

INTRODUCTION

Perioperative pulmonary complications contribute significantly to overall perioperative morbidity and mortality rate. Such complications account for about 25% of deaths occurring within 6 days of surgery. The goal of perioperative pulmonary management is to identify patients at high risk of significant postoperative pulmonary complications so that appropriate interventions can be provided to minimize that risk.

In most cases even in high risk patients, the procedure can be performed safely as planned but occasionally postponement, modifications or cancellations are warranted. The risk of postoperative pulmonary complications varies with the type of surgery being performed.

Pulmonary complications occur much more often than cardiac complications in patients undergoing elective surgery to the thorax and upper abdomen. Operation at sites farther from the diaphragm are associated with a much lower incidence of postoperative pulmonary complication.

Postoperative pulmonary complications are also more common in patients with preexisting lung disease, medical comorbidities, poor nutritional status, overall poor health and smokers

Not all of these risk factors are modifiable although strategies exist to reduce postoperative pulmonary complications even among high risk patients.

Chapter (1)

ANATOMICAL AND PHYSIOLOGICAL REVIEW

The principal function of the lungs is to transport the respiratory gases into and out of the blood stream. In the human lung, this is done by a remarkable system of air and blood – containing vessels, folded together in an elastic structure in such a way that these vessels are in intimate contact, yet their contents do not physically mix (*Scharf, 1998*).

The normal adult human requires to transfer about 200-250 ml /min of oxygen into the body at rest. However the transport of oxygen can increase to 3000 – 4000 ml/min at maximum exercise. In diseased lungs, oxygen transport capacity can be limited which in turn limit the transport of oxygen into the peripheral tissue, the ultimate target (*Scharf, 1998*).

The pulmonary vascular bed resemble the systemic bed except that the walls of pulmonary artery and its large branches are about 30% as thick as the wall of the aorta, and the small arterial vessels, unlike the systemic arterioles, are endothelial tubes with relatively little smooth muscles in their walls. There is also some smooth muscles in the walls of the post capillary vessels. The pulmonary capillaries are large and there are multiple anastomosis so that each alveolus sits in a capillary basket (*Ganong, 1989*).

The pulmonary artery extends only 5 centimeters beyond the apex of the right ventricle and then divide into right and left main branches, which supply blood to the two respective lungs. All the pulmonary arteries and arterioles have much larger diameter than their counterpart systemic arteries. The pulmonary vessels are very thin and distensible, giving the pulmonary arterial tree a very large compliance, averaging almost 7 ml/mmHg. This large compliance allows the pulmonary arteries to accommodate about two thirds of the stroke volume output of the right ventricle (*Guyton, 1991*).

The pulmonary veins, like the pulmonary arteries are also short, but their dispensability characteristics are similar to those of the veins in the systemic circulation (*Guyton, 1991*).

Anatomy of the lungs

The lungs arise during development as epithelial outgrowth of the anterior foregut forming a system of tubes which become invested by mesenchymal tissues rich in blood vessels. This developing mass of tissue is lined externally by mesothelium which eventually forms the lining of the serous coat of the pulmonary pleura .Within the lung, the proximal parts of the tubular system become the bronchial tree and the more distal ones expand to form the cavities across the walls of which respiratory exchange takes place (*McMinn, 1994*).

Each lung is conical in shape and is covered by a visceral pleura, being attached to the mediastinum by its root. Each lung has an apex upward into the root of the neck 2-3 cms above the midclavicular point, a concave base rests on the dome of the diaphragm, a convex costal surface related to the ribs and costal cartilages and intercostals spaces, a concave mediastinal surface which is molded to the mediastinal structures (*McMinn, 1994*).

Each lung also has 3 borders: anterior border is thin and sharp, lies between the pericardium and chest wall and on the left side the lower part of this border is concave to form the cardiac notch, the posterior border is rounded to fit the paravertebral gutter and is continued up to the apex, the inferior border is sharp and separates the costal surface from the base (*McMinn, 1994*).

