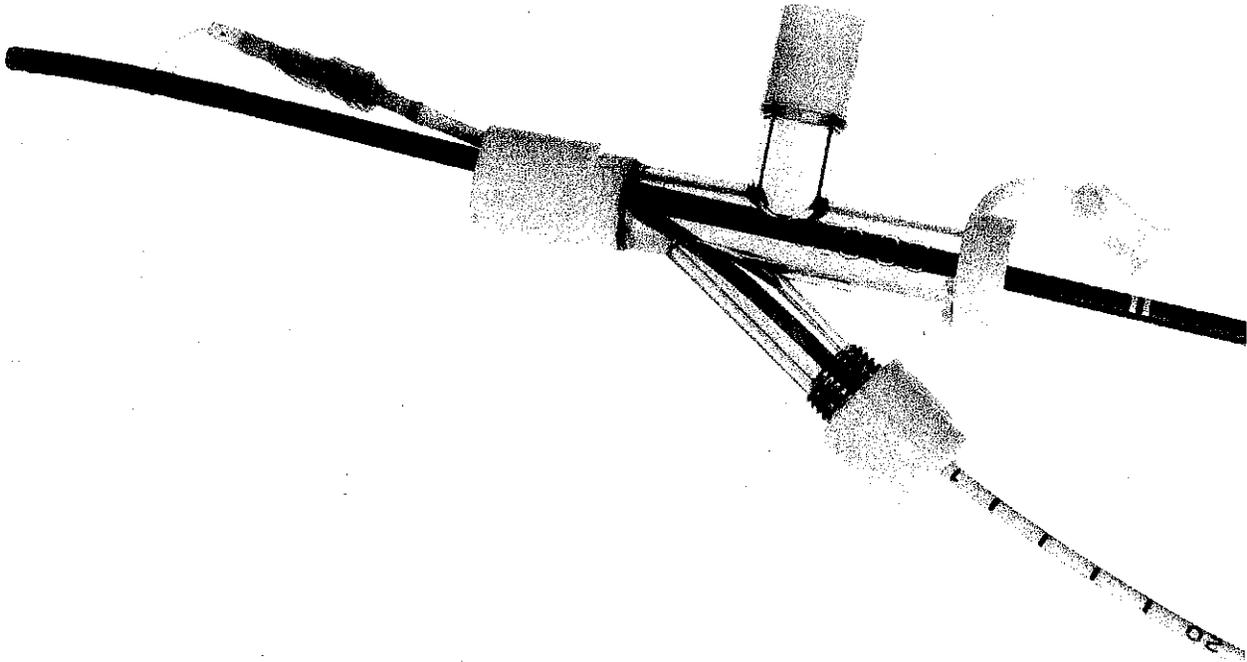


Figure 6.6 Arndt wire guided endobronchial blocker coupled to fiberoptic bronchoscope via the three-way multiport adaptor.

The independent bronchial blocker catheters can all be positioned coaxially down a standard tracheal tube under FOB guidance. A three-way, multiport adaptor allows insertion of blocker and bronchoscope without interrupting ventilation of the patient. The Arndt wire guided endobronchial blocker (Cook) is coupled to the FOB via a monofilament loop protruding from the distal end of the central channel, allowing the blocker to be guided directly into the required bronchus (Figure 6.6). The guide wire loop should be withdrawn for the central channel to allow egress of air, lung deflation and enable suction of secretions. Oxygen can be insufflated if required. The loop can be reinserted to allow repositioning of the blocker. The Cohen Flextip blocker is not directly coupled to the scope but it can be positioned under vision during FOB by a combination of catheter rotation and by flexing the tip which is controlled by a wheel at the proximal end of the catheter. This allows repositioning at any time during its use. The Uniblocker and Coopdech catheters both have a slightly flexed

tip which enables the blocker to be directed to either main bronchus with a combination of advancement and rotation of the catheter under vision via FOB.

The Arndt blocker catheter is available in two sizes; 5F and 9F with corresponding minimum recommended tracheal tube sizes of 4.5 mm and 7.5 mm respectively. A pediatric bronchoscope is required and the FOB and catheter should be lubricated prior to insertion to facilitate passage through the tracheal tube.

The Cohen Flextip catheter is currently available in size 9F only.

Complications associated with the use of bronchial blockers

Complications with the use of bronchial blockers include malposition and displacement resulting in life-threatening airway obstruction and hypoxia.

Failure to clear secretions through the small central channel of a bronchial blocker resulting in

Table 6.8 Comparison of lung isolation techniques.

Double lumen tubes	Independent bronchial blockers	Univent torque control blocker (TCB)
<p>Advantages</p> <p>Rapid inflation and deflation of either lung</p> <p>FOB inspection and suction to either lung possible</p> <p>Stable position (Left)</p> <p>Disadvantages</p> <p>Difficult to place in difficult airway</p> <p>Tube change required if patient already ventilated</p> <p>Unsuitable for prolonged post-operative ventilation</p>	<p>Advantages</p> <p>Difficult airway (can be used with single lumen tracheal tube)</p> <p>Patients already intubated</p> <p>Tube change not required for post-operative ventilation</p> <p>Lobar collapse possible</p> <p>Disadvantages</p> <p>FOB required for placement</p> <p>Slow lung deflation</p> <p>Small channel for suction</p> <p>Repositioning required for sequential lung collapse</p>	<p>Advantages</p> <p>Difficult airway (although bulkier than equivalent single lumen tracheal tube)</p> <p>Tube change not required for post-operative ventilation</p> <p>Lobar collapse possible</p> <p>Disadvantages</p> <p>Tube change required if patient already ventilated</p> <p>FOB required for blocker placement</p> <p>Small suction channel</p> <p>Blocker needs repositioning for sequential lung collapse</p>

soiling of normal lung after cuff deflation has been reported.

Which device to use? (Table 6.8)

Studies have shown that the DLT, independent bronchial blocker and Univent tube can all produce lung isolation and satisfactory operating conditions. In one study by Campos *et al.* the incidence of failure to position the device correctly by infrequent thoracic anesthetists was similar for all three methods. Lung deflation tends to be slower with BB devices whereas the DLT gives unrestricted access to both lungs for suction, bronchoscopy and insufflation.

However, BB devices have a role in the management of patients requiring lung isolation who are already intubated or who require post-operative ventilation, where exchanging tubes may be hazardous. They may also be specifically indicated where lobar blockade (Figure 6.7) is required. This may be required in patients who have undergone previous lung resection and are undergoing further thoracic surgery.

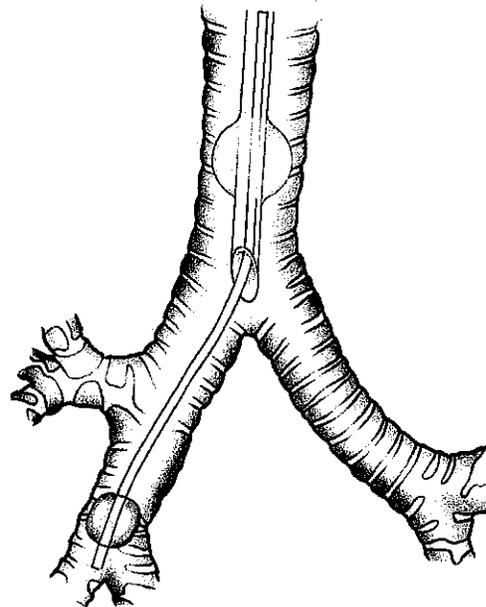


Figure 6.7 A diagram showing a bronchial blocker placed in the right bronchus intermedius enabling blockade of just the middle and lower lobes.

Lung separation and the difficult airway

Because of the size and shape of DLTs, orotracheal intubation can be difficult in situations where

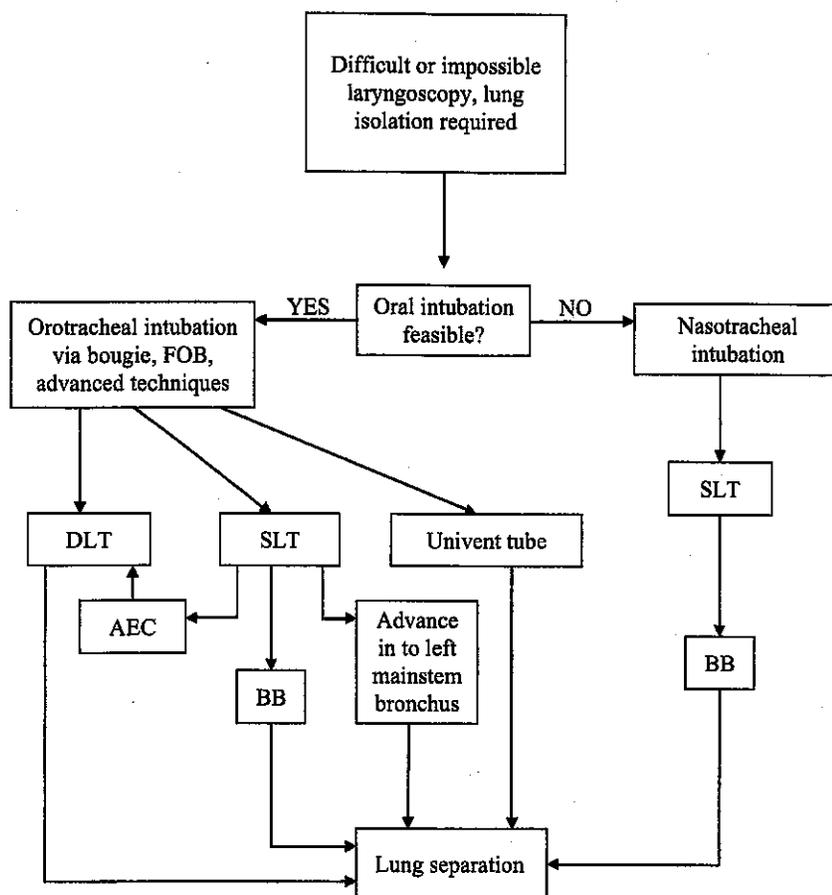


Figure 6.8 Methods of lung isolation in the patient with difficult laryngoscopy. AEC, airway exchange catheter; BB, bronchial blocker; SLT, single lumen tube; DLT, double lumen tube; FOB, fiberoptic bronchoscope.

intubation with a single lumen tube can be performed with ease. This problem is greatly amplified in patients with difficult laryngoscopy. Awake fiberoptic intubation with a DLT has been reported, but it is likely to be technically challenging. An alternative is to secure the airway with a single lumen tube and then proceed with bronchial blockade or change to a DLT using an airway exchange catheter. Figure 6.8 outlines some of the possibilities for airway management in a patient with difficult laryngoscopy. Where airway exchange catheters are being employed it is essential to ensure the free passage of the catheter inside the bronchial lumen of the DLT before proceeding.

Alternative laryngoscopes have been employed with success in patients with difficult laryngoscopy and the surgeon may be able to pass a bougie or

airway exchange catheter into the trachea during rigid bronchoscopy.

Patients with tracheostomy

Single lumen tracheal tubes, standard DLTs and bronchial blockers have all been used to provide lung isolation in patients with tracheostomy. Rusch manufacture right- and left-sided DLTs for use through a tracheostomy. These tubes are shorter and appropriately curved compared with standard DLTs and are appropriate for longer-term ventilation where lung isolation is required. The choice of other techniques will be governed by the size of the tracheostomy stoma and the reason for lung isolation. The availability of bronchial blockers has simplified the management of these patients.

Key points

- The left-sided DLT remains the device of choice for the majority of procedures requiring lung isolation in adult patients.
- Familiarity with the bronchoscopic appearance of tracheobronchial anatomy is essential for the safe and effective placement of devices for lung isolation.
- The position of the right upper lobe bronchus can prevent correct placement of the right-sided DLT and prevent complete right lung isolation by a single bronchial blocker.
- Bronchial blockers have a key role in providing lung isolation in patients with a difficult airway, those requiring post-operative ventilation and those already intubated and receiving critical care.
- The free passage of bougies, airway exchange catheters, bronchial blockers and bronchoscopes through the intended tracheal tube should be established before proceeding with clinical intervention.

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Management of one-lung ventilation

LEENA PARDESHI AND IAN CONACHER

Physiologically, on the institution of one-lung ventilation (OLV), the patient, by continuing to perfuse a non-ventilated lung, is positioned on the steep part of the oxygen dissociation curve: what has been described as analogous to *in the mountaineer "death zone."* Unlike the latter the inhalation of high concentrations of oxygen may not avert disaster. One-lung ventilation is never safe. It is this simple fact that makes attention to detail vital. The double lumen endobronchial tube (DLT) sited "too far," or the inadequately ventilated dependent lung adds the few critical degrees to the percentage of the cardiac output that is desaturated to take the situation beyond one salvageable with high inspired oxygen.

