Introduction

Postoperative pulmonary complications (PPCs) are one of the most common perioperative adverse events in patients undergoing surgery and contribute to significant increases in morbidity, mortality, and length of postoperative hospital stay **(Jeong et al., 2014).**

The aim of this review is to describe the current evidence underpinning our understanding of PPCs and highlight measures that might become necessary at different points during the course of perioperative care **(Canet and Mazo, 2010).**

The reasons for the occurrence of these complications are multifactorial. The surgery, anaesthesia method that is applied and preoperative risk factors of the patients play an important role, obesity, smoking, age, current chronic lung diseases and other comorbidities are patient risk factors. Apart from these, whereas anaesthesia type, duration, use of different agents and efficacy of postoperative pain treatment are anaesthesia-related risk factors, intervention time, surgical techniques and incision size represent surgical risk factors **(Saracoğlu et al., 2014).**

Interventions aimed at decreasing pulmonary complications associated with anesthesia should begin prior to operation and continue throughout the perioperative periods. These interventions should be carried out regardless of the risk of development of postoperative pulmonary complications **(Bapoje et al., 2007).**

General prevention strategies include smoking cessation, mechanical ventilation strategies, Pulmonary hygiene, early ambulation, anesthetic and analgesic technique, prevention of gastric-to-pulmonary aspiration, management of intravenous fluids, avoiding hyperoxia, management of neuromuscular blockade, use of postoperative pulse oximetry, minimizing perioperative sedation, prevention of Transfusion related acute lung injury (TRALI), and Prevention of ventilator-associated pneumonia (VAP) **(Bohman and Kor, 2014).**

Urea dosage above 21 mg dL and serum albumin below 3.5 g dL were predictors of PPCs, particularly pneumonia and acute respiratory failure in postoperative noncardiac surgery. Perioperative mortality was also higher in patients with serum creatinine greater than 1.5 g dL,due to both pulmonary and cardiovascular and hemorrhagic and infectious adverse events **(Degani-Costa et al., 2014).**

Aim of the Work

This work discusses the important risk factors to identify during a preoperative pulmonary evaluation and then focuses on recent advances in strategies for reducing postoperative pulmonary complications.

Chapter 1
Functional anatomy of the respiratory system

The respiratory passages are functionally divided into upper and lower respiratory tracts. The upper respiratory tracts composed of oral and nasal cavities, the pharynx and the larynx. The lower respiratory tracts composed of tracheobronchial tree and lung parynchyma **(Sircar, 2008)**. The upper airways comprise the nose, the nasopharynx and the oropharynx. Their relative positions are indicated in Fig.1.

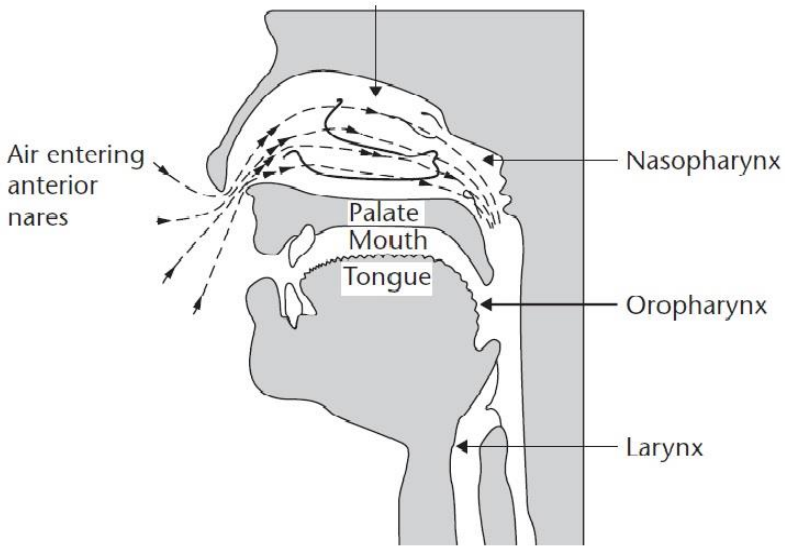

Fig.1 Functional anatomy of the nose. The small cross-sectional area of the anterior nares results in a high linear velocity (indicated by arrows). This leads to the larger particles impacting on the anterior tip of the turbinate bones (**Cotes et al., 2006)**.