Anatomy of the pulmonary circulation

Pulmonary circulation was first discovered and published by "Ibn Nafis" in his Commentary on anatomy in Avicenna's Canon (1242), for which he is considered the father of circulatory physiology.

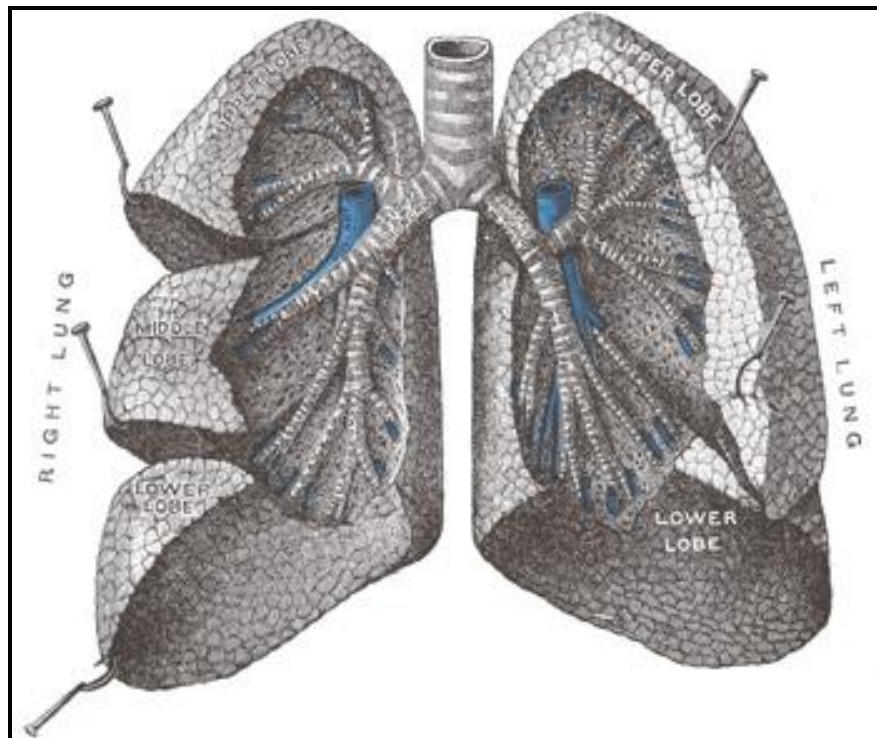


Fig. (1): human lung (Gray, 1918).

The pulmonary arteries

The **pulmonary artery** conveys the venous blood from the right ventricle of the heart to the lungs. It is a short, wide vessel, about 5 cm. in length and 3 cm. in diameter, arising from the conus arteriosus of the right ventricle. It extends obliquely upward and backward, passing at first in front and then to the left of the ascending aorta. The right branch of the pulmonary artery (ramus dexter a. pulmonalis) is longer and larger than the left, runs horizontally to the right, behind the ascending aorta and superior vena cava and in front of the right bronchus, to the

root of the right lung, where it divides into two branches. The lower and larger of these goes to the middle and lower lobes of the lung; the upper and smaller is distributed to the upper lobe. The left branch of the pulmonary artery (ramus sinister a. pulmonalis) is shorter and somewhat smaller than the right, passes horizontally in front of the descending aorta and left bronchus to the root of the left lung, where it divides into two branches, one for each lobe of the lung as shown in (Figure 2) (*Gray, 1918*).

The Pulmonary Capillaries:

Capillaries account for most of the vasculature in the lung and have a distinct appearance, regardless of their size. They do not change caliber with distance or branching and have no directionality. They branch sharply, at nearly right angles, which facilitate the slow lateral flow of blood as it exchanges gases and solutes. Right-angle branching conserves pressure in a low-pressure system such as the pulmonary circulation and can cause significant focal variation in hematocrit. Capillaries may attach directly to large veins but not to arteries. The capillaries on the pleural surface, around bronchioles, and in the bronchovascular bundle are less dense, making up about 67% of the surface. They are planar and branch less often. Pleural capillaries are less likely than alveolar capillaries to have flattened and widened areas at bends (*Schraufnagel and Schmid, 1988*).