Notwithstanding interference of anesthetic agents with natural controls in matching of ventilation/perfusion, and of an open pneumothorax and lateral decubitus position compromising pulmonary and cardiovascular physiology, in practice, all problems of gas exchange should be seen as reflecting the conduct of OLV, and correctable by safe anesthetic or surgical manoeuvre.

Fundamentals of fractional inspired oxygen (FiO_2)

There is a paradox with using fractional inspired oxygen (FiO_2) 1.0. If necessary, then almost by definition there is a problem. Safe-enough oper-

ating conditions for pulmonary *resection* can be achieved with less, providing criteria for defining fitness have been met and by attention to detail in the conduct of OLV. This is not a statement of a cavalier attitude with the requirement for 100% oxygen or for its use in those already close to respiratory failure who may have to undergo OLV to facilitate a diagnostic or therapeutic process. Automatic use remains paramount as a therapeutic and first aid measure. Requirement is, however, to be read as a clinical signal of a treatable OLV problem (see Table 7.1).

Planning OLV

An expectant attitude is required, with some sense of those who may not cope with anything other than perfect lung separation and in whom a default position of recruiting the "surgeon's' lung" may be necessary. As a working guide, those with a predicted $\text{FEV}_1 > 40\%$ pre-operative values should pass through an OLV process without too much difficulty and few problems. Those with a predicted $\text{FEV}_1 < 40\%$ may well require support including post-operative ventilation. Planning, prediction and preparedness for improvisation are important: the former essential for guiding the process, and the latter for dealing with the unexpected. These define the level of monitoring required, early and direction of interventions, intra-operative conduct and

Table 7.1 First aid.

1. Increase (if not already done) the FiO_2 to 1.0
2. Go to manual IPPV. Request surgeon to stop operating
3. Aspirate for secretions etc. ventilated lung
4. If desaturation continues recruit surgeon's lung
5. Once patient safely oxygenated, define problem, adjust and resolve situation (see below for diagnoses and options)
6. Invite the surgeon to continue

Table 7.2 Operation groups as guide to level of care.

Group 1	Group 2
Pulmonary resection	Pneumonectomy
Lobectomy	Chest wall resection
Wedge resection	Lung volume reduction surgery
Open thoracotomy	Lung transplantation
Lung biopsy	Esophagectomy
Thorascopic	
Lung biopsy	
Pleurodesis	
Sympathectomy	
Thymectomy	

the post-operative management. A pleural operation in an ASA I patient, or a lobectomy in an ASA II patient, for instance, needs a level that is similar to that required by a patient undergoing a relatively minor abdominal operation (see Table 7.2, Group 1). Whereas, for the pneumonectomy, complicated lobectomy or esophagectomy, the level required is that of the patient undergoing cardiopulmonary bypass (Group 2). In the event of problems arising, e.g. hemorrhage, shift from lobectomy to pneumonectomy, prolonged OLV or lung handling (< 90 min) then Group 1 category should be assigned to the Group 2 level of intervention,

Table 7.3 A checklist of immediately available equipment.

Stethoscopes
 Standard endotracheal tubes
 Lung separators: both right- and left-sided*
 Rigid bronchoscope
 Fiberoptic bronchoscope
 Bougies and airway exchangers
 Regional analgesia equipment and drugs – paravertebral and epidural

* For women: 35, 37, and 39 Fr. For men: 37, 39 and 41 Fr. In general the largest tube that will fit should be tried. This decreases the airway resistance and makes fiberoptic examination easier.

care and management. Theoretically, it should not matter whether the surgical approach is via a lateral thoracotomy or thoracoscopy, but in practice when surgical access is restricted, the options for keeping the lung collapsed and out of the operating field are more limited.

Preparation for OLV in the anesthetic room

The institution of OLV can be problematic in approximately 20% of any case mix. A significant number will prove difficult at the lung separator insertion stage. The usual conditions of difficult intubation pertain, as well as some specific to the thoracic discipline and the pathologies encountered and exacerbated by the structure and bulk of some lung separators. The checklist in Table 7.3 is designed to cover the consistently and historically described difficulty posed in securing left lung isolation. The choice of lung separator has been discussed in Chapter 5.

Choice of anesthetic technique

There is now little place for nitrous oxide as a carrier gas in thoracic practice: air-oxygen mixes are

the routine. A dominant theme for 40 years has been the influence of general anesthetics on hypoxic pulmonary vasoconstriction (HPV) (see also Chapter 2). Although recognized as a potential problem, modern perspectives and evidence suggest that importance is over-rated and distracts from more likely reasons for changes in the shunt fraction. These are in practice more mechanical than biological: the effect of an open pneumothorax, collapse of lung and gravity on assumption of the decubitus position all in some measure favor flow to ventilated areas. It remains to be reinforced that the phenomenon of HPV is scientifically interesting but should never be invoked to justify poor technique or use of the hypothetical in the clinical arena. There is now little difference when modern volatile agents or total intravenous anesthesia are used, suggesting that the effects of techniques using nitrous oxide and/or potent cardiac depressants such as halothane may have been confounders in some of the early studies. As a general rule the observed shunt of OLV in clinical studies is 20–28%.

Ventilation

The use of OLV and the adoption of the lateral decubitus position results in specific physiological changes, such as the shifts in West Zones from vertical to horizontal, which are best countered by positive pressure ventilation (see Table 7.4). It is physical forces mainly that must be dealt with – all these effectively summing to compress the FRC. The open pneumothorax, weight of mediastinum, abdominal contents on adoption of the lateral decubitus position and the surgeon at work compress the dependent lung; and, all must be opposed through the narrow conduit of, often, the single lumen of a DLT.

Simplistic though it may appear, it needs to be reiterated that the minute volume for OLV is that of normal two-lung ventilation. In compensating for the dynamics of OLV, some advocate for the routine a tidal volume of 8–10 ml/kg with appropri-

Table 7.4 The lung that does not collapse.

Stop ventilation
Aspirate with suction tracheal and bronchial lumen
Allow the lung to deflate with gentle decompression
Put more air into bronchial cuff (simple leak)
Reventilate with reduced driving pressure
If still a problem
Reposition endobronchial tube (use flexible bronchoscope)
Insert a blocker into the tracheal lumen of DLT and inflate (a Foley catheter has been used in the past)
or
Accept the situation and alternate surgery with ventilation

ate respiration frequency to maintain normocapnia. Increasingly, there has been recognition that the operative ventilation conditions are incriminated in post-operative pulmonary dysfunction. Others now recommend a peak inflation pressure limitation (< 35 cm H₂O) and the use of smaller tidal volumes (4–6 ml/kg). However, airway pressure parameters need to account for the increase in resistance of the small size DLT, and driving pressure increased appropriately. In extreme cases such as lung volume reduction and lung transplantation normocapnia may have to be sacrificed, and a degree of permissive hypercapnia accepted (< 12 kPa).

Common problems

Oxygen desaturation (< 90%)

If hypoxia occurs once OLV has been satisfactorily established, the problems in Table 7.5 are sufficiently common in ranking or importance to be routinely checked for. Reference is to be made to Table 7.1 (first aid) while the cause of hypoxia is established and treated.

If these simple measures fail to correct hypoxia, consider recruiting the operative lung. The most common method is to employ a cPAP system, of which several are described, to the operative

Table 7.5 Physiological changes due to OLV.

Effects on hypoxic pulmonary vasoconstriction
 Lung volume alterations
 Atelectasis
 Gas trapping in distal airways
 Increased muscle activity
 Decreased outward chest wall recoil
 Increased elastic recoil of lung
 Increased thoracic blood volume
 Changes in airways resistance
 Depression of ventilatory control mechanism

lung. Although this is usually done as a reaction to hypoxia, there is argument for using it proactively to reduce the risks of post-operative acute lung injury (ALI) through achieving gas exchange by overworking and over-stressing the ventilated lung.

Other methods have been suggested to reduce the shunt, such as soft clamping pulmonary artery or insertion and inflation of pulmonary artery balloon catheters. In general these are not recommended; there is significant risk of long-lasting damage. The concept of producing a chemical pneumonectomy circulation using vasoactive drugs has been suggested. The current drugs (nitric oxide, almatrine) are toxic and indiscriminate and the idea has little to recommend it for routine use.

Lung that does not collapse

Some of these events are, in practice, pathological rather than clinical with the presence of pleural adhesions a common culprit for this source of irritation to our surgical colleagues. Nevertheless, the ritual of trying to improve surgical conditions by manipulating the OLV system has to be undertaken (see Table 7.4).

Re-inflation problems

Usually, the lung re-inflates once the tracheal lumen is ventilated. The most common reason

Table 7.6 Desaturation scenarios.

Separator moved. Tube "too far"; lobar bronchus blocked
 Secretions in the endotracheal tube or DLT
 Ventilated lung – FRC fall. Change driving pressure to compensate for small tracheal lumen
 Muscle relaxant worn off (usually a late phenomenon)
 Fall in cardiac output
 Dynamic hyperinflation (disconnect ventilator)
 Ipsilateral pneumothorax
 Surgical cause (pressure on bronchus, mediastinum, PA or heart)

for difficulty re-inflating a lung is the herniated bronchial cuff obstructing the contralateral bronchus. A graduated response is required. Ultimately it may be necessary to deflate the bronchial cuff and apply extra manual driving pressure. This may be an occasion that warrants the use of a fiberoptic bronchoscope to confirm or discount this problem.

Special considerations

Positive end-expiratory pressure (PEEP)

The reduction in FRC and closing volume that is further exacerbated by the effects of decubitus position potentially can lead to atelectasis and is influenced by PEEP. The clinical evidence is that it is not necessary as routine and indeed may be harmful: the effects on right ventricular function and cardiac output potentially exaggerated. In the event of oxygen desaturation those with clear evidence of background obstructive airways disease should be considered. However, it is a trial and error exercise.

The use of high frequency ventilation techniques has been attempted. There may be occasional circumstances such as the presence of bronchopleural fistulae, or in emphysema, but studies have shown no benefit to justify a routine use of complex equipment. There remains the enhanced risk for a

vibrating PEEP dynamic being generated with volutrauma and barotrauma of friable pulmonary tissue and circulation.

Dynamic hyperinflation

One of the main consequences of operating on patients for lung transplantation and lung volume reduction with background of emphysematous lung disease is the frequent occurrence of dynamic hyperinflation due to air trapping on institution of positive pressure ventilation. It is important to recognize the precursors of this rapidly fatal complication, which are the combination of an acute fall in oxygenation and blood pressure. The test effect of disconnecting the ventilator and rapid resolution is pathognomic. A protective form of ventilation should be instituted with peak inflation reduced to < 20 cm H₂O fall and a prolonged expiratory time of I:E ratio 1:>3. Permissive hypercapnia may be necessary and frequent periods of apnea with ventilator disconnection required to tide over the crisis.

Terminating OLV

It is axiomatic in this field that the ventilation system most appropriate and safe for a patient is spontaneous respiration. This is an early and prime goal. In these authors' practice, tracheal extubation is conducted with the patient still anesthetized and preceded by careful suction to ensure secretions and debris at the carina are not inhaled.

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SECTION 2 Operative Procedures

8. **Anesthetic Implications of Bronchoscopy**
Ian Conacher

10. **Anesthesia for Mediastinoscopy & Mediastinal Surgery**
David C. Smith

11. **Anesthesia for Video-assisted Thoracoscopic Surgery**
Mahesh Prabhu

Anesthetic implications of bronchoscopy

IAN CONACHER

Rigid bronchoscopic removal of inhaled foreign bodies has been a constant theme for a century. It will continue to be so. Rigid bronchoscopy (RB) was a necessary art of assessment of fitness for lung resection surgery and placing lung separator devices (LSD) (large foreign bodies!). Effective use of endobronchial blockers (e.g. Vernon–Thompson) and endobronchial tubes (e.g. Macintosh–Leatherdale, Brompton–Pallister) depended on expertise particularly for left lung isolation. The requirement was lessened as surgery shifted from that of tuberculosis to that of cancer and the role of the anesthetist shifted to peri-operative and post-operative conduct. But there remains a core of conditions where the essence of operational and therapeutic process is anesthesia for RB. And, RB technology has been – and continues to be – modified, for interventions such as lasers and tracheo-bronchial stents (Figure 8.1).