The main function of the upper airways is to condition the air that is inspired. In addition, the pharynx and epiglottis are adapted to compensate for the defect in design that led to the conduits to the lungs and stomach crossing over in the pharynx instead of being separate **(Cotes et al., 2006)**.

The intrinsic muscles of the larynx are broadly divided into abductors and adductors supplied by the recurrent laryngeal branch of the vagus nerve, paralysis of the abductor muscles leads to inspiratory stridor. In anesthetized patients, glottic closure may be incomplete and vomitus may enter the trachea, causing aspiration pneumonia. The adductor muscles main function is protective. During swallowing there is a reflex contraction of the adductor muscles that closes the glottis and prevent aspiration. Another protective function of the glottis is its role in cough reflex (**Sircar, 2008)**.

The lower airway consists of the lower trachea and the lung parenchyma. The lung parenchyma can be subdivided into three airway categories based on functional lung anatomy, the *conductive* airways provide basic gas transport but no gas exchange, the next group, which has smaller diameters, is the *transitional* airways, they are conduits for gas movement, and additionally perform limited gas diffusion and exchange, finally, the smallest *respiratory*

airways primary function is gas exchange (**Barash et al., 2013)**.

The respiratory bronchiole, which follows the terminal bronchiole is the first site in the tracheobronchial tree where gas exchange occurs. In adults, two or three generations of respiratory bronchioles lead to alveolar ducts, of which there are four to five generations, each with multiple openings into alveolar sacs. The final divisions of alveolar ducts terminate in alveolar sacs that open into alveolar clusters (**Connelly and Silverman, 2009).**

The acinus is a structural unit of the lung distal to a terminal bronchiole and is supplied by first order respiratory bronchioles it contains alveolar ducts and alveoli. It is the largest unit in which all airways participate in gas exchange and is approximately 6-10 mm in diameter. One secondary pulmonary lobule contains between three and 25 acini (**Hansell et al., 2010)**.

The alveoli are surrounded by pulmonary capillaries, lined by two types of epithelial cells or pneumocytes. *Type I pneumocytes* (squamous alveolar cells) are the primary lining cells. *Type II pneumocytes* (granular alveolar cells) are thicker and contain numerous lamellar inclusion bodies. These cells secrete surfactant. The lung also contain alveolar

macrophages or the dust cells, lymphocytes, plasma cells, Clara cells, APUD cells, and mast cells (**Sircar, 2008)**.

