Chapter 18: Anesthesia for Thoracic Procedures

Anne E. Cossu; Doris M. Hardacker

INTRODUCTION

FOCUS POINTS

1. 1.Type II pneumocytes develop at 24 to 26 weeks of gestation and begin producing surfactant. Eight to ten percent of the number of adult alveoli are present at birth
2. Infants and children have reduced functional residual capacity (FRC) and higher oxygen consumption (6 to 8 ml/kg/min) rendering them susceptible to faster oxygen desaturation.
3. Etiologies for increased ventilation to perfusion (V/Q) mismatch during thoracic surgery include lateral positioning, general anesthesia and blunting of hypoxic pulmonary vasoconstriction, mechanical ventilation and surgical manipulation or single-lung ventilation.
4. One-lung ventilation (OLV) can be achieved utilizing single lumen endotracheal tubes inserted into the main stem bronchus, endobronchial blockers, Univent tubes or double lumen tubes dependent on the size of the patient.
5. Management of hypoxemia during OLV includes 100% oxygen, continuous positive airway pressure (CPAP) to the nondependent lung, positive end-expiratory pressure (PEEP) to the dependent lung, double lung ventilation and in extremis occlusion of the pulmonary artery to the operative lung.

   Thoracic and mediastinal masses are lesions that may cause airway compromise prior to or during a procedure under anesthesia. Thorough preoperative evaluation that includes review of echocardiogram, available imaging, and symptoms allow for a safer anesthetic. Spontaneous ventilation with min sedation may be the anesthetic of choice.

DEVELOPMENT OF THE RESPIRATORY SYSTEM

The morphological development of the lung begins several weeks after conception and continues well into the first decade of life. Four stages of lung development and maturation were described initially by Dubreuil,[1] which was then updated in 1988 by Thurlbeck.[2] The first stage, called the embryonic stage, spans from 2–3 to 7–8 weeks of gestation and begins as an outgrowth of endoderm, surrounded by mesenchymal tissue, from the ventral foregut. This outgrowth extends into the pleuroperitoneal cavity and eventually forms lung buds.[3] A concurrent event is the development of the tracheoesophageal ridge. Incomplete separation of this ridge may lead to formation of a tracheoesophageal fistula.[4] The next phase, the pseudoglandular stage, extends from 7 to 16 weeks postconception and is punctuated by the formation of the conducting airways and pulmonary vasculature. Therefore, a fetus cannot survive if born at this stage of development.[5] The diaphragm forms during this time frame and consequently, any disruption may lead to herniation of abdominal organs into the thorax and hypoplasia of the lung, that is, congenital diaphragmatic hernia.[6] The canalicular phase, extending from 17 to 28 weeks postconception, is characterized by the development of acini and adjacent vascular growth. During this stage, bronchi and bronchioles increase in luminal diameter.[7] Type II pneumocytes develop by 24 to 26 weeks' gestation and potentially as early as 20 weeks.[8,9] These cells produce surfactant, which acts to reduce surface tension and stabilize airspaces.[10] Capillary networks surrounding the acini proliferate and allow for sufficient respiratory exchange by 26 to 28 weeks.[11] Premature infants can survive if born at 24 weeks; however, exogenous surfactant may be required to reduce pulmonary morbidity at this point. From 29 to 36 weeks of gestation, lung development enters the saccular phase

at which time acini mature into saccules and there is formation of an extensive capillary network.[12] The last stage called the alveolar stage may begin as early as 32 weeks in some fetuses but is not consistently present until 36 weeks and extends well beyond birth. At this point, alveoli begin to develop from saccules.[13,14]

There are approximately 20 to 50 million terminal air sacs (mostly saccules) present at birth in the neonatal lung, approximately 8% to 10% of that present in the adult lung. During the next 12 to 18 months of postnatal life, the remaining saccules mature into alveoli and there is further development of alveolar ducts connecting to new alveoli.[15] By age 9, there is an estimated 280 million alveoli, which closely approximates adult levels. As the chest wall increases in size from childhood to adulthood, there is a matched expansion of lung surface area due to an increase in size of alveoli and the conducting airways.[16]

NORMAL RESPIRATORY PHYSIOLOGY

Perinatal Adaption to Respiration

Rapid removal of liquid from the fetal lungs is essential to the establishment of pulmonary gas exchange at birth. With the initial gasps of air after birth, there is generation of a negative inspiratory force of up to −70 to −100 cm of H_2O that fills the lungs with air.[17] Residual amniotic fluid is removed from the lungs over days through lymphatic channels and pulmonary capillaries. Rhythmic breathing, which begins during fetal life and hypothetically may be necessary for normal pulmonary development, is maintained after birth due to interrupted umbilical blood flow and hyperoxia relative to the hypoxic fetal environment.[18]

Control of Respiration

After birth, control of respiration is mediated by a complex interplay of the brainstem, stretch receptors in the airways and lung via the vagal nerve, peripheral and central chemoreceptors, and spinal reflexes.[19] The dorsal respiratory group (DRG) of neurons, located in the dorsomedial medulla, contains inspiratory neurons. The ventral respiratory group (VRG), in the ventrolateral medulla, contains both inspiratory and expiratory neurons.[20–22] Rhythmic breathing in humans is thought to be due to the pre-Botzinger complex, a small cluster of pacemaker neurons, which acts to initiate and maintain breathing in neonates and fetuses.[23]