For the practice of the modern thoracic anesthetist, the differences between RB and fiberoptic bronchoscopic (FOB) instrumentation are brought in to focus by the new technologies. Principally, these relate to RB as conduits for tools versus FOB in diagnostics. The latter are the dominant device for insertion of bronchial blockers (e.g. Uni-vent, Arndt, Cohen) and elucidating lung separator problems. Rigid bronchoscopy, by enabling gas

exchange at the same time as the variety of instrument changes and maneuvers, has a new lease and gives a constancy that has facilitated the therapeutic advances for the endoscopic management of tracheal and endobronchial pathology. Those whose professional lives will be dominated by fiberoptic technology should be aware of the illusions that can be created by taking shortcuts with lessons learnt from the art of RB. Time spent observing latter day masters and acquiring a working skill will not be amiss for the 21st-century thoracic anesthetist. The particular value is in the importance of gaining knowledge of the true value of the “sniffing the morning air” position for sight and access to the larynx.

Anesthesia for bronchoscopy

Rigid bronchoscope insertion is very stimulating. A large, even massive pressor response is generated. Anesthesia must take account of this in a patient's fitness for process assessment and, if not ablate it, at least obtund it. The risks of hemodynamic, cardiac or cerebral complications are significant particularly in a case-mix likely to be preconditioned with allied comorbidities.

Non-anesthetists practise most FOB. Local anesthesia is applied topically when advancing the scope. Increasingly, in context, FOB are passed

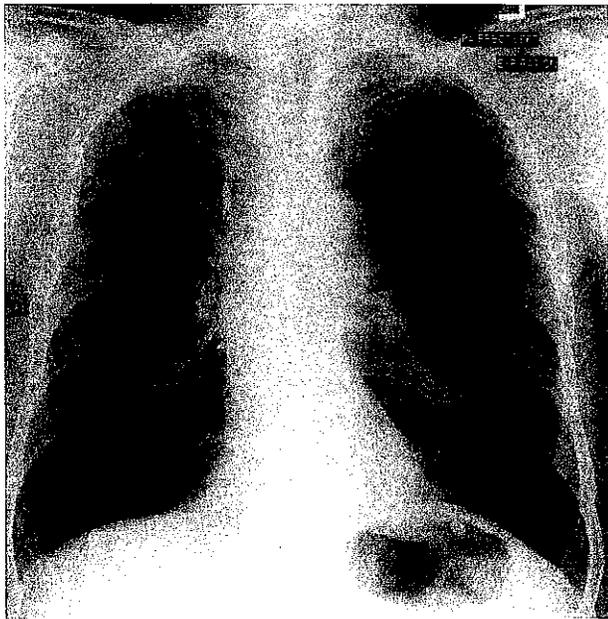


Figure 8.1 A chest X-ray showing a stent in position in the right main bronchus.

through RB under general anesthesia. Nevertheless, local and regional techniques for RB should not be discounted, particularly for the very sick in whom preserving of consciousness and self-ventilation is paramount. Organe, writing 60 years ago, describes the experience of RB under local anesthesia. So artfully can this be done that this eminent anesthetist was able to state: it was preferable to "going to the dentist."

For RB, general anesthesia is the norm. For short procedures a propofol and opioid supplemented (alfentanil, fentanyl) induction is a usual regimen, followed by a short-acting non-depolarizing agent such as mivacurium. There remains an occasional need (e.g. managing broncho-pleural fistulae) for suxamethonium. This should be preceded by precurarization to prevent fasciculations causing fouling of a lung that is vulnerable until a secure ventilation system is in place.

Rigid endoscopy, in general, had a bad record of awareness. By any measure this is no longer acceptable but it is less likely. Propofol is protective but until confidence in such advances as bispectral

index (BIS) and entropy monitors are established, it is wise to include benzodiazepine prophylaxis e.g. midazolam, either as premedication or on induction for the diagnostic or short process.

For more prolonged procedures, total intravenous anesthesia (TIVA) has proved remarkable. Propofol and remifentanyl by controlled infusion are the local standard. Pressor response ablation and hemodynamic stability are easily achieved for the periods of stimulation that result from protracted processes that are in the nature of foreign body removal, tracheal and bronchial laser resection and tracheo-bronchial stenting, despite often intense competition for the airway from therapists and anesthetist. Mivacurium as an adjunct for muscle relaxation is proving satisfactory and has the merit of not often requiring the use of reversal agents. There are mixed messages about the use of topical anesthesia as supplement. The author's view is that its use does not add any benefit and in the unfit may interfere with recovery (*vide infra*).

Positive pressure ventilation

Manufacturers of current equipment pay little attention to the needs of patient ventilation during operation of their devices. The concessions are small and at the operational end, employing one of three attachments which make positive pressure ventilation possible, but only just!

1. A Luer fitting for jet or Sanders' type.
2. A port for attachment of a jet or high frequency jet ventilator.
3. An attachment for an anesthetic system in conjunction with a cap that fits on the top of the bronchoscope to seal the system so that volatile agents also can be used in addition to continuous-flow oxygen or air. A clear window and fenestrated slide (to allow instrument insertion) sometimes are integral to the cap attachment. This set up is referred to as a ventilating bronchoscope.

Jet systems can be relatively uncontrolled; not uncommonly close to direct pipeline pressure. Overinflation of lungs, barotrauma and air trapping are potential dangers to be alert to. End-tidal CO₂ monitoring is difficult and all things withstanding generally is dispensed with.

In an emergency the suction port can be used for jet ventilation. If all else fails, a standard tracheal tube can be jammed into the top of an RB and the patient oxygenated from a reservoir bag by hand.

Emerging from the anesthetic

The termination of a procedure can be difficult: recovery of cough reflex and consciousness protracted and complicated. Sick patients are destabilized by the excess of pharmacology and surgical manipulation so that delicate and precarious physiological adaptations to their airway pathology may be slow to re-establish. Carbon dioxide retention may follow. The recovery end point is the cough. *"The cough and nothing but the cough"* in its entirety must be heard and seen to have been retrieved before the patient is safe – *"so help me cough"* is a good mantra in this endeavor! Coordinated deep inspiration, the closed glottis and forced expectorating expiration are the gold standard of criteria for transfer to recovery. Secretions, blood clot, tumor, laser debris and pus lurk in the depths ready to obstruct without warning. Reinsertion of an RB commonly (10%) is an urgent requirement; and a recovery period of IPPV an occasional one.

Rigid bronchoscopy for major events

Airway obstruction

Intrinsic and extrinsic lesions of the trachea can present as life-threatening emergencies. Some patients are in such dire straits that the energy to generate telltale stridor is not possible. First aid measures include airway humidification, steroids on an empirical basis, and inspiration of helium oxygen mixes until an RB can be inserted. There is an experience-base that a rapid sequence inser-

tion of the RB is least likely, in contrast to gas induction sequences, to precipitate total obstruction. Oxygen can be forced past the obstruction: in so doing it must be remembered that the risk of creating dynamic hyperinflation is significant. Nevertheless, the presence of an RB then allows for accurate assessment of the situation, the potential of a variety of treatment options and physical interventions, all within a relatively efficient gas-exchanging environment. The work up to tracheal resection and decisions for management of the airway during transection are best made on bronchoscopic findings.

Difficult intubation

Before the fiberoptic revolution, difficult airways were not so common but nevertheless occurred. Early photographs by pioneers of RB clearly illustrate that the "sniffing the morning air" position was naturally adopted by those whose operating tables were little more than wooden platforms with an assistant to support the occiput of the patient in the optimum position to avoid esophageal intubation. The straight blade laryngoscope design unconsciously followed on this experience. Thus one of the stratagems to deal with the difficult airway was to use an RB; for even if the trachea is not cannulated there is an easy route to place a bougie over which to railroad a tracheal tube. Oxygenation can be prevented from developing to hazardous levels by intermittent jet ventilation directed along the RB.

Hemoptysis

The erosion of a major vessel in the bronchial tree occasionally results in unstoppable hemoptysis. With much of the cardiac output coming up an RB, it is impossible to do anything to intervene. However, most bleeds are small and temporary but surprisingly difficult to define. Rigid bronchoscopy is necessary at some point in diagnosis or therapy. Acute hemorrhage (e.g. post biopsy) is best dealt with by tamponade with an adrenaline-soaked

pledget on an applicator through the RB, while gas exchange is assisted by a jet or Sanders' type device and directed to the non-afflicted lung. Airway and visibility in the area are maintained, enough for holding measures, such as double lumen tubes or bronchial blockers, to be applied.

Broncho-pleural fistula

Most are pin-hole leaks presenting post-operatively, through which the contents of a pneumonectomy-space leak to cause infection and aspiration in the remnant lung. The totally blown bronchial stump case, rare though it is, is a stereotype for an RB method of management. In this model, up to a liter of pus may be present in the space. The first aid measure is to attempt to drain, via an intercostal space, as much of the pneumonectomy space as possible but to be aware that this may not be sufficient to prevent loculated fluid entering the trachea.

1. Sit the patient up and tilt them to the side of the lesion.
2. Pre-oxygenate, precurarize (to prevent fasciculations), induce (etomidate) and then administer suxamethonium.
3. With the onset of apnea introduce the RB. Under direct vision observe the fistula, aspirate any contaminants with a large-bore suction attachment, place the tip of the bronchoscope in the non-affected bronchus and ventilate.
4. If lung isolation is required, pass an airway exchange catheter into the non-affected bronchus, remove the RB and "rail-road" an endobronchial or double-lumen tube into position.

Foreign bodies

Almost anything, small enough, can be inhaled. Toy whistles, bits of Christmas tree, Biro caps, chicken gristle are amongst the exotica seen by the author in addition to a diet of peanuts, pins and dentistry. A variety of instruments or contraptions may have to be employed. Extraction can be prolonged, and

fiddly but all are made feasible by an anesthetic technique centered on the rigid scope and TIVA.

Rigid bronchoscopy for the emergent technologies

Cryoprobes, lasers, and tracheo-bronchial stents have in succession evolved from the cumbersome to the convenient. The case-mix contains the most challenging of patients, frequently with major airway pathology, in respiratory failure, with a background of symptomatic comorbidities of cardio-respiratory disease. These situations are best done using both RB (to secure airway and ventilate) and FOB (to treat and apply therapy options). Although FOB access means that primary care with laser or stents can be done without access to RB, the latter indisputably remains necessary when problems arise.

First aid measures include positioning of the patient, humidification, non-invasive positive pressure ventilation and inhalation use of helium oxygen mix. Most will have been given steroids empirically. The use of local anesthesia supplementation is tempting. However, this does delay the re-establishment of the full and efficient cough reflex.

Lasers in the airway

The real attribute of the RB in the situation of operating lasers in the airway is the metal construction. This reduces the fire hazard, a risk never totally negated. The modern fiber delivery laser instruments are small enough to be inserted through a FOB, enabling a combined rigid/fiberoptic system that extends laser treatment options further into the airway and for more complex lesions. However, the plastic material of FOB is a hazard: it is ignitable by the laser fiber. It is thus mandatory that the RB is ventilated by an air (21% oxygen) driven system so that a sequence that potentially leads to fire or explosion is not initiated.

Box 8.1 Learning points

1. In thoracic anesthetic practice rigid and fiberoptic bronchoscopy are complementary, not alternatives.
2. There is significant pressor response to the presence of a bronchoscope.
3. There is a risk of awareness.
4. Jet ventilation systems are efficient but there are options.
5. Problems in thoracic practice are more easily solved when viewed as situations of anesthesia and ventilation for rigid bronchoscopy.
6. Fire in the airway is always a risk when lasers are used.
7. Fiberoptic bronchoscopes can delude and create inappropriate mindsets.
8. Once the airway is secure, make an opportunity to experience esophageal intubation with a flexible fiberoptic bronchoscope.

Stents

The advent of self-expanding devices has considerably eased the burden of sharing access to the airway with surgeons or physicians. The latter can make adjustments with FOB or rigid end viewing fiberoptic scopes. A few patients still require Montgomery or solid wall stents, with RB of necessity both for insertion and maintenance of life during the procedure and periodic servicing.

Words about fiberoptics (Box 8.1)**LIMITATIONS**

The optics of the FOB are unrivalled enabling sight of subsegmental bronchi: the RB limited by bulk and poorer optics to major bronchi. However, diagnostically there are some features which are not emulated by FOB.

1. Depth of field. With FOB this is difficult. Illustrated by the need to get some sense surgically of clear margins at the point of bronchotomy – sometimes the difference between a lobectomy or a pneumonectomy.

2. The inoperable tumor, bronchogenic or esophageal, causes a characteristic rigidity that can be sensed with an RB.

AS A TOOL

An earlier generation commonly were faced with being unable to correct a misplaced tube or use an endobronchial tube without having to disengage it from a main bronchus: conditions of lung separation were difficult to re-establish. The FOB to diagnose and site problems and to be used as a railroad guide is a great boon and valuable aid.