The Pulmonary Veins:

The pulmonary veins commence in the pulmonary capillaries, the radicals coalescing into larger branches which run through the substance of the lung, independently of the pulmonary arteries and bronchi. Eventually, two venous trunks emerge at each hilum to enter, after a short extra-pulmonary course, the upper part of the left atrium (*Plank et al., 1979*).

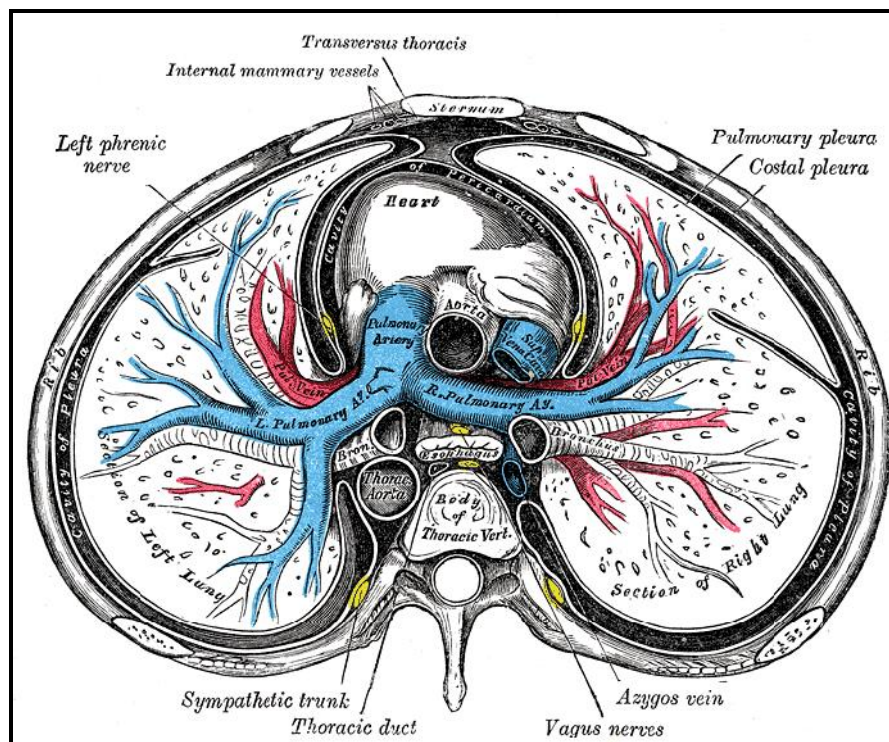


Fig. (2): Transverse section of the thorax (*Gray, 1918*).

Pulmonary physiology

Lung volumes and capacities

Lung volumes are important parameters in respiratory physiology and clinical practice. The sum of all the named lung volumes equals the maximum to which the lung can be inflated.

Lung capacities are clinically useful measurement that represents a combination of two or more volumes (*Morgan et al., 2006*).

The amount of air that moves into the lung with each inspiration (or the amount of air that moves out with each expiration) is called the tidal volume. The air inspired with a maximal inspiratory effort in excess of the tidal volume is called the inspiratory reserve volume. The volume expelled by an active expiratory effort after passive expiration is called the expiratory reserve volume (Fig 3) (*Ganong, 2005*).