The upper airways, trachea, bronchi, and chest wall contain mechanoreceptors and chemoreceptors, which function to affect respiration. Stimulation of receptors in the nose produce sneezing, apnea, and alter bronchomotor tone.[24] Infants, neonates, and premature neonates have highly sensitive upper airway reflexes, which may be directly stimulated with inhalational induction. Receptors in the pharynx are involved with swallowing, which coordinate the inhibition of breathing, closure of the larynx, and contraction of the pharyngeal muscles. When pharyngeal reflexes have been blunted, accumulating secretions can stimulate laryngeal receptors leading to prolonged breath holding, apnea, and laryngospasm via the superior laryngeal nerve.[25,26] Hypocapnia and hyperthermia are both known to worsen this response, whereas hypoventilation with hypercapnia, positive end-expiratory pressure, and deep anesthesia can suppress this response.[27]

Tracheobronchial and pulmonary receptors have been classified into three types: the slowly adapting (pulmonary) stretch receptors, the rapidly adapting (irritant or deflation) receptors, and the unmyelinated C-fiber endings (J receptors). Both the slowly adapting and rapidly adapting receptors lead to vagal afferent fibers.[28–31] Slowly adapting receptors (SARs) are located in the smooth muscle of the trachea and central airways.[32] SARs are activated by distension during inhalation and cause inhibition of inspiratory neurons, which is also called the Hering-Breuer inflation reflex. Rapidly adapting receptors (RARs) are located in epithelial cells lining the carina and large bronchi.[30,33] They respond to mechanical and chemical stimuli, such as smoke, inhalation anesthetics, and other irritating gases. RAR stimulation may cause coughing, mucus production, and bronchospasm.[34] J receptors are located near the pulmonary or capillary walls and are stimulated by pulmonary edema and congestion, pulmonary emboli, and inhaled irritants such as inhaled anesthetics. Stimulation may lead to rapid breathing, mucus production, bronchospasm, decreased tidal volume, hypotension, and bradycardia.[35] The muscles of the thorax, including the diaphragm and intercostal muscles, contain various types of mechanoreceptors, such as muscle spindles and Golgi tendon organs.[36,37] Muscle spindles are located in intercostal muscles and are a type of slowly adapting mechanoreceptor that detect stretch. Golgi tendon organs, a type of slowly adapting mechanoreceptor, are located at the point of insertion of muscle fiber into tendon.[22]

The main function of the peripheral and central chemoreceptors is the maintenance of normal pH, PaO_2 and $PaCO_2$ (P and a, respectively, are abbreviations for partial pressure and arterial).[38] The central chemoreceptors, which are located in the ventrolateral medulla, are sensitive to hydrogen ion concentration in the adjacent cerebrospinal fluid. The blood-brain barrier is highly permeable to CO_2 and it easily diffuses into the surrounding cerebrospinal fluid (CSF), which has poor buffering capacity. For this reason, respiratory acidemia readily stimulates respiration via the central chemoreceptors.[39] Conversely, with acute metabolic acidemia or alkalemia, alterations in hydrogen ion concentration in the blood are not transferred to the CSF as rapidly. With chronic metabolic acid-base disturbances, pH of the CSF remains rather close to 7.3 regardless of blood pH. Consequently, ventilation in these circumstances is dependent on the response of the peripheral chemoreceptors.[40]

The peripheral chemoreceptors, called the carotid bodies (located at the bifurcation of the common carotid artery), are sensitive to changes in pH, $PaCO_2$ and the PaO_2. The main role of the carotid bodies is to detect changes in PaO_2.[41] In adults, acute hypoxemia stimulates carotid bodies to increase ventilatory rate, which is partially opposed by hypocapnia due to suppression of central chemoreceptors.[42] Chronic hypoxemia, occurring over years, may cause the carotid bodies to lose their hypoxic response.[43] Neonates exhibit a biphasic response to hypoxemia and hyperoxia in the first several weeks of life.[44] Moderate hypoxemia produces a transient increase in respiratory rate followed by sustained respiratory depression, whereas 100% O_2 causes a transient decrease in respiratory rate, which is then followed by a sustained increase in respiratory rate.[45,46] This biphasic response to hypoxemia is lost by 3 weeks after birth, and conversely hypoxemia produces a sustained increase in ventilation.[46] Premature infants, on the other hand, continue to have this biphasic response to hypoxemia for a longer amount of time. Thus, the proper response to hypoxemia may occur only when the respiratory system has matured on a time course consistent with postconceptual age rather than postnatal age.[47]

