Management of Intra-Operative Pulmonary Atelectasis

An Essay

Submitted For Partial Fulfillment of the Master Degree in **Anesthesiology**

BY

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First and foremost, I feel always indebted to AUAH, the Most Kind and Most Merciful.

I wish to express my deepest thanks, gratitude and appreciation to Prof. Dr. Bassem Boulos Gabriel, Professor of Anesthesiology, Intensive Care & Pain Management Faculty of Medicine, Ain-Shams University for his meticulous supervision, kind guidance, valuable instructions and generous help.

Special thanks are due to Dr. Hala Salah EL-Din El-Ozairy, Lecturer of Anesthesiology, Intensive Care & Pain Management Faculty of Medicine, Ain-Shams University for her sincere efforts, fruitful encouragement.

I am deeply thankful to Dr. Mohammed Osman Awad Taeimah, Lecturer of Anesthesiology, Intensive Care & Pain Management Faculty of Medicine, Ain-Shams University for his great help, outstanding support, active participation and guidance.

Mohammed Gamal El-Kotb

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List of Abbreviations

Abb.	Full term
ARDS	Adult Respiratory Distress Syndrome
	Alveolar Recruitment Maneuver
	Oxygen Content of Arterial Blood
	Capillary Oxygen Content
	Chest Wall Compliance
	Lung Compliance
	Continuous Positive Airway Pressure
	CardioPulmonary Bypass
	Respiratory System Compliance
	Computed Tomography
	Mixed Venous Blood Oxygen Content
	Extra-corporeal Circulation
	Expiratory Reserve Volume
	Functional Residual Capacity
	Fetal Hemoglobin
	Inspiratory Capacity
IL	
	Inductive Plethysmography
	Inspiratory Reserve Volume
LIP	Lower Inflection Point
	Morbidly Obese
MRS	Maximal Recruitment Strategy
	Open Lung Approach
PA	Alveolar Oxygen Tension
PaO ₂	Partial Pressure of Oxygen
Pavg	Average Inspiratory Pressure
Paw	Airway Pressure
PEEP	Positive End Expiratory Pressure
Pex	End Expiratory Alveolar Pressure
	Peak Inspiratory Pressure
	Prolonged Recruitment Maneuver
	Static or plateau Pressure
	Pressure - Volume Curve

List of Abbreviations cont...

Abb.	Full term
QT	Total Blood Moving Through the System
RM	Recruitment Maneuver
RV	Reserve Volume
Ti	Inspiratory Time
TLC	Total Lung Capacity
TNF	Tumor Necrosis Factor
UIP	Upper Inflection Point
VC	Vital Capacity
VILI	Ventilator Induced Lung Injury
VT	Tidal Volume
WOB	Work of Breathing

Abstract

For monitoring of the success of the maneuver and its efficacy several tools have been described. They include: Pulse Oximetry, Bronchoalveolar lavage Fluid Sampling, Inductive plethysmography (IP), Ultrasonography, Computed Tomography (CT).

Possible side effects of alveolar recruitment include hypotension, desaturation, barotrauma, and decrease in cardiac output, hypo-ventilation and acidosis.

Key words: Pulmonary Atelectasis - Computed Tomography - Lung Compliance- Vital Capacity- Tidal Volume

INTRODUCTION

eneral anesthesia, even in the lung-healthy subject, causes an increase in intrapulmonary shunt, which may impair oxygenation. The magnitude of shunt is correlated with the formation of pulmonary atelectasis. Atelectasis appears within minutes after the induction of anesthesia in 85%–90% of all patients. The amount of atelectasis is larger in obese patients or when a high fraction of inspired oxygen (FiO₂) is used (*Rusca et al.*, 2003).

Atelectasis occurs in the dependent parts of the lungs of most patients who are anesthetized. Development of atelectasis is associated with decreased lung compliance, impairment of oxygenation, increased pulmonary vascular resistance, development of lung injury and postoperative infection. The adverse effects of atelectasis persist into the postoperative period and can impact patient recovery and hence the need for measures to be taken to avoid atelectasis formation and reverse it if it had already been produced (Maceiras and Kavanagh, 2005).

Although intraoperative lung atelectasis is almost inevitable, special groups of patients are known to be affected more than others; for instance, in infants, morbidly obese patients, laparoscopic surgeries and cardiac surgeries, etc. (Maceiras and Kavanagh, 2005).

Anesthesia providers may employ a variety of ventilation strategies to prevent or reverse the pulmonary changes induced by general anesthesia. Some commonly employed techniques include recruitment maneuvers (RMs); for example, positive end-expiratory pressure (PEEP), intervals of large tidal volume ventilation (>15 ml/kg), intermittent "sigh breaths", and sequential increases in PEEP (Talley et al., 2012).

Recruitment maneuver denotes the dynamic process of an intentional transient increase in trans-pulmonary pressure aimed at opening unstable airless alveoli, which has also been termed alveolar recruitment maneuver. The rationale for recruitment maneuvers is to open the atelectatic alveoli, thus increasing end expiratory lung volume, improving gas exchange, and attenuating VILI (Ventilator Induced Lung Injury) (Pelosi et al., 2010).