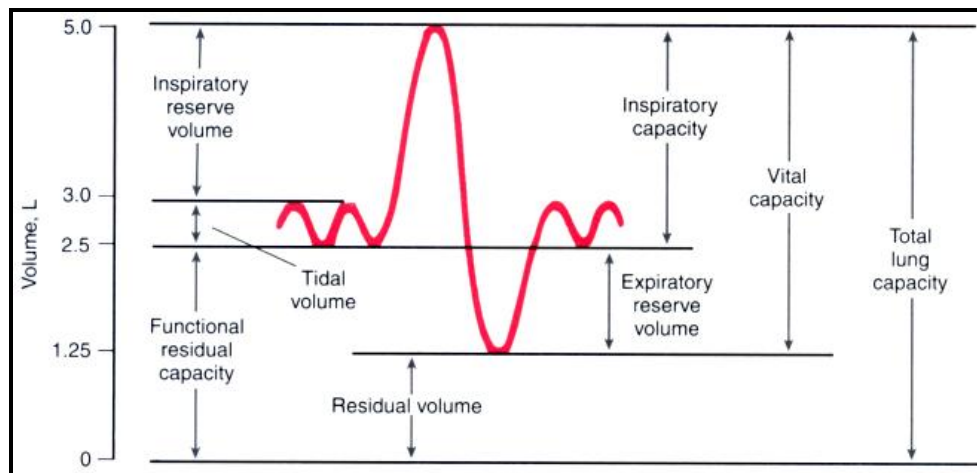


Fig. (3): The dynamic lung volumes that can be measured by simple spirometry are tidal volume, inspiratory reserve volume, expiratory reserve volume, inspiratory capacity, and vital capacity. The static lung volumes are residual volume, functional residual capacity, and total lung capacity. Static lung volumes cannot be measured by simple spirometry and require separate methods of measurement (e.g., inert gas dilution, nitrogen washout, or whole-body plethysmography) (*Miller, 2005*).

Physiological dead space:

It comprises both anatomical and alveolar dead spaces.

Anatomical dead space is the gas in the conducting areas of the respiratory system, such as the mouth and trachea, where air does not come into contact with the alveoli of the lungs. It is normally equal in milliliters to body weight in pounds. A 150 lb (68 kg) male would have an anatomical dead space of about 150 mL. 1 mL per lb or 2.2 mL per kilogram of body weight. This is about a third of the resting tidal volume (450-500 mL). Anatomic dead

space is the volume of the conducting airways. It may be measured by Fowler's method, a nitrogen washout technique (*Heller et al., 1999*).

Functional Residual capacity (FRC):

The lung volume at the end of a normal exhalation is called functional residual capacity (FRC). At this volume, the inward elastic recoil of the lung approximates the outward elastic recoil of the chest (including the resting diaphragmatic tone), thus, the elastic properties of both chest and lung define the point from which normal breathing takes place (*Morgan et al., 2006*).

Closing volume is defined as the lung volume at which small airways begin to close in the dependent zones of the lung. This is the volume of gas that can be breathed out to the residual volume after the onset of airway closure. The total volume of gas within the lung at the onset of airway closure is the closing capacity (Which equals closing volume + RV) (*Dosman et al., 1997*).

Effects of age and posture on functional residual capacity (FRC) and closing capacity (CC):

Normally CC becomes equal to FRC at the age of 66 years in the up-right position and at age 44 at supine position. FRC increases approximately 30% by changing

from supine to up-right position. The CC on the other hand is independent on body position (*Miller, 2005*).

Effects of anesthesia on FRC and CC:

During anesthesia, FRC is reduced approximately 20% with spontaneous breathing and about 16% with artificial ventilation. This is due to changes in the chest wall shape and diaphragm position. After induction of general anesthesia, a reduction occurs in the cross sectional area of the rib cage corresponding to a decrease in lung volume of about 200 ml. recent studies have consistently shown a cephalad movement of the dependent regions of the diaphragm, with little or no movement of non dependent regions. However, the changes in FRC that can be related to changes in diaphragm is on average less than 30 ml (*Miller, 2005*).

CC was previously reported to be unchanged during anesthesia, but later studies concluded that CC has reduced in parallel with FRC during anesthesia. First, when the FRC is decreased to below CC, airways close in the dependent parts of the lung during certain periods of normal tidal ventilation. Airway closure results in shunting of pulmonary blood flow through the unventilated alveoli. Therefore, arterial oxygenation is decreased. Second, pulmonary circulation and alveolar gas exchange are continuous during both inspiratory and expiratory phases of respiration. Whether or not there is airway closure, blood

oxygenation during the expiratory phase is mainly dependent on the remaining lung volume, which is FRC. Therefore, when FRC is high, blood oxygenation is better and there is more time for oxygenation before hypoxia occurs during apnea. FRC is decreased in the supine position during general anesthesia and in patients with acute respiratory distress syndrome. Positive end expiratory pressure (PEEP) increases FRC and decreases airway closure (*Miller, 2005*).