WORDS OF WARNING

The illusion of a correctly sited lung separator such as a double lumen tube is easily created. A CEPOD assessment found that the left-sided tube incorrectly sited and not being detected as in the right main bronchus was probably contributory to several operative deaths. It was apparent that this misplacement could inspire a sense of correct siting which would be confirmed by FOB. A correct process of anticipation (10% of left-sided tubes will tend to enter the right main bronchus), observation and auscultation in sequence before use of the FOB helps ensure that this illusion does not get created in the minds of the anesthetist.

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Anesthesia for mediastinoscopy and mediastinal surgery

DAVID C. SMITH

The prognosis for patients with mediastinal pathology has improved significantly in recent years as a result of better understanding of the impact of chemotherapy and radiotherapy on malignant mediastinal tumors. However, diagnostic biopsy and subsequent tumor resection are still hazardous procedures. In contrast, although thymectomy is still a major undertaking in patients with myasthenia gravis, fewer patients now require prolonged post-operative respiratory support as a result of improved understanding of the disease.

Mediastinal anatomy

The mediastinum is the space in the center of the chest bounded by the plurae on either side, the sternum anteriorly and the thoracic vertebral column posteriorly (Figure 10.1). The upper boundary is the thoracic inlet, and the lower boundary is the diaphragm. It is divided into superior and inferior portions by a line joining the sternal angle to the fourth thoracic vertebra. The inferior portion is further divided into anterior, middle and posterior parts (Table 10.1).

Mediastinal pathology

The relative incidence of mediastinal masses is given in Table 10.2. Approximately 90% of lymph node masses in the mediastinum are metastatic.

The signs and symptoms of mediastinal pathology range from trivial to life-threatening, and include airway compression, superior vena cava syndrome, compression of the right heart and pulmonary arteries, and dysphagia from esophageal compression (Table 10.3). Venous obstruction is a particular problem, because the smaller veins expand to allow collateral flow, increasing the risk of bleeding during anesthetic or surgical instrumentation. Neural compression is also common, and may produce severe pain, vocal cord palsy (recurrent laryngeal nerve) or Horner's syndrome.

In order to establish a firm diagnosis in mediastinal disease it is important to obtain a tissue biopsy, because many of these lesions are malignant and the decision to use radiotherapy, chemotherapy or a combination of these, is highly tumor-dependent.

Assessment of patients for mediastinal surgery

Many mediastinal masses are asymptomatic, and are discovered during routine examination or chest radiography. Most lesions in asymptomatic patients are benign, whilst symptomatic lesions are frequently malignant, although large masses tend to produce more severe symptoms irrespective of the pathology. Patients may therefore present for mediastinal surgery in a range of conditions, but the

Table 10.1 Contents of the mediastinum.

Superior	Inferior
Aortic arch and branches	<i>Anterior</i>
Innominate vein	Lymph nodes
Superior vena cava	<i>Middle</i>
Trachea	Heart
Esophagus	Ascending aorta
Thoracic duct	Pulmonary vessels
Thymus	Superior vena cava
Recurrent laryngeal nerves	Phrenic and vagus nerves
Lymph nodes	Lymph nodes
	<i>Posterior</i>
Retrosternal extension of the thyroid	Main bronchi
	Esophagus
	Descending aorta
	Azygos veins
	Thoracic duct
	Lymph nodes

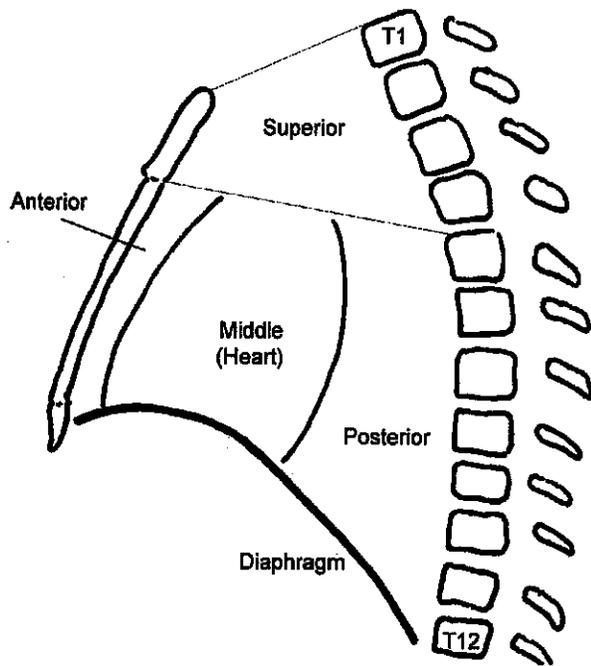


Figure 10.1 The anatomical divisions of the mediastinum.

Table 10.2 Incidence of mediastinal mass lesions.

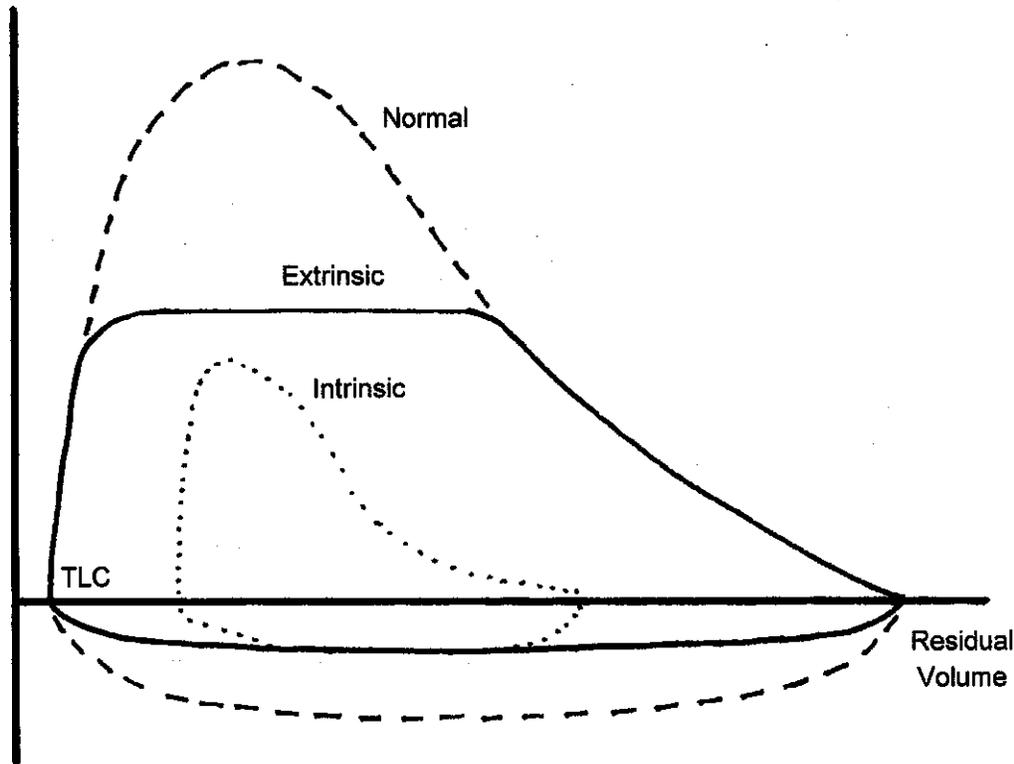
Neurogenic tumor	20%	Usually posterior
Thymoma	19%	30% have myasthenia
Cysts	18%	Pericardial 6%, Bronchogenic 6%
Lymphoma	13%	Usually anterior or middle mediastinum
Germ cell tumor	10%	
Mesenchymal tumor	6%	
Endocrine tumor	6%	
Primary carcinoma	5%	

Table 10.3 Symptoms in mediastinal disease.

Chest pain	30-40%
Dyspnea	22%
Cough	18-40%
Fever	13-24%
Weight loss	9-24%
SVC syndrome	8-16%
Myasthenia gravis	7%
Fatigue	6%
Dysphagia	4%
Night sweats	3%

principal problems for the anesthetist are difficulties with airway management, potentially complex surgery if tumors are being resected and the risk of major hemorrhage. Significant weight loss is unusual, and these patients are no more prone to endocrine or cardiovascular abnormalities than other patients. The surgical approach for diagnostic procedures is via cervical mediastinoscopy or anterior mediastinotomy, while for tumor resection the usual approaches are via median sternotomy or lateral thoracotomy.

Inhalation



Exhalation

Figure 10.2 Spirometry in airways obstruction. The effort-dependent part of the expiratory curve reaches a plateau in the case of extrinsic airways compression, whilst it has a concave appearance in intrinsic airways obstruction such as chronic obstructive pulmonary disease. Erect and supine spirometry enables identification of those patients in whom extrinsic compression of the airways only occurs on lying down. TLC, total lung capacity.

Careful evaluation of the airway is necessary during pre-operative assessment for surgery within the mediastinum. Chest X-ray, including thoracic inlet views when necessary, and thoracic computerized tomographs should be studied for evidence of airway compression or displacement. Occasionally it may be difficult to decide whether respiratory symptoms result from intrinsic or extrinsic airway disease. In these patients erect and supine spirometry may be helpful (Figure 10.2). Although radiotherapy and chemotherapy have improved the prognosis of mediastinal tumors and enabled tumor mass to be reduced before definitive surgery, these treatments often obscure the tissue diagnosis, which is crucial to further management of the disease. It

is possible to shield a portion of the tumor from the radiation beam, reducing symptoms while leaving a portion of the tumor unaffected by radiation, but this approach is uncommon and these patients often present for mediastinoscopy with significant respiratory or vascular obstruction.

The patient presenting for mediastinal surgery may also have systemic disease as a result of chemotherapy. Doxorubicin may produce a dose-related cardiomyopathy, especially if the dose exceeds 500 mg/m². Bleomycin may give rise to post-operative respiratory failure in association with high inspired oxygen concentrations, especially in patients over 70 years of age or who have received more than 400 units of bleomycin.

Myelodysplasia is a side-effect of most chemotherapeutic agents.

Anesthesia for cervical mediastinoscopy

Mediastinoscopy is usually performed to obtain a tissue diagnosis for mediastinal tumors, and to stage, or determine the operability of, other intrathoracic tumors. It was first described by Carlen in 1959, and provides information which would otherwise require thoracotomy.

The patient's head is placed on a head ring and the neck is fully extended. The upper half of the patient is tilted slightly head-up to reduce venous engorgement, although this maneuver slightly increases the risk of air embolism during the procedure. Access for the mediastinoscope is through a small incision above the suprasternal notch, followed by blunt dissection through the pretracheal fascia and behind the manubrium into the superior mediastinum between the trachea and the aortic arch.

Mediastinoscopy is a short procedure which is often performed immediately following a diagnostic bronchoscopy. It may be performed under local anesthesia, but general anesthesia is more commonly used. Although it is feasible to allow the patient to breathe spontaneously during mediastinoscopy, even via a laryngeal mask, tracheal intubation and positive pressure ventilation is a safer approach which also minimizes the risk of air embolism through open mediastinal veins. Endobronchial intubation is not indicated for mediastinoscopy, although an armored tracheal tube may be useful to prevent kinking of the tube during the procedure.

The choice of induction technique may be dictated by pre-existing respiratory compromise, and inhalation induction may be the safest approach in selected patients. The maintenance anesthetic agents and neuromuscular blocking drugs should

be chosen to enable a rapid return to consciousness, so that the patient may be safely extubated at the end of the procedure.

A large-bore intravenous cannula should be inserted, because of the risk of major hemorrhage, and if there is superior vena caval obstruction the cannula should be sited in a leg vein. Recommended minimal monitoring standards should be observed. The blood pressure cuff or arterial cannula, as appropriate, may best be sited on the left arm since compression of the right-sided head and neck vessels is common during mediastinoscopy. However, placement of an arterial cannula or pulse oximeter on the right arm may help to identify arterial compression, though this approach will often provide misleading pressure readings; palpation of the right radial artery during the procedure is an alternative approach.

Complications of mediastinoscopy

Death as a result of mediastinoscopy is unusual (around 0.1%), but the complication rate is 1.5–3% and complications may require rapid intervention (Table 10.4). The commonest complication is bleeding, and blood should be readily available. Although catastrophic bleeding is rare, its control may require emergency sternotomy or lateral thoracotomy. Effective fluid resuscitation may require vascular access in a leg vein if a large mediastinal

Table 10.4 Complications of mediastinoscopy.

Common	Unusual
Hemorrhage	Infection
Pneumothorax	Tumor implantation
Recurrent laryngeal nerve injury	Phrenic nerve injury
Arterial compression	Esophageal injury
Tracheal compression	Air embolism
Dysrhythmia	Chylothorax
	Stroke

vein is damaged. Pneumothorax is also common, but does not often require a chest drain. Damage to the recurrent laryngeal nerve may occur, especially on the left side, and this may be permanent in 50% of cases. It is impractical to assess vocal cord function routinely at the end of the procedure, but a high index of suspicion is required as bilateral recurrent nerve injury may result in airway obstruction. Arterial compression is also common, and may cause cerebral hypoperfusion and transient or permanent cerebral damage, especially in patients with cerebrovascular disease. Compression of the aortic arch may cause a reflex bradycardia.