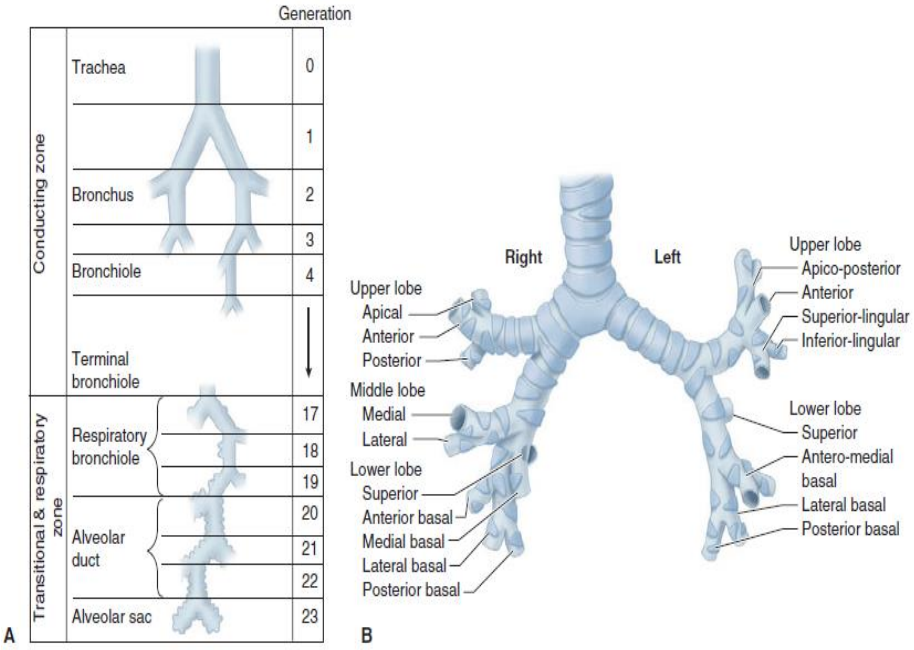

Fig.2 A: Dichotomous division of the airways.

B: The segmental bronchi (**Butterworth et al., 2013)**

The pulmonary vasculature is engineered to ensure a high compliance, low resistance network that provides an extensive surface area for gas exchange. As in systemic vascular beds, it is composed of three vascular compartments connected in series: arteries, capillaries and veins (**Townsley, 2011).**

The pulmonary circulation begins at the main pulmonary artery, which receives mixed venous blood pumped by the right ventricle, then branches successively

like the system of airways as far as the terminal bronchioles (**West, 2011).**

The pulmonary capillaries form a dense network in the alveolar wall that makes an exceedingly efficient arrangement for gas exchange, the individual capillary segments are so short that the blood forms continuous sheet. The oxygenated blood is then collected from the capillary bed by the small pulmonary veins which run between the lobules and eventually unite to form four pulmonary veins which drain into the left atrium (**Behera, 2010).**

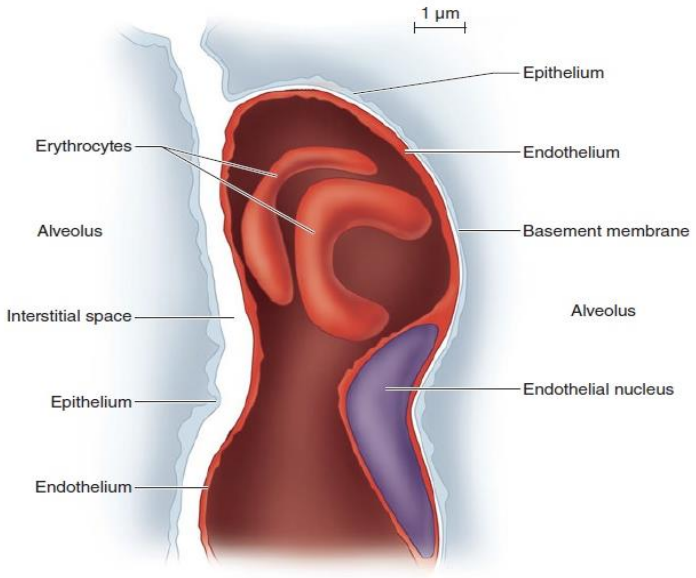

Fig.3 The pulmonary interstitial space, with a capillary passing between the two alveoli. The capillary is incorporated into the thin (gas-exchanging) side of the alveolus on the right. The interstitial space is incorporated into the thick side of the alveolus on the left (**Butterworth et al., 2013).**

Lymphatic vessels are found superficially around the lungs just beneath the visceral pleura and in the dense connective tissue wrapping of the bronchioles, bronchi, pulmonary arteries, and pulmonary veins. The primary function of the lymphatic vessels is to remove excess fluid and protein molecules that leak out of the pulmonary capillaries. Deep within the lungs, the lymphatic vessels arise from the loose space of the interstitium. The vessels follow the bronchial airways, pulmonary arteries, and veins to the hilum of the lung, and end in the pulmonary and bronchopulmonary lymph nodes **(Des Jardins, 2013).**