Alveolar Recruitment Maneuvers has no specific counter indications, but their exclusion criteria are hemodynamic instability, pneumothorax, pneumo-mediastinum and subcutaneous emphysema, recent lung biopsy and resections. High inspiratory pressures may induce complications, such as hemodynamic changes and risk of barotrauma. Sustained airway pressure has hemodynamic repercussions (decreased venous return and increased left ventricle afterload during maneuver) and exposes lung to higher risk of barotrauma. with improvement Hypotension fast after maneuver



interruption is more frequent in hypovolemic patients (Gonçalves and Cicarelli, 2005).

Since postoperative pulmonary complications are a major contributor to the overall risk of surgery, so, strategies that will help prevent these complications would improve the quality of medical care and decrease hospital costs (Shander et al., 2011).

AIM OF THE WORK

The research is a review and a discussion of the different causes of intraoperative pulmonary atelectasis, different techniques of alveolar recruitment, the variation in their indication, and their expected outcomes.

PATHOPHYSIOLOGY OF ATELECTASIS

Mechanics of Respiration

muscle contraction. These muscles include the diaphragm and the external intercostal muscles of the rib cage and accessory inspiratory muscles. Contraction of these muscles enlarges the thoracic cavity, creating a sub-atmospheric pressure in the alveoli. Contraction of the diaphragm leads to downwards displacement of the thoracic cavity and contraction of external intercostal muscles leads to lifting of the thoracic cage leading to increase in the antero-posterior diameter (Petros and Sheehan, 2012).

As alveolar pressure declines, atmospheric air moves into the alveoli by bulk flow until the pressure is equalized. Expiration is usually passive, resulting from relaxation of the inspiratory muscles and powered by elastic recoil of lung tissue that is stretched during inspiration. With relaxation of the inspiratory muscles and lung deflation, alveolar pressure exceeds atmospheric pressure, so gases flow from the alveoli to the atmosphere by bulk flow. Active expiration is due to internal intercostal muscles and the abdominal recti muscles (*Petros and Sheehan, 2012*).

Lung Volumes

Lung volumes and lung capacities refer to the volume of air associated with different phases of the respiratory cycle (figure 1). Lung volumes are directly measured; Lung capacities are inferred from lung volumes.

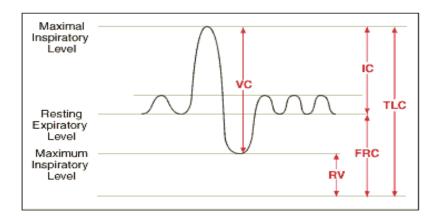


Figure (1): Lung volumes and Capacities (Morgan and Mikhail's, 2013).

- **1- Tidal Volume (VT)**: The lung volume representing the normal volume of air displaced between normal inhalation and exhalation when extra effort is not applied. It's about 500 ml in a normal relaxed breath.
- **2-** *Inspiratory Reserve Volume (IRV):* It's about 3,100 ml, and it is the maximal amount of additional air that can be drawn into the lungs by determined effort after normal inspiration.
- **3-** Expiratory Reserve Volume (ERV): About 1,200 ml and it's the additional amount of air that can be expired from the lungs by determined effort after normal expiration.

- **4-** *Inspiratory Capacity (IC):* It is approximately 3,600 ml, and it is the volume of air that can be inspired after a normal expiration; it is the sum of the tidal volume and the inspiratory reserve volume.
- 5- Functional Residual Capacity (FRC): The volume of gas left in the lungs at the end of normal tidal expiration. It is usually around 2,400 ml of volume (Morgan and Mikhail's, 2013).

FRC is the lung volume in which gas exchange is taking place small fluctuations of alveolar and arterial gas tensions occur with each tidal breath as fresh gas mixes with alveolar air (*Petros and Sheehan*, 2012).

FRC therefore acts as a buffer; maintaining relatively constant atmospheric (A) and alveolar (a) gas tensions with each breath preventing rapid changes in alveolar gas with changes in ventilation or inspired gas, e.g. During induction or recovery from anesthesia, increasing the average lung volume during quiet breathing, and reducing work of breathing due to shape of compliance curve.

- 6- Total Lung Capacity (TLC): The inspiratory capacity plus the functional residual capacity; the volume of air contained in the lungs at the end of a maximal inspiration; also equals vital capacity plus residual volume, in a normal adult, it's about 6000 ml.
- 7- Residual Volume (RV): It's approximately 1,200 ml, and it represents the volume of gas in the lung at the end of

maximal expiration determined by the balance of expiratory muscle activity and the resistance to volume decrease by the lungs and chest wall.

8- *Vital Capacity (VC):* It's about 4,800 ml, and it is the maximum volume that can be exhaled following a maximal inspiration.

VC = IRV + VT + ERV

VC and its components are measured by spirometer. Wedge variations in VC occur due to different causes, like height, which is roughly proportionate to VC. Also, age, were the VC decreases with increasing age, Sex: Males >Females, while posture causes less VC when supine, compared to when measured sitting or standing (*Petros and Sheeha*, 2012).

On the other hand, there are factors that will lead to reduction of the vital capacity, which can be subdivided into; Pulmonary causes as reduction in the distensibility of lung tissue or, reduction in the absolute volume of lung, (obstruction, atelectasis, pneumonia), or Non-pulmonary causes which may be due to the limitation of respiratory movements, neuromuscular thoracic expansion, musculoskeletal, position, diaphragmatic descent as seen in pregnancy, obesity, ascites, etc. (*Petros and Sheeha, 2012*).