Anesthesia for anterior mediastinotomy

Anterior mediastinotomy is performed via a small incision to the left of the sternum, through the second intercostal space or the bed of the second costal cartilage. This approach permits examination of anterior mediastinal structures, especially the thymus, which are inaccessible during cervical mediastinoscopy. It is also a convenient route for open lung biopsy. Although anterior mediastinotomy is an extrapleural procedure, pleural tears are common and a small pneumothorax is common. A post-operative chest X-ray is therefore advisable, but a chest drain is not often required. Anesthetic considerations are the same as for cervical mediastinoscopy.

Anesthesia for resection of mediastinal masses

Access to mediastinal structures may be via median sternotomy (sometimes limited hemisternotomy) or lateral thoracotomy. Anesthetic considerations are the same as those for mediastinoscopy, as mentioned above, except that a double-lumen endobronchial tube will be required for lateral thoracotomy. Surgery will often be complex and bloody. Steps should be taken to maintain normothermia,

and consideration should be given to forced air warming blankets and warming systems for intravenous fluids.

Myasthenia gravis

Myasthenia gravis is an autoimmune disease in which IgG auto-antibodies destroy the postsynaptic nicotinic acetylcholine receptors of the motor endplate, reducing the number of functional receptors by about 70–80%. The reduced number of receptors eliminates the margin of safety in neuromuscular transmission and results in rapid muscle fatigue, the clinical presentation depending on the muscle groups affected (Table 10.5). Antibodies to acetylcholine receptors are present in 90% of patients with myasthenia, but it is the antibody activity, rather than the titer in plasma, which predicts the severity of the disease. Diagnostically the muscle fatigue is reversed by a small dose of edrophonium (the Tensilon test). The incidence of myasthenia in the population is around 1:30 000, with a 3:2 female:male preponderance; the peak incidence in women is in the third decade, while in men it is in the fifth decade.

Myasthenia is commonly associated with other autoimmune disorders. A thymoma is common (around 50%) but patients without thymoma will usually have thymitis. Death from myasthenia gravis is now rare, as anticholinesterase therapy with pyridostigmine and immunosuppression with steroids or azathioprine have improved muscle

Table 10.5 Osserman classification of myasthenia gravis.

Group I	Ocular symptoms only
Group IIA	Mild generalized weakness
Group IIB	Moderate bulbar and skeletal symptoms
Group III	Acute severe disease (with respiratory compromise)
Group IV	Chronic severe disease

function for most patients. Thymectomy is often effective in producing an improvement in symptoms, though it is less beneficial to older patients and those with severe myasthenia. Thymomas may become malignant, or grow to a huge size, so resection is often recommended for these reasons alone.

The mainstay of chronic treatment for myasthenia is with a long-acting oral anticholinesterase, usually pyridostigmine. Although rare, an excessive dose of pyridostigmine may precipitate a "cholinergic crisis," with abdominal colic, diarrhea, miosis, lachrimation and excessive salivation. A "myasthenic crisis" results from withdrawal of anticholinesterase therapy, but may also be precipitated by emotion or stress, infection, pregnancy and menstruation. Myasthenic patients learn to manipulate their anticholinesterase medication, and may develop emotional or psychological dependence, becoming fearful if medical staff interfere with treatment. The patients are frequently worried about paralysis in the post-operative period.

Anesthesia for thymectomy in myasthenia

Thymectomy is a major undertaking in a myasthenic patient, and comprehensive pre-operative preparation of the patient, together with communication between surgeon and anesthetist, are important to success. Surgery is best performed while the disease is in remission, but early thymectomy is usually the treatment of choice and excessive delay may result in worsening of myasthenic symptoms. Optimization of anticholinesterase therapy improves muscle function, and plasmapheresis to reduce the concentration of circulating auto-antibodies may be useful in some cases, producing improvement in post-operative respiratory function. Psychological preparation of the patient is important, firstly because stress may precipitate a myasthenic crisis and secondly because the improvement in symptoms following thymectomy is not always immediate, and patients may be disappointed with the result in the early post-operative period.

Pre-operative assessment should include baseline respiratory function tests, and appropriate thoracic imaging as described above for mediastinoscopy. Patients with bulbar muscle involvement may have an impaired cough reflex, leading to tracheobronchial soiling which predisposes to chest infection. Thyroid function should be checked, as there may occasionally be associated thyroid abnormalities. There may also be some myocardial degenerative change associated with myasthenia, and consideration should be given to pre-operative echocardiography. Opinions vary as to the best way to manage anticholinesterase therapy, but on balance it is probably best to continue it on the day of surgery. Patients will be reluctant to omit their pyridostigmine altogether, and post-operative respiratory function will probably be better if it is continued. However, omitting the anticholinesterase may allow avoidance of neuromuscular blocking drugs during surgery.

Thymectomy is usually performed via a median sternotomy, although a limited upper hemisternotomy or a trans-cervical approach (similar to the incision for mediastinoscopy) may also be used. There is little difference in functional outcome between these approaches, although there is less disruption of chest mechanics with the trans-cervical approach, which may make it easier to avoid prolonged post-operative mechanical ventilation. Median sternotomy is easier for the surgeon and allows for more radical surgery, which is better for large masses or suspected thymoma.

An endobronchial tube is rarely required with any surgical approach for thymectomy. There is little to choose between anesthetic agents, provided the problems associated with myasthenic patients are appreciated. Following induction of anesthesia immediate assisted ventilation may be required, even before neuromuscular blocking drugs are given. In severe myasthenia neuromuscular blocking drugs may be avoided completely, as the muscle-relaxing effect of volatile anesthetics

is enhanced. Competitive neuromuscular blocking drugs are not contraindicated in myasthenic patients if used in small doses with adequate monitoring. Suxamethonium is best avoided, though it rarely causes problems, as myasthenic patients are resistant to it and a prolonged phase II block may develop (myasthenic patients do not fasciculate following depolarizing neuromuscular blocking drugs).

Median sternotomy is the least painful approach to major thoracic surgery, but adequate analgesia is vital for effective chest physiotherapy post-operatively. A thoracic epidural is effective in the early post-operative period, but concerns about respiratory depression from opioid analgesia in the presence of neuromuscular disease should not prevent adequate post-operative analgesia. Tracheostomy was routine when thymectomy was first introduced, but in modern practice extubation at the end of the procedure should be the aim. However, this is not always possible and around 50% of patients who have a trans-sternal thymectomy require prolonged mechanical ventilation. Scoring systems have been devised to predict the need for post-operative ventilation, but there is debate about the reliability of these and patients should be considered individually. Severity of disease (Osserman groups III and IV), low forced vital capacity (<15 ml/kg), surgery via median sternotomy, a history of respiratory failure secondary

to myasthenia, and pre-operative steroid therapy are all associated with prolonged post-operative ventilation.

Key points

- Patients with mediastinal pathology often have few symptoms at presentation.
- Reliable tissue diagnosis is the key to management of mediastinal masses.
- Mediastinoscopy is potentially a hazardous procedure.
- Effective multidisciplinary management and preparation reduces the risks of thymectomy in myasthenic patients.

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Anesthesia for video-assisted thoracoscopic surgery

MAHESH PRABHU

Thoracoscopic inspection of the pleura was first performed under local anesthesia in 1910 by Jacobaeus, a Swedish physician. The inability to illuminate the thoracic space and lack of adequate field of vision held back the development of the technique. The phenomenon of total internal reflection enabled light to be transmitted through a glass fiber and the development of flexible endoscopes. Until the late 1980s thoracoscopic surgery was limited largely to diagnostic techniques. Advances in optical systems, endoscopy equipment and video technology have contributed to increasing the range of video-assisted thoracoscopic surgery (VATS) from diagnostic procedures to more complex therapeutic procedures.

Principles of thoracoscopic surgery

- The minimal requirements for VATS include a rigid telescope, a light source with cable, a camera and an image processor. The optional devices include a slave monitor, a semi-flexible telescope and a video-recorder.
- VATS need higher light output power because blood in the operation field will absorb up to 50% of the light.
- The camera and endoscopic instruments are orientated to face the same way towards the target pathology "baseball diamond concept."

- The access sites should be placed at a sufficient distance from the target pathology to expose a panoramic view and provide room for manipulation.
- The thoracic cage is rigid and the sites of access are limited to the intercostal spaces.
- Single-lung anesthesia is necessary to deflate the lung. Once the lung has collapsed there is no need for insufflation or sealed ports.
- All movement should be under direct vision to prevent any damage to surrounding tissues.
- The operator should be capable of handling any complications and converting to an open procedure if necessary.
- Specific instruments include stapling devices, lasers, dissectors and retractors.

Indications

The common indications for VATS are diagnosis of pleural diseases, cancer staging, management of persistent pneumothorax, retained hemothorax, infected pleural space and collections including empyema, pericardial drainage or window, apical bullectomy and thoracic sympathectomy. Improved technology and growing surgical expertise has led to more procedures being added to the list: thoracic duct ligation, removal of thoracic cyst,

vagotomy, lobar resection and even thymectomy and esophageal surgery.

Contraindications

Pleural symphysis caused by previous thoracic surgery or pleurodesis, bleeding disorders, end-stage pulmonary fibrosis, respiratory insufficiency and hemodynamic instability are some of the contraindications for VATS.

Advantages

Video-assisted thoracoscopic surgery plays a bridging role between the medical and aggressive surgical managements. It is associated with shorter length of hospital stay and less use of pain medication than thoracotomy in the treatment of pneumothorax and minor resections. In the treatment of pneumothorax, VATS is superior to pleural drainage and has a complication profile similar to that for thoracotomy. There is not enough evidence for its use in lobectomies. VATS is associated with better preserved cellular immunity and less inflammatory and immunomodulatory response compared with conventional thoracotomy, which may have an effect on tumor biological behavior.

Pre-operative evaluation

All thoracoscopic surgery should be treated as major procedures because of the pathophysiological changes and the potential for morbidity and mortality. Ambulatory surgery is therefore not appropriate, except for the simplest procedures. A thorough history and physical examination with special attention to the cardiorespiratory status is necessary for all patients. The pre-operative visit is useful to explain the procedure, associated risks, peri-operative care including options for pain relief as well as to provide pre-medication, if necessary. Routine laboratory investigations include full blood count, serum electrolyte levels and electrocardiogram (ECG). Chest radiographs and CT scans help

to make a diagnosis and identify potential problems with airway management. Spirometry tests including forced vital capacity (FVC), forced expiratory volume in 1 second (FEV₁) and the ratio FEV₁/FVC provide information about the severity of restrictive or obstructive disease. Pre-operative optimization of respiratory function is achieved by bronchodilators, cessation of smoking, incentive spirometry and physiotherapy.

Intra-operative management

The goals of anesthesia include maintaining stable cardiovascular function, optimizing oxygenation and ventilation, minimizing airway reactivity and preventing ventilatory depression in the post-operative period. The pathophysiological changes of lateral decubitus position, one-lung ventilation, existing disease process and carbon dioxide (CO₂) insufflation must be recognized.

Monitoring: Standard monitoring includes ECG, non-invasive blood pressure measurement, pulse oximetry, capnography, volatile anesthetic agent concentration, temperature and peripheral nerve stimulator. Invasive arterial pressure and central venous pressure monitoring may be needed for patients with poor cardiorespiratory reserve. Monitoring airway pressure, tidal volume and minute volume assist management of ventilation. Pressure-volume loops help in detecting changes in lung compliance and elastance.

Anesthesia: Thoracoscopic surgery has been performed under local, regional or general anesthesia. Pre-medication may include anxiolytics. General anesthesia is usually induced with an intravenous agent such as propofol or thiopentone and maintained with an inhalational agent such as isoflurane in an air/oxygen mixture. Nitrous oxide is preferably avoided because of the risk of expansion in closed air-filled spaces. The inhalational agents provide anesthesia, suppress airway reflexes and induce bronchodilation but have some inhibitory effect

on the mechanism of hypoxic pulmonary vasoconstriction (HPV). HPV, a unique autoregulatory mechanism which results in pulmonary vasoconstriction in response to regional alveolar hypoxia ($\text{PaO}_2 = 30 \text{ mmHg}$), is helpful in minimizing the shunt. Total intravenous anesthesia has no effect on HPV. Narcotic analgesia attenuates stress response, reduce minimum alveolar concentration (MAC) requirement of inhalational agents and provide analgesia. Neuromuscular blockade is used to facilitate endotracheal intubation and maintain muscle relaxation.