In normal adults, ventilation increases linearly with increasing concentration of inspired CO_2 but only to a point, after which ventilation starts to decrease.[48] Hypoxemia potentiates the CO_2 response through carotid body stimulation, which shifts the CO_2 response curve to the left and increases the slope of the curve.[49] Anesthetics, opioids, and barbiturates suppress the medullary chemoreceptors, decrease the slope, and shift the CO_2 response curve to the right. Thus, with increasing concentration of anesthetics, there is a decrease in ventilatory response to an increasing concentration of CO_2.[50] Neonates increase ventilatory rate in response to hypercapnia but to a lesser extent than older infants. With increased gestational age and postnatal age, the slope of the CO_2 response curve increases, which may be due to increased sensitivity of the chemoreceptors and/or maturing mechanics of the thorax. In contrast to adults, hypoxemia in newborn infants decreases the slope and shifts the CO_2 response curve to the right, whereas hyperoxemia has the opposite effect.[51]

Lung Volumes

In normal children and adolescents, lung volumes are proportional to body size and height. In the early stages of lung development after birth, the total lung capacity of neonates and infants is disproportionately small in relation to body size. Additionally, due to high infantile metabolic rate per kilogram of body weight, ventilation per unit of lung volume is much higher versus older children and adults. In sum, the infant has less reserve in lung surface area for oxygen exchange.[52]

Total lung capacity (TLC) is the maximal volume allowed during inspiration and is divided into a number of subsets. Residual volume is the volume of air in the lungs after maximal expiration. Functional residual capacity (FRC) results from the balance between outward distention of the thorax and inward elastic recoil of the lungs. FRC is typically 50% of the total lung capacity in healthy children in upright position but decreases to 40% while supine.[52]

Infants have a smaller FRC, a factor that causes a greater and more rapid decrease in oxygen saturation with hypoventilation versus adults.[53] However, dynamic FRC is maintained in awake infants and young children by several mechanisms to prevent lung collapse. These mechanisms include sustained tonic activities of the inspiratory muscles throughout the respiratory cycle, narrowing of the glottis during expiration, inspiration starting at mid-expiration, and rapid respiratory rate in relation to the expiratory time rate.[52] Under the age of 6 years, closing capacity is greater than the FRC when supine.[53] Elevated closing capacity can lead to small airway collapse and decreased arterial oxygen saturation while in the supine position. Positive end

expiratory pressure (PEEP) is important in restoring FRC. Infants and children under the age of 3 greatly benefit from PEEP because it is thought to improve compliance by 75%.[52] General anesthesia, surgery, abdominal distention, and lung disease may all negatively affect lung volumes. When in supine or prone position versus sitting or standing, infants and children experience a reduction in FRC due to the cephalic shift of abdominal contents. The reduction in FRC is more profound in young infants and greatly reduces oxygen reserve.[54]

Ventilation Mechanics

The elastic properties of the lung and thorax are measured in terms of compliance which is expressed in units of volume change per units of pressure change. Expansion of the lung and chest wall by the inspiratory muscles is counteracted by elastic recoil of the lungs and thoracic cage. In general, pediatric patients have decreased lung compliance.[52] As a whole, compliance of the respiratory system increases in the first 12 months of life due primarily to an increase in lung compliance.[55] The infant chest wall, conversely, is highly compliant at birth and decreases over time. As such it provides minimal support to the noncompliant lungs at birth. Conversely, chest wall elastic recoil is low at birth and increases with age primarily due to ossification of the cartilaginous structures and development of intrathoracic muscles.[56] The combination of a highly compliant chest wall and poorly compliant lungs leads to increased work of breathing and decreased FRC. Therefore, neonates and infants are even more prone to atelectasis while under general anesthesia compared to children and adolescents.[57] Muscle relaxation due to general anesthesia and paralytic medications decreases FRC further.

Ventilation

Ventilation is the movement of air in and out of the lungs. The intercostal and accessory inspiratory muscles are utilized with maximum inspiratory effort, but the diaphragm is the most important muscle for normal, “quiet” inspiration. Expiration is considered passive and is due to diaphragm relaxation and elastic recoil of the lungs and chest wall.[52] However, expiration in the newborn is an active process, and involves use of the intercostal and particularly the abdominal muscles.[58] Tidal volume (V_t) is the amount of air moved in and out of the lungs with each breath. Minute ventilation (V_e) is the amount of air moved in and out of the lungs per minute of time. The frequency of quiet breathing decreases with increasing age.[52] Humans may adjust respiratory rate to minimize work, although the exact mechanism for this adjustment is unknown.[59]

Only part of the minute ventilation is used in effective gas exchange, referred to as alveolar ventilation (V_a). The rest is referred to as noneffective, dead space ventilation (V_d). Airway obstruction can increase dead space. Physiological dead space is influenced by the relative quality of gas distribution. Therefore, when gas exchange is uneven as it is, eg, in patients with cystic fibrosis, physiological dead space will increase. This is also the case when blood supply to an area or areas of the lung decreases; physiological dead space will increase as well.[52]

Anatomic dead space serves to warm and humidify air as it enters the lung. Both endotracheal intubation and tracheostomy negate these functions. The dead space in children and at adolescence mirrors body height. The dead space to tidal volume (V_d/V_t) in normal lungs is consistent from infancy to adulthood at 0.3. However, increased dead space is much more detrimental to infants than adults, and this is because an infant has a much smaller tidal volume and a much larger proportional increase in dead space.[52]