Physiology of the Respiratory System

1. Respiratory mechanics:

The movement of the lungs is passive and determined by the impedance of the respiratory system, which can be divided into the elastic resistance of tissues and the gas–liquid interface and the non-elastic resistance to gas flow. Elastic resistance governs lung volume and the associated pressures under static conditions (no gas flow). Resistance to gas flow relates to frictional resistance to airflow and tissue deformation. The work necessary to overcome elastic resistance is stored as potential energy, but the work necessary to overcome non-elastic resistance is lost as heat (**Butterworth et al., 2013).**

A - Elastic recoil of the lung and chest wall

Movement of the chest wall by the muscles of ventilation generates pressure gradient between the alveoli and the surrounding air, enabling the gas to move in and out. Two major factors responsible for the elastic recoil of the lung: (1) lung connective tissue - collagen and elastin - and (2) surface tension related to the air-liquid interface of the alveolar surface (**Broaddus et al., 2015)**.

B - Pulmonary compliance

The extent to which the lungs will expand for each unit increase in transpulmonary pressure. The total compliance of both lungs together in the normal adult human averages about 200 milliliters of air per centimeter of water transpulmonary pressure. That is, every time the transpulmonary pressure increases 1 centimeter of water, the lung volume, after 10 to 20 seconds, will expand 200 milliliters.

Graphically Figure.4 is a diagram relating lung volume changes to changes in pleural pressure, The two curves are called, respectively, the *inspiratory compliance curve* and the *expiratory compliance curve*, and the entire diagram is called the *compliance diagram of the lungs (***Hall and Guyton, 2015).**

Mathematically

$$C = \frac{\Delta V}{\Delta P}$$ *where* C = compliance

ΔV = change in volume

ΔP = change in pressure

The relationship between total respiratory system compliance and lung and chest wall compliance is indicated by the following formula :

$\frac{1}{Ct} = \frac{1}{Cl} + \frac{1}{Cw}$ *where* Ct = Total compliance of the respiratory system

Cl = Compliance of the lung

Cw = Compliance of chest wall

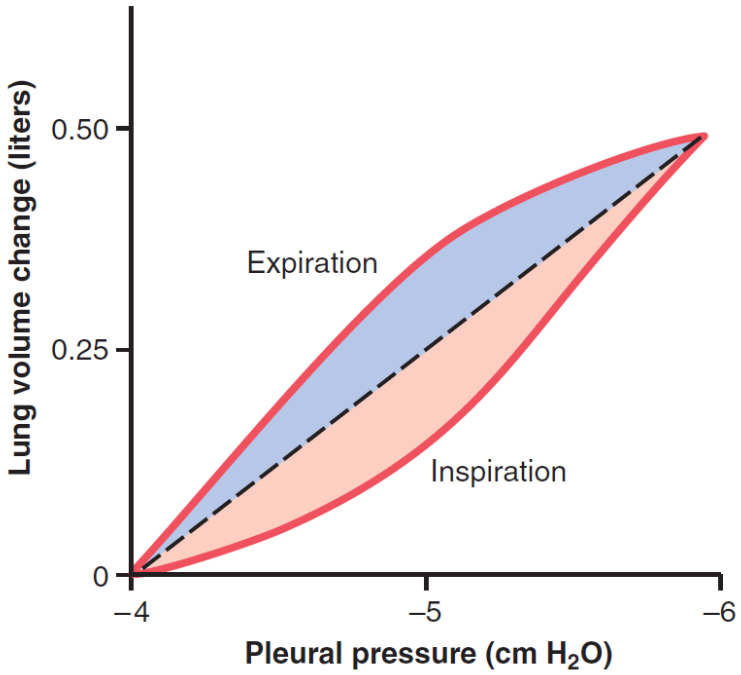

Fig.4 Compliance diagram in a healthy person. This diagram shows changes in lung volume during changes in transpulmonary pressure (alveolar pressure minus pleural pressure) (**Hall and Guyton, 2015)**.