Positioning: Most VATS require patient to be placed in a lateral decubitus position with arching of the table to widen the intercostal spaces on the operated side. Care should be taken to prevent displacement of the endobronchial tube. Protection of eyes and nerve areas is essential to prevent potential nerve damage. Humidification of anesthetic gases and forced air warming may be used to maintain body temperature.

One-lung ventilation: One-lung ventilation is defined as physiological and anatomical separation of the two lungs by manipulation of the airway. Although thoracoscopic surgery has been considered as a relative indication for one-lung ventilation, the onset of complex surgical procedures has justified the need for lung separation. Lung isolation may be achieved using either a double-lumen endotracheal tube (DLT), single-lumen endotracheal tube with a built-in bronchial blocker (Univent Tube) or an endobronchial blocker such as Arndt (wire-guided) endobronchial blocker or balloon-tipped luminal catheter.

The largest diameter DLT that will pass through the patient's glottis should be used. Smaller tube diameters increase the resistance and the work of breathing. Current practice favors left-sided intubation in the majority of cases. Malposition of DLT is common and position must be confirmed using auscultation techniques and the fiberoptic bron-

choscope. Displacement of the tube is manifested by sudden inflation of the non-dependent lung or ventilation difficulty of the dependent lung with increases in airway pressure.

The atelectatic operative lung is fully reinflated and two-lung ventilation is recommenced. The balloon on the bronchial blocker is deflated and the blocker may be retracted from the bronchus. After discontinuing the inhalational agent and reversing muscle relaxation, spontaneous ventilation is resumed. After ensuring adequate oxygenation, ventilation and consciousness, the trachea should be extubated. If the patient is hemodynamically unstable or demonstrates respiratory insufficiency and extubation is not feasible, the double lumen tube should be exchanged for a single lumen endotracheal tube.

Analgesia: Post-operative thoracic pain may contribute to atelectasis and pulmonary complications by preventing deep respiration and coughing. Pain management strategies include paracetamol, NSAIDs, oral opiates, intravenous patient-controlled analgesia, local anesthetic infiltration, intercostal nerve blockade, paravertebral block and even epidural analgesia.

Post-operative care: Routine monitoring of the patient is vital in the recovery room. A portable chest X-ray may be needed to confirm re-expansion of the collapsed lung. Patients should be nursed in upright position and given supplemental oxygen.

Carbon dioxide insufflation

Carbon dioxide gas may be used to insufflate the pleural cavity so as to accelerate lung deflation. Rapid or excessive insufflation of the gas may cause mediastinal shift resulting in hemodynamic instability, bradycardia and hypotension or hypoxia and surgical emphysema. Gas flow is restricted to 2 l/min with the pressure limited to 10 mmHg.

Complications

Video-assisted thoracoscopic surgery is a safe procedure, but extra caution is recommended for patients with a higher risk profile. Factors such as patient age, duration of the VATS procedure, redo-VATS, patients with immune deficiency and conversion to open thoracotomy have been shown to increase the incidence of complications.

Hypoxemia: Arterial hypoxemia, caused by ventilation perfusion mismatch, is treated by increasing inspired oxygen concentration, positive end-expiratory pressure to the ventilated lung, continuous positive airway pressure to the non-ventilated lung and intermittent two-lung ventilation.

Chest pain: The earliest problem is chest pain either in the axillary, scapular or back region. This probably results from thermal damage to the parietal pleura and the periosteum over the ribs.

Respiratory complications: Pre-existing lung disease, lung deflation and pain may encourage sputum retention, decreased functional residual capacity, ventilation perfusion mismatch and atelectasis.

Bleeding: Bleeding may be caused either by injury to blood vessels or by lung perforation. This can be prevented by safe points of entry and electrocoagulation.

Video-assisted thoracoscopic lung resection

Thoracoscopic lobectomy can be oncologically equal to conventional open procedures with an experienced surgeon and have similar survival for early stage non-small cell lung cancer. Peterson *et al.* have demonstrated that thoracoscopic lobectomy is feasible, safe and effective after induction therapy. The advantages compared with a thoracotomy are:

- Shorter length of hospital stay.
- Decreased post-operative pain.
- Preserved pulmonary function.
- Superior cosmetic result.
- Shorter recovery time.

- Improved delivery of adjuvant chemotherapy.
- Lower morbidity in patients with poor lung function.

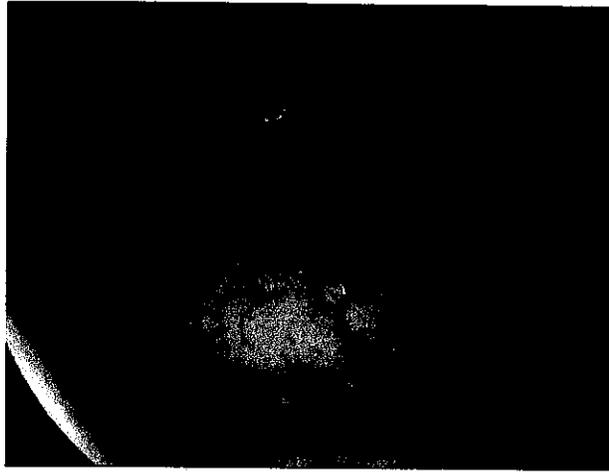
Relative contraindications to thoracoscopic lobectomy include the inability to achieve complete resection with lobectomy, T3 or T4 tumors, N2 or N3 disease and patients with tumor involving the chest wall. The criterion for tumor size precluding VATS resection has not been defined, although tumors greater than 6 cm in diameter may not be removed without rib spreading.

As thoracoscopic procedures are largely dependent on complete lung isolation, communication between the surgeon and anesthetist is critical. When difficulties with one-lung ventilation are encountered, the surgeon should be aware as soon as possible, so that the problem is managed smoothly and efficiently. If such communication is not established and the operative lung needs to be re-inflated without notice, disaster could result if this coincided with a critical stage of the procedure and can result in bleeding.

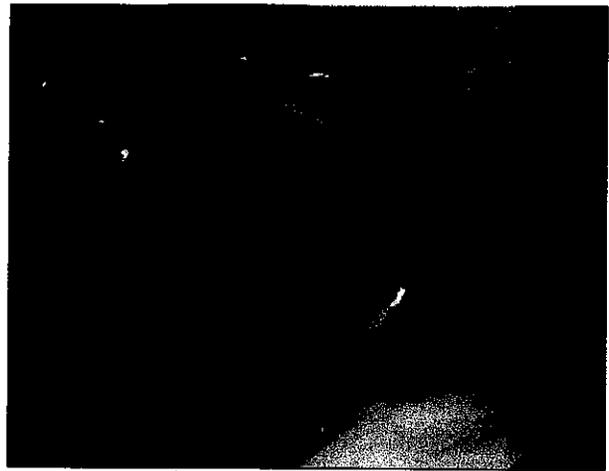
The isolation and division of the bronchi and pulmonary blood vessels require more accurate and extensive dissection with VATS than conventional surgery (see Figure 11.1). Thoracoscopic pulmonary resections can be performed by capable surgeons without an increased bleeding risk.

Conversion to thoracotomy

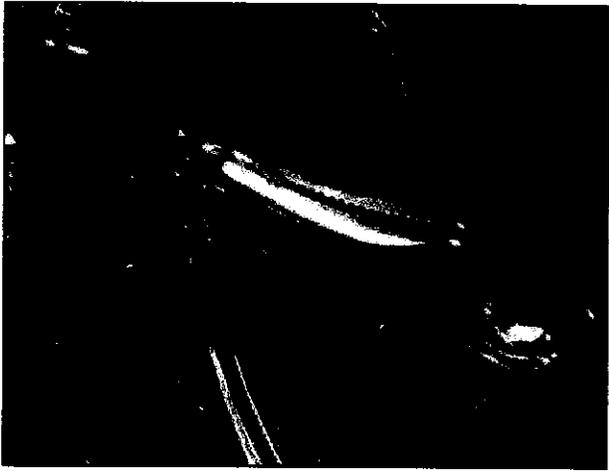
Conversion to a thoracotomy is sometimes required if there is an unexpected change in the patient's condition such as chest wall invasion or the need for a sleeve resection. However, although conversion to thoracotomy should always be considered as a tool available to manage any unexpected situation, conversion rates have been shown to be as low as 1.6–2.5% by McKenna *et al.* in large series by experienced thoracoscopic surgeons. Conversion to an open procedure also becomes necessary



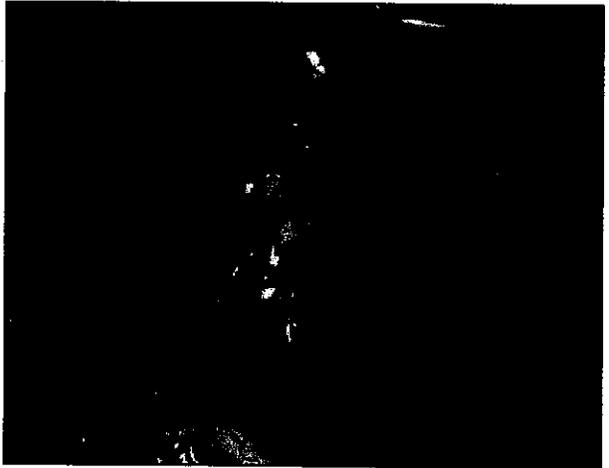
(a)



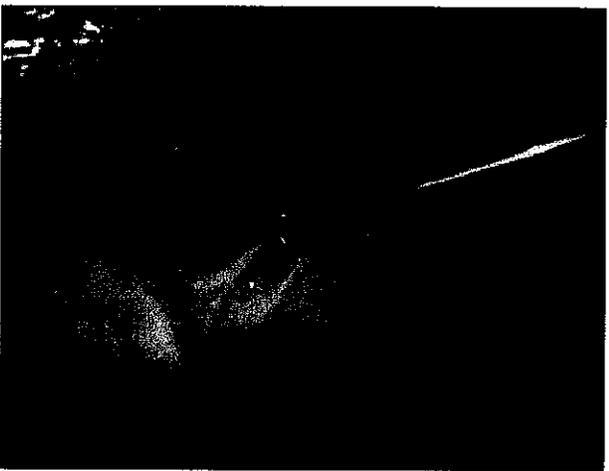
(b)



(c)



(d)



(e)

Figure 11.1 Video-assisted thoracoscopic lobectomy. Following establishment of one-lung ventilation, the ports are placed in the 4th interspace anteriorly, in the 7th interspace (for the camera) and a further one posteriorly. The anatomy is displayed and structures identified starting with the anterior fissure (1a), the main hilar vessels and bronchi and the phrenic nerve (1b: here lying on the SVC). Dissection of the anterior hilum follows with division first of the veins; followed by the bronchial artery (1c) and finally the bronchus (1d). The lymph nodes are sampled and the specimen bagged and removed. Prior to closure a paravertebral catheter is placed, the phrenic nerve is infiltrated (1e) and a drain placed. (Photographs courtesy of Mr. SA Stamenkovic.)

if the patient cannot tolerate one-lung ventilation or develops cardiovascular instability.

Conclusion

Video-assisted thoracoscopic surgery has become a vital part of the armamentarium of the surgeon, however, enthusiasm must clearly be tempered with caution; VATS is only a method, rather than the goal of the treatment, and conversion to open procedures should be performed if necessary. There are extra demands on the anesthetist to provide excellent operating conditions whilst ensuring patient safety.

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SECTION 3 Post-operative Management

- 23. Peri-operative Intravenous Fluid Management**
Sameena T. Ahmed

- 28. Pain Management After Thoracotomy**
Alexander Ng & Christine Tan

Peri-operative intravenous fluid management

SAMEENA T. AHMED

Management of intravenous fluids during thoracic surgery, particularly lung resection, is a longstanding contentious issue between anaesthetists and thoracic surgeons. The anaesthetists tend to focus upon the undesirable effects of tissue and organ hypoperfusion while the surgeons worry about problems caused by fluid overload and pulmonary edema after lung resection. The theory of fluid overload precipitating pulmonary edema was suggested by Zeldin *et al.* Their study consisted of ten patients who developed post-pneumonectomy pulmonary edema (PPO) and compared them with controls retrospectively. Increased amounts of intravenous fluids in the peri-operative period associated with large diuresis were found to be risk factors for this complication. However, fluid input and output data were only available for four of the ten cases. These authors further confirmed their findings in a dog model.

They recommended that "the most important thing we can do as surgeons in terms of recognising this problem is to watch our anaesthesiologists as they start loading the patients up with fluids."

Subsequent studies were unable to replicate this finding if the left heart filling pressures remained normal. Other investigators found no difference in the post-operative fluid balance of the patients who developed PPO and those who did not. These differences in the reported literature regarding fluid

administration and the potentially fatal complication of PPO may be due to variable clinical practices and poor quality of retrospectively collected data.