Ventilation is variably distributed to the lung from apex to base. At the end of expiration, alveolar pressure is zero (atmospheric). Intrapleural pressure is negative and increases from apex to base (more negative at the apex and less negative at the base). Due to this, transmural pressure is higher and regional FRC is larger at the apex than at the base. Consequently, at the end of inspiration a larger proportion of the inspired air will be distributed to the lung base.[52] Similarly, with adults in the lateral decubitus position, the dependent lung receives a greater proportion of the tidal volume.[60] The opposite is true of infants such that oxygenation and ventilation are improved in the upper or nondependent lung when in the lateral decubitus position.[61,62] In infants up to 27 months of age with or without lung disease, krypton-81m ventilation scans have shown that ventilation is preferential to the uppermost or apical portion of the lung.[61] This distribution of ventilation may be explained by premature airway closure at the lung base and due to the fact that the infant’s chest is more compliant, creating near atmospheric intrapleural pressure rather than negative intrapleural pressure.[62] In this situation, airway closure occurs and when in the lateral decubitus position, oxygenation and ventilation preferentially shift to the upper, nondependent lung.[63] When adults are paralyzed and mechanically ventilated, tidal ventilation is shifted to the uppermost part of the lung presumably due to similar reasons.[64]

For the pediatric anesthesiologist, it is important to be mindful that general anesthesia causes decreased FRC, contributes to uneven ventilation, and increases physiological dead space. Other factors to consider are the dead space and internal compliance of the anesthesia circuit, both of which increase dead space. This necessitates an increase in tidal volume with mechanical ventilation, perhaps using 10 to 12 mL/kg. An inspiratory to expiratory ratio of 1:2 is typical for pediatric patients. Respiratory rate should be set to 10 to 14 for adolescents, 14 to 20 for children, and 20 to 30 for infants as a starting point. Further refinement can be accomplished with capnography or arterial blood gas monitoring. The addition of low-level PEEP at 5 to 7 cm of H_2O should be used to restore FRC.[52,65]

Perfusion

In adults, perfusion is nonuniform from the apex to the base due to the effect of gravity on pulmonary blood flow. The same is true of ventilation in adults; the result is well-matched ventilation and perfusion. As mentioned previously, ventilation in infants is normally distributed to nondependent regions of the lung. Perfusion is more evenly distributed in the infant due to high pulmonary arterial pressure and also because the effect of gravity is lessened.[65,66]

West divided properties of upright lung perfusion into four zones.[67-69] The interrelationship of three pressures, alveolar pressure, pulmonary arterial pressure, and pulmonary venous pressure, determines the distribution of perfusion. In zone I, alveolar pressure exceeds both pulmonary arterial and pulmonary venous pressure. Therefore, alveolar capillary blood flow is absent and ventilation is wasted here. High PEEP can increase zone I and create excessive dead space ventilation. In zone II, pulmonary arterial pressure exceeds alveolar pressure and is referred to as the waterfall zone. Perfusion pressure in this zone is the difference between pulmonary arterial pressure and alveolar pressure, which then determines blood flow. Blood flow increases linearly toward the lung base, until the pulmonary venous pressure exceeds the alveolar pressure, as it does in zone III. Similarly, perfusion pressure in zone III is the difference between pulmonary arterial pressure and pulmonary venous pressure. Typically, perfusion pressure remains constant in zone III, but flow increases toward the base due to increases in both pulmonary arterial and venous pressures. In zone IV, blood flow decreases toward the base due to increased interstitial pressure surrounding the extraalveolar vessels. Zone IV increases in size with reduction in total lung volume near residual volume.[67-69,]

Ventilation and Perfusion

Normal alveolar gas exchange depends on the balance between regional ventilation and perfusion. A normal relationship of ventilation to perfusion (V/Q) is 0.8.[68] With a patient in upright position, both blood flow and ventilation is less at the apex and greater at the base. The difference in blood flow from apex to base is greater than the ventilation difference such that the V/Q ratio decreases from apex to base. In supine position, there is a similar but smaller change in ventilation and perfusion between anterior and posterior lung. In infants, ventilation is normally distributed to the nondependent areas of the lung and perfusion is more evenly distributed for reasons mentioned previously, resulting in higher V/Q.[69]

With pulmonary disease, V/Q mismatch occurs due to uneven ventilation, uneven perfusion, or both. Pulmonary embolism, reduced pulmonary capillary bed, and intrapulmonary/anatomic right to left shunting may all cause V/Q mismatch. In congenital heart disease (CHD) with increased pulmonary blood flow due to left to right shunting, V/Q is decreased. In CHD with decreased pulmonary blood flow due to right to left shunting V/Q is increased.[52]

There is an intrinsic mechanism in the lungs that functions to preserve V/Q matching. In areas of high V/Q and low pCO_2, the airways constrict and the vessels dilate. The opposite occurs in areas of low V/Q with high pCO_2. In addition, hypoxic pulmonary vasoconstriction (HPV) works to further constrict blood flow thereby increasing V/Q. Certain drugs themselves can diminish or abolish HPV including nitroprusside, nitroglycerin, isoproteronol, and inhaled anesthetics.[70–73]