Table (1) : Factors affecting respiratory compliance

Aspect	Low compliance	High compliance
Normal lung	Small person	Large person
Lung surfactant	Respiratory distress Syndrome, Surfactant protein B Deficiency	
Fibrous stroma	Disorders of lung parenchyma (fibrosis)	Age, emphysema
Visceral pleural	Thickening secondary to TB, Asbestos exposure, haemothorax	
Tone in muscle of alveolar ducts	Histamine, Serotonin, hypoxia	Bronchodilator drugs
Pulmonary blood Volume	Mitral stenosis, Left ventricular failure	isocapnoeic hypoxia Pulmonary stenosis

(Papandrinopoulou, Tzouda and Tsoukalas, 2012)

Pressure-volume loop for the respiratory system

If a pressure-volume curve for the respiratory system is plotted through a cycle of inspiration and expiration a 'loop' is obtained, as the inspiratory and expiratory limbs of the curve do not exactly coincide (**Smith, 2009).**

The slope of this curve is equal to the compliance. The inspiratory and expiratory curves are separated on the PV curve; this area of separation is termed *hysteresis* . In the lungs, which results both from the collapse of small airways and from the surface tension at the gas-liquid interface of alveoli that must be overcome to inflate the lungs (**Grinnan and Truwit, 2005)**.

PV curves can potentially indicate unique characteristics and the condition of the lung, and clinicians have been relying on this data to determine ventilator settings **(Jonson, 2005).**

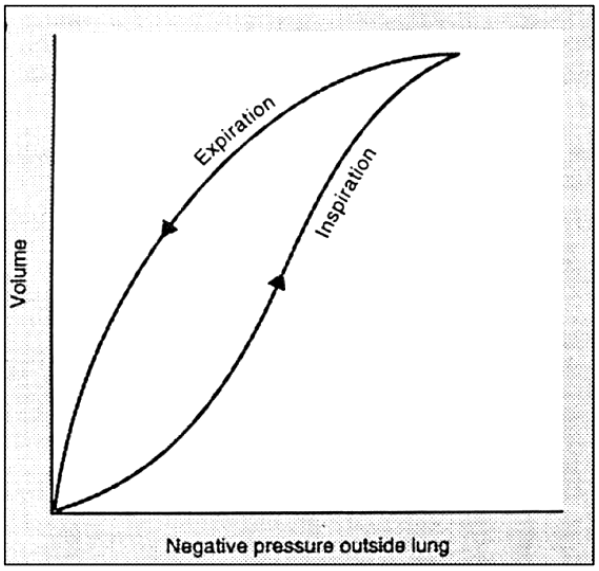

Fig.5 Pressure–volume curve. Shown is a pressure–volume curve developed from measurements in isolated lung during inflation (inspiration) and deflation (expiration). The slope of each curve is the compliance. The difference in the curves is hysteresis (**Grinnan and Truwit, 2005)**

Measurement of respiratory system compliance

Compliance can be obtained from the gradient of a P-V curve. However, this leads to two different values depending on the measurement technique. **Static compliance** obtained when P-V curve is plotted by applying known distending pressures to the respiratory system, and measuring the corresponding changes in volume. Appropriate time must be allowed between measurements, for equilibration of the lung, when all gas movement has ceased. **Dynamic compliance** obtained during spontaneous breathing or mechanical ventilation. In this case the changes in volume and pressure are recorded continuously with no pause between measurements (**Smith, 2009).**

So in practical terms, *dynamic* compliance is the volume change divided by the peak inspiratory transthoracic pressure, and *static* compliance is the volume change divided by the plateau inspiratory transthoracic pressure (**Hagberg and Benumof, 2013)**.

Pulmonary surfactant

The surface tension between gaseous-aqueous interface in the lungs is decreased by the presence of a thin layer of fluid known as pulmonary surfactant. The pulmonary surfactant is produced by the alveolar type-II (AT-II) cells of the lungs. It is essential for efficient exchange of gases and