A cohort analysis of risk factors for acute lung injury (ALI) after surgery for lung cancer by Licker *et al.* demonstrated that excessive fluid infusion was a risk factor (odds ratio 2.9; 95% confidence interval). They found that patients who developed ALI had received in excess of 3 liters in the first 24 hours after surgery.

Effects of surgical trauma *Physiological changes resulting in fluid retention*

Surgical trauma elicits a stress response via endocrine and inflammatory processes. The effects on fluid balance are mediated via the antidiuretic hormone (ADH), aldosterone and the renin-angiotensin II systems. It results in an increase in sodium and water retention with excessive potassium excretion.

- Increased ADH reduces diuresis and decreases plasma concentration of sodium.
- Increased aldosterone and renin-angiotensin activity leads to sodium retention and potassium excretion.

There is no evidence to suggest that renal function deteriorates in normovolemic patients with reduced

urinary output. Protection of the fluid compartment is a physiological response to surgical stress.

Physiological changes to the lungs

Fluid homeostasis in the lungs is maintained via Starling's equilibrium via hydrostatic and colloid oncotic pressures.

- Net pressure pulling fluid out of the capillaries = hydrostatic pressure in the capillary minus the hydrostatic pressure in the interstitium.
- Net pressure pulling the fluid into the capillary = blood oncotic pressure minus the oncotic pressure of the interstitial fluid.

The net flow of lymph in the lungs is about 20 ml/min. This is reduced with pulmonary resection. As a result of fluid overload, the peri-bronchial and peri-vascular tissues become engorged. When the drainage capacity of the interstitial tissue is exceeded, the fluid passes into the alveoli, flooding them and causing pulmonary edema. The left and right lung lymphatic drainage in humans is different. On the right side, 94% of the lymphatic drainage is via the right hilum and 6% transverse the carina to drain into the left mediastinum. On the left side, 56% of lymph drains into the right superior mediastinum. Seventy-eight percent of the left lower lobe drains via the right side. Thus right pneumonectomy with associated trauma to the lymphatic drainage is more likely to cause post-pneumonectomy pulmonary edema than a left pneumonectomy.

Pulmonary capillary wall integrity is impaired after lung manipulation. This occurs after prolonged surgery, lung retraction, tissue handling and lung resection. The remaining lung tissue remains prone to acute lung injury which can progress to ARDS. A recent ARDS clinical trial network has concluded that conservative fluid management in the first 7 days after injury was better than liberal fluid administration. The patients had shorter duration of mechanical ventilation, better lung function and shorter duration of intensive

care. This also points to the fact that maintaining normovolemia in the peri-operative period is preferred in thoracic patients. However, patients having large tissue dissection and mobilization such as esophagectomy and esophago-gastrectomy have higher fluid requirement than patients having pulmonary resection.

Early cardiac response to lung resection

The extent of acute cardiovascular changes after lung resection depends upon the amount of lung removed and underlying lung disease such as COPD. The resting mean pulmonary artery pressure and pulmonary vascular resistance tend to normalize after surgery. They increase by 30% with exercise in patients after pneumonectomy.

However, the right ventricular ejection fraction continues to fall in the first few days after surgery. This decrease in RV function is most likely due to an increase in RV afterload. This also occurs when supplemental oxygen is discontinued. Right ventricle dilation can also alter the left ventricular (LV) compliance by ventricular interdependence. Higher LV filling pressures may be required to maintain cardiac output.

The right ventricle fails if challenged by fluid overload, pulmonary edema, ALI or ARDS.

Assessment of the right ventricle pump functions in the thoracic surgical patient is often subjective and technically limited using transthoracic echocardiography. Thermodilution measurements of cardiac output using a pulmonary artery catheter have the advantage of supplying the pressures within the right heart. Unfortunately, the accuracy of these measurements is reduced in the presence of tricuspid valve regurgitation and exaggerated by acute increases in right ventricular afterload.

Fluid management in the peri-operative period

The aim of fluid management in thoracic surgical patients is to maintain normal hydration and accept

reduction in urine output in the post-operative period.

Traditional anesthetic training dictates that replacement of fluid losses during surgery is the key target for peri-operative fluid management. Fluid losses are calculated based upon the length of fasting, surgical blood loss, evaporative losses from exposed body cavity and urine excretion. General anesthesia produces vasodilatation which further exposes these fluid deficiencies when the blood pressure drops. This is traditionally replaced by a large volume of crystalloid intravenous infusion. Thus intravenous fluid loading is considered indispensable.

However, fluid preloading and large intra-operative fluid administration are not evidence-based practice for best outcomes after surgery. Scientific evidence for an optimal fluid administration regimen resulting in adequate peri-operative organ function is lacking. Hence there is a large variation in the amount and type of fluid administered during surgery. Holte *et al.* demonstrated a reduction in pulmonary function within one hour of infusion of 22 ml/kg or 1–2 liters of normal saline. The same group further demonstrated that infusion of 40 ml/kg of Ringer's lactate over 3 hours reduced pulmonary function test up to 8 hours in healthy volunteers. This was also associated with weight gain. Infusion of fluid in excess of normal hydration can have physiologically adverse effects. These may be attributed to fluid accumulation in the interstitial tissues.

In the modern anesthetic practice, the recommended pre-operative fasting period is 4–6 hours. This does not normally cause intravascular hypovolemia.

- The measured evaporated fluid loss is 0.5 ml/kg. This increases to 1 ml/kg in patients having major surgery.
- Multiple clinical studies have demonstrated that fluid administration of more than 3 liters in the first 24 hours is a risk factor for ALI. Licker *et al.*

also demonstrated that patients who developed ALI received fluids in excess of 1 liter in the intra-operative period.

- Recommendations of standard anesthetic teachings for "third space fluid loss" of 6 ml/kg per hour in thoracic surgery are excessive. Except in unusual situations, it is best to assume there is no "third space" in the thorax.
- Unless there are other indications of developing renal failure, urine output greater than 0.5 ml/kg per hour is unnecessary in the normovolemic thoracic patient post-operatively.
- If increase in tissue perfusion is required in the peri-operative period, inotropes and vasoconstrictors should be used (guided by invasive monitoring) instead of risking volume overloading.

The management of acute right ventricular failure primarily involves inotropes, pulmonary vasodilators and inodilators. It is important to remember that vasoconstrictors such as pitressin and norepinephrine may be necessary to maintain right ventricular perfusion pressures.

Assessment of intravascular filling status in the peri-operative period

Blood pressure and cardiac filling pressures by themselves alone are a poor predictor of volume status. High central venous pressure can be due to right heart failure, raised pulmonary pressures, patient coughing on the endotracheal tube, etc. It does not reflect filling status. Similarly, raised blood pressure reflects raised systemic vasoconstriction and not adequate filling status. Findings from the physical examination such as mental status, blood pressure, heart rate, skin turgor, urinary output and serum electrolytes/osmolality can be used in awake patients. However, these have their drawbacks in anesthetised patients.

Trends of mixed venous saturation from the SVC and PA can indicate a change in tissue oxygenation.

Peri-operative goal-directed intravenous fluid optimization using esophageal Doppler to guide an increase in cardiac stroke volume has demonstrated a reduction in hospital stay and fewer complications after colorectal surgery. There are no studies to indicate its effect on the thoracic surgical population.

However, less invasive measures such as systolic arterial pulse pressure variations in mechanically ventilated patients and central venous pressure variations in spontaneously breathing patients can distinguish between responders and non-responders to fluid challenges.

At present, there are no evidence-based data that using this can improve outcomes in thoracic surgical patients.

Blood transfusion in thoracic surgery

The purpose of blood transfusion in the peri-operative period (Box 23.1) is for the following:

- Improve oxygen carrying capacity.
- Improve hemostasis.
- Support circulating volume.

In addition to the well-known complications of blood transfusion, there is evidence that peri-operative blood transfusion can lead to immunosuppression. This effect can be detrimental in patients with cancer. Patients undergoing esophagectomy for cancer can have a worse outcome in terms of survival after receiving red cell transfusion.

Box 23.1 Factors that predispose to blood transfusion in thoracic surgery

Pre-existing anemia.
Previous thoracic operations.
Resection of inflammatory and infective disease processes.
Decortication of empyema.
Chest wall resection.

Patients with lung malignancies can become anemic pre-operatively. Anemia can be a sign of a more aggressive tumor. Studies have demonstrated that patients with non-small cell lung cancer (NSCLC) having a low hemoglobin concentration pre-operatively have a worse outcome. Early retrospective studies had concluded that patients with NSCLC having a blood transfusion did not have a good survival outcome. Berardi *et al.* studied the effects of peri-operative anemia and blood transfusion in patients undergoing lung resection for NSCLC. They demonstrated a worse prognosis in patients with hemoglobin less than 10 g/dl and those transfused in the peri-operative period. They concluded that anemia could be an important prognostic factor. Correction of anemia did not reduce the risk of relapse.

The immunosuppression induced by transfusion results from both an early unspecific pathway mediated by monocytes and a later phase induced one from increased suppressor T cell activity. Both effects are dependent on the number of transfusions. Blood transfusion has been shown to impair natural killer cell function and lower the CD4 to CD8 ratio. In addition, prostaglandin E₂ levels are increased after transfusion. This may result in a direct inhibition of interleukin-2 production from CD4 cells with subsequent effect, as interleukin-2 is obligatory for natural killer cell activity. Although it is not clear at a molecular level which factors influence immunosuppression after allogeneic blood transfusion in cancer surgery, there is good evidence that leukocytes in the blood mediate the effects seen.

Transfusion of fresh frozen plasma (FFP) has been linked with the development of pulmonary edema after lung resection. This might be due to the immunological effect. Fresh frozen plasma contains the complete (stable and unstable) humoral components of blood coagulation, fibrinolytic and complement systems. The presence of antibodies

against the leukocytes in the FFP can trigger the onset of pulmonary edema. The activated leukocytes and granulocytes can migrate into the interstitial space between the alveolar and capillary endothelium resulting in capillary leaks and pulmonary edema. This is also known as transfusion related lung injury (TRALI). It is a variant of ARDS.

In the UK, all allogenic blood for transfusion is leukocyte depleted to minimize the possibility of new variant Creutzfeldt-Jakob disease transmission.

In the author's institute, there was not a strict blood transfusion policy for thoracic patients. However, care is taken to limit the amount of blood given peri-operatively. Other variables such as hemodynamic status and oxygen delivery are more important than an arbitrary hemoglobin concentration. Some patients will tolerate a lower hemoglobin level while others benefit from a transfusion.

Major blood loss may be unavoidable in operations. However, every effort should be made to reduce the amount of blood lost at the time of operation to an absolute minimum, and the importance of meticulous surgical technique cannot be overemphasized.

Other strategies that may limit the requirement of allogenic transfusion include use of either pre-donated autologous blood or a Cell Saver. The use of a Cell Saver for transfusion of shed blood peri-operatively can increase the risk of dissemination of malignant cells and should be avoided in patients with cancer.

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Pain management after thoracotomy

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The purpose of this chapter is to provide the reader with a concise account of locations of pain after thoracotomy, operative considerations relevant to pain relief and current methods of analgesia.

Neural input and locations of pain

Acute pain after thoracotomy occurs because of afferent nociceptive transmission along the intercostal, vagus and phrenic nerves. Patients experience incisional pain which is attributable to neural transmission along the intercostal nerves. In addition, there may be visceral pain which occurs as a result of vagal innervation of the thoracic organs.

Ipsilateral shoulder pain after thoracotomy is another frequent problem. It is of at least moderate intensity and occurs despite blockade of the suprascapular nerve supplying the shoulder. Deposition of local anesthetic in the interpleural space above the diaphragm does not obviate its occurrence. However, infiltration around the phrenic nerve with lidocaine has been shown to significantly reduce its occurrence.

Ipsilateral shoulder pain after thoracotomy is an example of referred pain rather than damage to the shoulder. Nociceptive transmission from the diaphragm and pericardium is transmitted along the phrenic nerve to the spinal cord. As

the phrenic nerve and the shoulder have similar nerve roots, convergence of visceral and somatic impulses is possible. Ascending spinal pathways may be misinterpreted as emanating from the shoulder joint rather than from the diaphragm. This convergence-projection theory differs from the convergence-facilitation theory. In the latter, visceral diaphragmatic afferent input to the spinal cord would appear to cause the formation of an irritable focus rather than to be nociceptive per se. Shoulder pain arises because of amplification of previously innocuous somatic impulses from the shoulder.

Operative considerations

From the analgesic perspective, operative considerations may be classified into standard posterolateral thoracotomy and muscle-sparing thoracotomy.