PERIOPERATIVE CONSIDERATIONS FOR THORACIC SURGERY

A detail-oriented and complete preoperative evaluation is essential for the neonate, infant, or child who is undergoing thoracic surgery. Choice of the type of induction, that is, inhalational versus intravenous induction, should be carefully considered depending on the potential for difficult intubation and the presence of intrathoracic lesions that may cause compression of the trachea or major vascular structures. Inhalational induction with maintenance of spontaneous respiration is preferred when there is concern for compression of the airway and major vascular structures by large

intrathoracic masses. In this instance, availability of a rigid bronchoscope may also be useful if the trachea becomes compressed by an intrathoracic mass and if there is an inability to ventilate either before or after intubation. Intravenous induction may be preferred if there is concern for lung contamination. Placement of adequate intravenous access, possibly a central line, and an arterial catheter should be weighed in light of the type of procedure to be performed, that is, thoracoscopy versus thoracotomy, potential for blood loss, and the need to monitor hemodynamics and arterial blood gases during one lung ventilation and manipulation of the thoracic structures.[74]

Inhalational anesthetics are commonly used for the maintenance of anesthesia in thoracic surgery, but have the disadvantage of attenuating hypoxic pulmonary vasoconstriction. In general, nitrous oxide should be avoided due to risk of pneumothorax after thoracoscopy or thoracotomy. Total intravenous anesthesia can also be utilized and thereby one can avoid the potential deleterious effects of inhaled anesthetics on hypoxic pulmonary vasoconstriction. A variety of techniques to treat postoperative pain can be utilized after thoracoscopy or thoracotomy. Bupivacaine infiltration of incision sites is one approach. A variety of regional anesthetic techniques can be utilized, including paravertebral and intercostal nerve blocks and caudal or epidural catheter placement.[74]

THORACOSCOPIC PROCEDURES

Video-assisted thoracoscopic surgery (VATS) has become more common in infants and children due to the advantage of smaller incisions, reduced pain, and shortened hospitalization with faster recovery.[75,76] With this may come the expectation of one lung ventilation (OLV) and the likelihood should be discussed between surgeon and anesthesiologist on a case by case basis, including risks, benefits, and indications. OLV is desirable during thoracoscopy as lung deflation leads to better visualization of the thoracic cavity and potentially reduces the risk of damage to surrounding structures by retraction. Thoracoscopic procedures in infants and children include diagnostic inspection, lung biopsy, lobectomy, sequestration removal, lung decortication, thymectomy, esophageal atresia repair, aortopexy, mediastinal mass resection, patent ductus arteriosus ligation, and sympathectomy.[74] The absolute indications for OLV in adults and adolescents include the following:

1. Prevention of contamination of healthy lung by bleeding or infection
2. Control of ventilation in patients with bronchopleural fistula, unilateral bullae, or tracheobronchial disruption
3. Bronchoalveolar lavage such as with alveolar proteinosis

Relative indications include the following:

1. Surgical exposure—high importance
    a. Thoracic aortic aneurysm
    b. Pneumonectomy
    c. Upper lobectomy
2. Surgical exposure—low importance
    a. Middle and lower lobectomies
    b. Esophageal resection
    c. Thoracoscopy
    d. Anterior thoracic spine surgery

TECHNIQUES FOR SINGLE LUNG VENTILATION IN INFANTS AND CHILDREN

Single-Lumen Endotracheal Tube

The simplest method to provide OLV in children is to purposefully intubate the mainstream bronchus with a single-lumen endotracheal tube (ETT).[77] Several approaches can be used when the goal is to intubate the left mainstem bronchus. One method is to rotate the bevel of the ETT 180 degrees to the left and turn the child's head to the right.[78] The ETT is advanced until breath sounds are no longer heard on the right side. Placement is confirmed with a fiberoptic bronchoscope (FOB).[74] Another approach is to use fluoroscopic guidance.[79]

Several problems can occur with the use of single lumen ETTs for OLV. First, an uncuffed ETT can lead to significant leak if there is not an adequate seal with the bronchial wall. This can cause lack of collapse of the operative lung and possible contamination with blood or infectious material to the healthy, ventilated lung. Placement of a cuffed ETT in the main bronchus may result in obstruction of the right upper lobe orifice if the distance from the proximal cuff to the tip of the ETT is longer than the length of the right main stem bronchus. Hypoxemia can then occur due to obstruction of the upper lobe. This is more likely to occur on the right due to the short distance from the carina to the right upper lobe bronchus.[74]

Balloon-Tipped Bronchial Blockers

Bronchial blockade and OLV can be provided with a Fogarty embolectomy catheter or an end-hole, balloon wedge catheter.[80,81] The distal tip of a Fogarty catheter has a bendable tip which facilitates placement into the mainstem bronchus on the operative side. The FOB can be used to confirm and facilitate placement of the Fogarty catheter. There are a variety of ways to place the Fogarty catheter outside the ETT. One approach is to intubate a mainstem bronchus with a single lumen ETT and then place a guide wire inside the ETT. The ETT would then be removed and the Fogarty catheter could be threaded over the guide wire. A single lumen ETT could be reinserted into the trachea alongside the Fogarty catheter. The Fogarty catheter balloon would then be positioned in the proximal mainstem bronchus using the FOB for visualization.[80]