Vital capacity:

Vital capacity (VC) is the maximum volume of gas that can be exhaled following maximal inspiration. In addition to body habitus, VC is also dependant on respiratory muscle strength and chest wall and lung compliance. Normal VC is about 60-70 ml/kg (*Morgan et al., 2006*).

Factors affecting vital capacity:

1. Alteration in muscle power by drugs of central effect or affecting neuromuscular junction or neurological disease as poliomyelitis or lesions at the level of neuromuscular junction as myasthenia gravis. All these would reduce the vital capacity.
2. Pulmonary disease: especially chronic bronchitis and pneumonia will reduce vital capacity markedly.

3. Space occupying lesion in the chest: such as tumors, pericardial and pleural effusion or pneumothorax. All these would reduce vital capacity.
4. Increased intra abdominal pressure: as in intestinal obstruction and abdominal tumors which impede the descent of the diaphragm.
5. Abdominal pain: post-operative pain after abdominal operations leads to reduction of the vital capacity by 70-75% in the upper abdominal operations and by 5% in the lower abdominal operations
6. Alterations in posture: the change in vital capacity is due to alteration in the volume of blood in the lungs.
 - Trendlenburg position 14.5%
 - Lithotomy position 18%
 - Left lateral position 10%
 - Right lateral position 12%
 - Bridge in dorsal position 12.5%
 - Prone position 10% (*West, 2004*).

Table (1): Lung Volumes and Capacities

Measurement	Value (Male/Female)	Calculation	Description
Total lung capacity (TLC)	= 6.0 / 4.7 L	$TLC = IRV + V_t + ERV + RV$	The volume of air contained in the lung at the end of maximal inspiration. The total volume of the lung.
Vital capacity (VC)	= 4.6 / 3.6 L	$VC = IRV + V_t + ERV$	The amount of air that can be forced out of the lungs after a maximal inspiration. Emphasis on completeness of expiration. The maximum volume of air that can be voluntarily moved in and out of the respiratory system.
Forced vital capacity (FVC)	= 4.8 / 3.7 L	measured	The amount of air that can be maximally forced out of the lungs after a maximal inspiration. Emphasis on speed.
Tidal volume (V_t)	= 500 / 390 mL	measured	The amount of air breathed in or out during normal respiration. The volume of air an individual is normally breathing in and out.
Residual volume (RV)	= 1.2 / 0.93 L	measured	The amount of air left in the lungs after a maximal exhalation. The amount of air that is always in the lungs and can never be expired (i.e.: the amount of air that stays in the lungs after maximum expiration).
Expiratory reserve volume (ERV)	= 1.2 / 0.93 L	measured	The amount of additional air that can be pushed out after the end expiratory level of normal breathing. (At the end of a normal breath, the lungs contain the residual volume plus the expiratory reserve volume, or around 2.4 litres. If one then goes on and exhales as much as possible, only the residual volume of 1.2 litres remains).
Inspiratory reserve volume (IRV)	= 3.0 / 2.3 L	measured or $IRV = VC - (V_t + ERV)$	The additional air that can be inhaled after a normal tidal breath in. The maximum volume of air that can be inspired in addition to the tidal volume.
Functional residual capacity (FRC)	= 2.4 / 1.9 L	$FRC = ERV + RV$	The amount of air left in the lungs after a tidal breath out. The amount of air that stays in the lungs during normal breathing.
Inspiratory capacity (IC)	= 3.5 / 2.7 L	$IC = V_t + IRV$	The maximal volume that can be inspired following a normal expiration.
Anatomical dead space	= 150 / 120 mL	measured	The volume of the conducting airways. Measured with Fowler method.
Physiologic dead volume	= 155 / 120 mL	$V_T \frac{P_{A\text{CO}_2} - P_{E\text{CO}_2}}{P_{A\text{CO}_2}}$	The anatomic dead space plus the alveolar dead space.

Quoted from (Palsson et al., 2003)