Posterolateral thoracotomy

During a standard posterolateral thoracotomy, division of latissimus dorsi is required to obtain access to the intercostal space. In addition, it may be necessary to transect serratus anterior and trapezius.

Muscle-sparing thoracotomy

In a muscle-sparing thoracotomy, latissimus dorsi is not incised and serratus anterior is dissected and

Table 28.1 Analgesic techniques for thoracotomy.

Technique	Example
Thoracic paravertebral blockade	Bolus: bupivacaine 0.375% 20 ml Maintenance: bupivacaine 0.25% 0.1 ml/kg per hour
Intrathecal opioids	Single shot of: Morphine 500–750 µg With or without fentanyl 25 µg
Thoracic epidural analgesia	Bolus solution: bupivacaine 0.25% 10–20 ml Background solution: bupivacaine 0.1% with fentanyl 2–5 µg/ml Background infusion rate 0.1 ml/kg/hour Patient-controlled boluses possible e.g. 2 ml, with lockout interval of 30 minutes
Intravenous opioid by PCA	Morphine loading during surgery. 0.1–0.2 mg/kg Bolus: morphine 1 mg Lockout 5 minutes Maximum hourly dose 12 mg Background infusion, not required usually
Paracetamol	Regular paracetamol Dose 1 g, oral, rectal or intravenous
NSAIDs	Non-selective NSAIDs Oral or rectal diclofenac 150 mg per day in divided doses Selective NSAIDs (COX-2 inhibitors) Intravenous parecoxib 40 mg, twice-daily
Others	Epidural clonidine Epidural epinephrine Oral gabapentin Intercostal nerve blockade Intravenous tramadol

Please note that the above drugs and doses are examples only. Drugs should be administered by appropriately trained and qualified practitioners. Adequate monitoring facilities are required.

reflected off the ribs to allow intercostal incision to be performed. However, the skin incision of a muscle-sparing thoracotomy traverses two to three dermatomes whereas that of the standard approach is over one dermatome. In addition, as access to the lungs is limited with a muscle-sparing thoracotomy, the ribs and surrounding tissue may have to be spread wider than when a posterolateral thoracotomy is performed. So, it can be seen that tissue trauma is not necessarily reduced after muscle-sparing thoracotomy.

Methods of analgesia

The main methods of analgesia are paravertebral analgesia, intrathecal opioids, epidural blockade and analgesic adjuncts (Table 28.1).

Paravertebral analgesia

ANATOMICAL CONSIDERATIONS

The paravertebral space is a potential wedge-shaped region, located lateral to the vertebral column and medial to the intercostal space. It lies posterior to

the parietal pleura, and anterior to the superior costotransverse ligament and posterior intercostal membrane. Its superior and inferior borders are uncertain but it is probable that they extend from the occiput to the alar of the sacrum.

The paravertebral space contains spinal nerves that emanate from the intervertebral foramen. Components of these spinal nerves include their anterior and posterior rami as well as the white and grey rami communicantes. These spinal nerves are in continuity with the intercostal nerves, sympathetic chain, brachial plexus, lumbar plexus as well as the cervical and stellate ganglia.

Nociceptive conduction in the spinal nerves travelling through the paravertebral space is susceptible to blockade because of the lack of a surrounding fascial sheath. Owing to its close proximity, the ipsilateral sympathetic chain may be blocked by local anesthetics. In theory, bilateral neural blockade is possible because the paravertebral space is in continuity with the epidural space via the intervertebral foramen and also with the contralateral paravertebral space via the prevertebral and epidural spaces.

TECHNIQUE

To provide paravertebral analgesia for thoracic surgery, a Tuohy needle similar to that used for an epidural is used. Typically, this needle is inserted approximately 3 cm lateral to the spinous process of the fifth thoracic vertebrae, on the operative side. As the needle is advanced, it impinges on the transverse process of the sixth thoracic vertebrae. The needle is then redirected over the superior border of this transverse process to enter the paravertebral space which may be identified by loss of resistance to air or saline.

After bolus administration of local anesthetic into the paravertebral space, onset of blockade occurs after 40 minutes and ipsilateral analgesia

two dermatomes above as well as two dermatomes below the level of insertion is obtained generally. However, the degree of spread of local anesthetic is variable in the same patient and does not appear to be attributable to previous thoracotomy, age, sex, height and weight.

Variations on the above description of administration exist, for instance, placement of a catheter and multiple injections.

- **Catheter:** A catheter may be inserted percutaneously at induction of anesthesia or may be placed under direct vision by the surgeon during surgery. After surgery, a continuous infusion of local anesthetic may be administered.
- **Multiple injections:** Another variation of the single-shot technique at T5 or T6 is to administer local anesthetic in divided doses at T3 and at T7. The basis for this method is that thoracotomy at the fifth intercostal space involves dissection of muscle and skin innervated by dermatomes above and below T5 or T6. Blockade of afferent neural transmission in these dermatomes relies on adequate paravertebral cephalad and caudad diffusion of local anesthetic after a single injection. However, by administration of local anesthetic to the upper and lower dermatomal borders, it is thought that this uncertainty and hence possibility of insufficient analgesia may be obviated.

COMPLICATIONS OF PARAVERTEBRAL ANALGESIA

Complications of paravertebral analgesia are uncommon but they may include: pleural puncture, pneumothorax, vascular puncture, hypotension, dural puncture and Horner syndrome.

Horner syndrome is caused by blockade of the stellate ganglion or of the sympathetic preganglionic fibers of the first three thoracic vertebrae.

Clinical signs include ipsilateral ptosis, miosis, anhidrosis and enophthalmos. Rarely, it may coexist with harlequin syndrome contralateral to the side of paravertebral blockade. Harlequin syndrome comprises unilateral facial flushing as well as sweating, and thus it contrasts with the hemifacial pallor of Horner syndrome. Both syndromes are transient.

Intrathecal opioid analgesia

Intrathecal opioids provide effective analgesia after thoracotomy. Studies have been performed with preservative-free morphine, sufentanil and fentanyl. Morphine has a delayed onset but longer duration of action than fentanyl and sufentanil.

A combination of intrathecal morphine and sufentanil has been found to be associated with significant reduction in rescue morphine consumption as well as pain intensity at rest and coughing, for approximately 8–24 h, compared with placebo.

COMPLICATIONS OF INTRATHECAL OPIOIDS

After administration of intrathecal morphine 650 µg to 800 µg, the reported incidence of pruritus and nausea or vomiting is 37% and 25%, respectively. Other complications, for instance, respiratory depression, postdural puncture headache and epidural patch for dural puncture headache, occur in 3%, 0.54% and 0.37% of patients, respectively. Life-threatening respiratory failure, nerve injury and infection in the cerebrospinal fluid are most unlikely to occur.

Epidural analgesia

Epidural analgesia is often considered to be the gold standard for management of pain after thoracotomy. In this section, a number of issues will be discussed, i.e. epidural solutions; a comparison with other methods of analgesia e.g. intravenous

morphine by PCA, paravertebral analgesia; and complications of epidurals.

EPIDURAL SOLUTIONS

The ideal epidural solution would be one which provides high-quality analgesia but without the adverse effects of its ingredients. Thoracic epidural solutions typically comprise local anesthetic, an opioid or a combination of both. Local anesthetics are associated with hypotension, motor blockade and urinary retention while opioids may cause pruritus, nausea and excessive sedation.

Administration of epidural bupivacaine and epidural morphine has been studied after thoracotomy. A bolus of 6–10 ml of bupivacaine 0.25% followed by an infusion of 3–5 ml of bupivacaine 0.25% for 3 days is as efficacious for analgesia as an initial dose of morphine of 2–3 mg and an infusion of morphine 0.1 mg/ml at 2 ml/h, for 3 days. However, the incidence of post-operative tachyarrhythmias may be significantly lower in patients who receive bupivacaine than in those who have morphine. This beneficial effect of epidural local anesthetic would appear to be attributable to sympathetic blockade and attenuation of a sympathotonic state during thoracic surgery.

The adverse effects of epidural local anesthetic and opioids are dose dependent and so the concentration of them would be one which provides effective analgesia with minimum adverse effects. The optimal concentration of bupivacaine in fentanyl 10 µg/ml has been studied. Bupivacaine 0.2% with fentanyl 10 µg/ml has been found to be associated with increased hypotension and use of vasopressors than bupivacaine 0.1% with fentanyl 10 µg/ml. Pain relief is similar in both groups and so it may be seen that bupivacaine 0.1% is the more favorable concentration after thoracotomy.

In another clinical trial of patients after thoracotomy, epidural fentanyl at 2, 5 and 10 µg/ml

in bupivacaine 0.1% have been compared. The solution containing fentanyl 5 µg/ml appears to be optimal because it provides better analgesia than that containing fentanyl 2 µg/ml and also because opioid-related adverse effects are reduced compared with fentanyl 10 µg/ml.

EPIDURAL ANALGESIA VS. INTRAVENOUS MORPHINE BY PCA

Epidural analgesia has been shown to provide better analgesia than intravenous morphine by PCA. In the recovery period, pre-incisional epidural bupivacaine 0.1% with morphine 0.05 mg/ml to 0.1 mg/ml is associated with significantly lower pain intensity scores at rest, cough and movement than intravenous morphine by PCA. In addition, the proportion of patients with pain lasting at least 2 months and of those with pain at 6 months would appear to be significantly lower in patients receiving epidural than in those who are prescribed intravenous morphine.

EPIDURAL VS. PARAVERTEBRAL ANALGESIA

It has been suggested that epidural analgesia may not be as effective as paravertebral blockade after thoracotomy. In a prospective randomized trial, patients who had epidural analgesia received 3 ml of bupivacaine 0.5% initially, 10–15 ml of bupivacaine 0.25% during surgery, 10 ml of bupivacaine 0.25% at the end of surgery and an infusion of bupivacaine 0.25% at 0.1 ml/kg per hour. The paravertebral group received bupivacaine 0.5% 20 ml prior to incision, bupivacaine 0.25% 20 ml at the end of surgery, and a continuous infusion of bupivacaine 0.5% at 0.1 ml/kg per hour. Pain scores at rest and on coughing, rescue morphine consumption, plasma cortisol concentration and rise in glucose were significantly higher in patients receiving epidural analgesia than in those given paravertebral analgesia. Post-operative peak expiratory flow rates and oxygen saturation were significantly lower in the epidural group than in the paravertebral group.

These results have been supported, in part, by a systematic review and meta-analysis of ten randomized clinical trials comparing bolus and infusions of local anesthetics into the epidural and paravertebral spaces of patients having a thoracotomy. Pulmonary complications were significantly higher in patients receiving epidural analgesia than in those having paravertebral blockade. In addition, epidural analgesia was associated with significantly more urinary retention, nausea and vomiting, hypotension and block failure than paravertebral analgesia. However, both methods of analgesia appeared to be equally efficacious as there was no significant difference between them in post-operative pain scores and supplemental morphine consumption.

COMPLICATIONS OF EPIDURALS

As described above, epidurals are associated with adverse effects related to the components of the solutions administered e.g. pruritus, nausea and hypotension. In addition, infection is a complication and patients have an infrequent but serious risk of developing an epidural abscess. Neurological complications may occur after epidurals; they include dural perforation, radicular pain during epidural puncture or catheter insertion, transient post-operative radicular pain, peripheral nerve lesions, peroneal nerve palsy, other peripheral nerve lesions and paraplegia. To minimize this risk of neurological complications, there has been discussion that thoracic epidurals should be inserted awake rather than under general anesthesia.

Analgesic adjuncts

Simple analgesics such as NSAIDs and paracetamol have been shown to be useful for pain relief after surgery. In thoracic surgery, they have three main advantages:

- They have a systemic effect and thus can be used for the treatment of shoulder pain, especially in patients who have had an epidural.

- They minimize the dose of opioids used in epidurals, spinals and intravenous opioid infusions. Thus there may be a reduction in the possibility of respiratory depression in patients with partial lung function.
- They bridge the analgesic gap when either epidural or intravenous opioids are discontinued. The analgesic gap represents the period of time when patients may experience increased pain because a form of advanced analgesic support is discontinued and simple analgesics have not been commenced.

In addition, other analgesics, for instance, gabapentin may be useful for post-operative analgesia. Compared with placebo, oral gabapentin 1.2 mg, on the day of surgery and for a further 2 days has been associated with significantly lower pain intensity scores, paracetamol consumption, rescue epidural boluses, duration of epidural analgesia and motor block.

Key points

- Shoulder pain and chronic pain after thoracotomy are common.
- To manage pain after thoracotomy, the main methods of analgesia comprise epidural blockade, local anesthetics in the ipsilateral paravertebral space and intrathecal opioids.
- Analgesic adjuncts are useful e.g. paracetamol.

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