There is risk of dislodgment and occlusion of the trachea, which could prevent ventilation of the lungs bilaterally or prevent collapse of the operative lung. The balloons of most bronchial blockers are low compliance with low volume and high pressure. As such, overdistention of the balloon can lead to mucosal ischemia or airway rupture.[82] When the blocker is placed outside the ETT, it is important to be cognizant of the total airway diameter and injury that could be caused by compression of the tracheal mucosa by the ETT and bronchial blocker together. Therefore, the combined diameter of the bronchial blocker and outer diameter of the ETT should not exceed the expected tracheal diameter. Table 18-1 lists the diameters of pediatric sized uncuffed ETTs. These numbers estimate tracheal diameter, which is the predicted size of an uncuffed ETT that should produce a seal with the tracheal wall.[74] Cuffed tubes have an additional 0.5 mm outer diameter.

Table 18-1

Single-Lumen Uncuffed Tracheal Tube Diameters

Inner Diameter (mm)	Outer Diameter (mm)
3.0	4.3
3.5	4.9
4.0	5.5
4.5	6.2
5.0	6.8
5.5	7.5
6.0	8.2
6.5	8.9
7.0	9.6
7.5	10.2
8.0	10.8

Combination ETT and Bronchial Blockers

The Univent tube is an endotracheal tube with a second internal lumen housing a bronchial blocker. The blocker can be advanced into a mainstem bronchus.[83] A FOB must be used to facilitate placement. The bronchial blocker has an internal lumen and a balloon at the distal tip. Therefore, ventilation, suction, and continuous positive airway pressure (CPAP) can be applied to the operative lung. The blocker position within the outer ETT is very stable and dislodgment is rather unlikely.[74]

The Univent tube is available with lumen diameters as small as 3.5 and 4.5 mm for patients as young as approximately 6 years.[84] The internal area occupied by the bronchial blocker is rather high and consequently the outer diameter is quite large. For example, a Univent tube with a 3.5 mm internal luminal diameter corresponds to a 7.5/8.0 mm outer diameter (Table 18-2). As with the Fogarty catheter, the Univent balloon has low volume and high pressure such that overinflation can cause mucosal ischemia.[74]

Table 18-2

Univent Tube Diameters

Inner Diameter (mm)	Outer Diameter (mm)
3.5	7.5
4.5	8.5
6.0	10.0
6.5	10.5
7.0	11.0
7.5	11.5
8.0	12.0
8.5	12.5
9.0	13.0

Double-Lumen Tubes

The double-lumen tube (DLT) consists of two unequal length tubes molded together as one. The shorter tube is designed to occupy the trachea while the longer tube should reside in a mainstem bronchus. Each tube has a balloon at the distal tip. Typically, the endobronchial balloon resides on the distal tip of the bronchial lumen and is blue colored for easy identification with the FOB. DLTs have balloons with high compliance and low pressure, which minimizes pressure on the bronchial and tracheal wall.[74] With the endobronchial balloon inflated in the mainstem bronchus, ventilation of either lung in isolation or at the same time is possible. There are right- and left-sided DLTs. The right-sided DLT has a long tube designed to go into the right mainstem bronchus, whereas the long tube of the left-sided DLT should occupy the left mainstem. The right DLT endobronchial balloon has a donut shape designed to allow right upper lobe ventilation. However, the right upper lobe orifice may still become occluded by the endobronchial balloon despite it altered design.[85] The smallest DLT size available in the United States is 26F, which is designed for children 8 years of age. There are also 28F and 32F DLTs that accommodate children 10 years and older. Adults' sizes range from 35 to 41F.[74] Table 18-3 lists the outer diameters of the various sizes of DLTs.

Table 18-3

Double-Lumen Tube Diameters

Size (French)	Main Body Outer Diameter (mm)
26	8.7
28	9.4
32	10.6
35	11.7
37	12.4
39	13.1
41	13.7

The same method used to insert a DLT into an adult can be used for a child. The tip of the tube is inserted just past the vocal cords and the tube is then rotated 90 degrees to the operative side and advanced in the trachea. Breath sounds should then be auscultated to confirm placement. With the tracheal cuff inflated, breath sounds should be auscultated on both sides. With the bronchial cuff inflated, breath sounds should be heard on both sides to ensure the bronchial cuff has not herniated across the trachea. Fiberoptic bronchoscopy can also be performed through the tracheal lumen to confirm placement of the bronchial cuff in the right or left mainstem bronchus. Additionally, the tracheal lumen can be clamped and breath sounds auscultated. When a left-sided DLT has been properly positioned, breath sounds should be heard on the left only when the tracheal lumen clamped. The opposite holds true for a right-sided DLT.[74]

One major disadvantage of the DLT is the possibility of the need for continued controlled ventilation in the postoperative period. Therefore, exchange to a single lumen endotracheal tube is necessary. This is of particular concern when the initial intubation was challenging. Further, reintubation may become challenging after surgery due to swelling, secretions, and blood in the airway or trauma from the first intubation. An exchange catheter may facilitate the exchange for a single lumen tube.[86] An additional advantage is that exchange catheters can be used for oxygen insufflation and jet ventilation.

MANAGEMENT OF SINGLE-LUNG VENTILATION

Once proper position of a SLT, bronchial blocker or DLT is confirmed, it is necessary to determine the peak airway pressures required for OLV. In general, peak airway pressures should be minimized and not exceed 40 cm of water (H_2O) during OLV. Once lateral decubitus positioning is obtained, position of the bronchial blocker, SLT, or DLT should be reconfirmed with fiber-optic bronchoscope as the position can be disrupted with patient positioning. After transition to OLV, an inspired oxygen concentration of 100% is typically necessary. The goal partial pressure of oxygen (PaO_2) should be approximately 100 to 200. Tidal volumes should be 8 to 10 mL/kg and respiratory rate should be adjusted to maintain a partial pressure of carbon dioxide ($PaCO_2$) at a range of 45 to 60, unless the patient's physiology cannot tolerate hypercapnia.[87]

If hypoxemia occurs during OLV, several steps can be undertaken. First the patient can be manually ventilated and placed on 100% oxygen. To determine if the tube or endobronchial blocker is in correct position, FOB may then be undertaken. Several measures that may improve oxygenation include application of continuous positive airway pressure to the nondependent lung if this is tolerable to the surgeon and use of PEEP to the dependent lung. One further option is to occlude the pulmonary artery to the operative lung to decrease shunt. If there is persistent hypoxemia despite these measures, return to two-lung ventilation is imperative.

Appropriate tube selection for OLV in children can be predicted using Table 18-4. There is great variability in size among the different age groups in the pediatric population and these recommendations are based upon average airway dimensions.

Table 18-4

Recommended Tube Sizes for One Lung Ventilation in Pediatric Patients

Age (yr)	Endotracheal Tube Inner Diameter (mm)	Bronchial Blocker (French)	Univent (mm)	Double-Lumen Tube (French)
0.5–1	3.5–4.0	2		
1–2	4.0–4.5	3		
2–4	4.5–5.0	5		
4–6	5.0–5.5	5		
6–8	5.5–6.0	5	3.5	
8–10	6.0	5	3.5	26
10–12	6.5	5	4.5	26–28
12–14	6.5-7.0	5	4.5	32
14–16	7.0	5, 7	6.0	35
16–18	7.0–8.0	7, 9	7.0	35, 37

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SURGICAL LESIONS OF THE THORAX

Neonates and Infants

Congenital Cystic Lesions

Congenital cystic lesions encompass various thoracic lesions that can be further subdivided into four groups: bronchogenic cysts, cystic adenomatoid malformations, congenital lobar emphysema, and bronchopulmonary sequestration. The presentation of these lesions may vary. Each lesion can be prenatally diagnosed and treated with fetal surgery. Though the rate of occurrence is only 5 per 10,000 fetuses, there is significant morbidity and mortality associated with these lesions.[88]

Bronchogenic cysts are also known as foregut duplications with 85% occurring in the mediastinum and 15% intrapulmonary. They can be filled with air or fluid and can be central or peripheral. Most are asymptomatic.[89]

Congenital cystic adenomatous malformations (CCAM) are intrapulmonary lesions that result from abnormal lung development of the terminal bronchioles. They are characterized by multicystic areas of overproliferation and dilation of terminal respiratory bronchioles that lack normal alveoli.[90] They are unilobar, unilateral, and account for 25% of all congenital lung malformations. Almost all are detected on antenatal ultrasound by 20 weeks.

Congenital lobar emphysema results from weakened or absent bronchial cartilage that permits focal bronchial collapse. This results in a ball-valve phenomenon which traps air, causing hyperinflation of the affected lobe. Causes of ball-valve phenomenon can be intrinsic (dysplastic bronchial cartilage, intraluminal obstruction) or extrinsic from vascular abnormalities such as anomalous pulmonary vascular return or pulmonary artery sling. This can result in contralateral mediastinal shift, decreased cardiac output, and ensuing hypoxia and hypotension. It occurs 1 in 20,000 births with a 3:1 ratio in males versus females. Fifty percent of the cases involve the upper lobe of the left lung. Although less severe cases may not initially present until childhood, most cases are encountered in the neonatal period. The neonatal cases may present with acute respiratory distress that necessitates surgical intervention and resection of the affected lobe. A total of 15% to 20% of neonates have concurrent congenital heart lesions involving atrial or ventricular septal defects or pulmonary hypertension.[91]

Surgical resection is recommended for congenital cystic lesions to avoid potential infection, hemorrhage, and respiratory complications. Induction and intubation are critical events and should be planned to avoid Valsalva maneuver that would increase air trapping. An inhalation induction and maintenance of anesthesia that preserves spontaneous ventilation until thoracotomy is preferred. Gentle manual ventilation with inspiratory pressures less than 20 cm H_2O may be required to maintain oxygenation. Nitrous oxide is contraindicated. Selective endobronchial intubation and high-frequency ventilation are additional options for ventilation until thoracotomy is achieved. Due to the possibility of lung overinflation on induction, a surgeon should be present in the event a thoracotomy is needed to relieve the overdistention.[90,92]

Bronchopulmonary sequestration is an embryonic mass of lung tissue that is isolated from neighboring lung tissue and has no connection to the bronchial tree and is nonfunctioning: 75% intralobar with the remaining 25% being extralobar. It receives its blood supply from an aberrant branch of the descending aorta and should be resected since heart failure can result from its large blood flow.[93]

Childhood

Anterior Mediastinal Masses

While the occurrence of anterior mediastinal masses is uncommon in children, anesthesia for such cases can be very challenging and fraught with hazard. The anesthetic plan will be dependent on the pathology, specific location, procedure, and age of the patient. Information obtained from the patient's history, exam, and radiological workup will guide and determine the anesthetic plan.

The anterior mediastinal space is defined by the sternum anteriorly, sternomanubrial junction superiorly, pericardium posteriorly and diaphragm inferiorly, and the parietal pleura laterally. Masses of the mediastinum encompass tumors that are both benign and malignant. While anterior mediastinal masses are predominantly lymphomas in origin, other less common lesions include thymomas, teratomas, lipomas, and cystic lymphatic malformations.[94]

A retrospective chart review by Garey et al demonstrated that a mean age of 11 years had poor correlation of symptoms with anatomical airway obstruction or complications.[95] Large masses can present without any airway compression.[96] King et al reported no statistically significant association between degree of compression/clinical symptoms and size of tumor.[97] Treatment plans rely on tissue diagnosis and therefore patients will present for various procedures including CT-guided needle biopsy, superficial lymph node biopsy, VATS, or mediastinoscopy. Any one of these procedures could require either sedation or a general anesthetic.

Any anesthetic should be well-planned in advance and undoubtedly will involve the expertise of the multiple care teams. In published studies, the overall complication rate in patients with mediastinal masses was 20%, and 5% of patients had a serious complication related to anesthesia.[98] There are numerous reports of sudden refractory cardiorespiratory collapse during induction or maintenance of anesthesia in both symptomatic and asymptomatic patients.[99] As such, multiple backup plans must be established prior to induction and appropriate equipment must be readily available along with adequate support staff.

Since pulmonary and cardiac symptoms may significantly contribute to or help predict complications, proceeding forward with an anesthetic is best guided by the patient's history, physical exam, and radiological/diagnostic information. Any preoperative evaluation should include a review of the patient's symptoms. Important factors for consideration include the effect of position on signs and symptoms such as dyspnea, stridor, dysphagia, chest pain/fullness, cough, or syncope.[94] Of the aforementioned symptoms, stridor was the only sign that predicted an anesthetic complication in a published study.[98] Imaging such as a chest x-ray and computed tomography (CT) will also provide useful information. CT imaging can provide

adequate images in approximately 20 seconds. The patient's head may be elevated during a CT scan to 30 degrees without compromising scan quality.[100] A greater than 50% decrease in the diameter of the tracheobronchial lumen may be associated with complete airway obstruction during the induction or emergence from general anesthesia.[101] Echocardiography may be used to evaluate cardiovascular compromise. In addition, awake bronchoscopy and flow-volume studies can provide information regarding airway dynamics and compression.

Mediastinal tissue biopsy can be performed with the application of EMLA cream, local anesthetics, and sedation with ketamine (0.5–1 mg/kg). Antisialagogue agents are useful to decrease salivation caused by ketamine. Ketamine is particularly useful for sedation in patients with hemodynamic compromise. Dexmedetomidine may also provide analgesia and amnesia while maintaining spontaneous respirations. Sedative agents such as benzodiazepines and opioids can cause undesired muscle relaxation and respiratory depression, respectively.[98] If a general anesthetic is required for either tissue biopsy or definitive surgery, induction of anesthesia may be accomplished with inhalational agents, etomidate, propofol, and/or ketamine. Spontaneous ventilation is the safest strategy to prevent complete airway collapse with institution of positive pressure ventilation. Muscle relaxants should be utilized only when positive pressure ventilation has been documented with the current airway in place. One approach to intubation would be an awake fiberoptic intubation with subsequent use of an armored endotracheal tube placed distal to the compression or a double lumen tube where the bronchial lumen is placed distal to the obstruction. If during the operative procedure ventilation is severely compromised, the anesthetic can be reversed to awaken the patient. Alternatively, the patient can be repositioned to either the prone or lateral position. Finally, the airway can be splinted with a rigid bronchoscope to ventilate distal to the obstruction. Sternotomy or thoracotomy with retraction of the mass from the airway and great vessels has also been utilized as an option. While cardiopulmonary bypass has been described as an option prior to the start of any procedure in a patient with an anterior mediastinal mass,[102] the logistics of instituting bypass once compromise has occurred may result in prolonged hypoxia and subsequent cardiac arrest.

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