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Applied Physiology & Pharmacology

for Anesthetists & Intensivists

Editors
Atul Prabhakar Kulkarni
Shilpushp Jagannath Bhosale
Vandana Agarwal

JAYPEE

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CHAPTER 13

Mechanisms of Atelectasis during Positioning during Anesthesia and Critical Care

Ruchira Wasudeo Khasne, Shreyas Laxman Bhor

INTRODUCTION

The word "atelectasis" is derived from two Greek words: Atelez (ateles meaning incomplete or imperfect) and ektasiz (ektasis meaning expansion) and means incomplete expansion.

An incomplete expansion (or collapse) of terminal bronchioles and alveoli is called pulmonary atelectasis. Atelectasis is commonly encountered in the perioperative settings, but it may not be always clinically significant. Causes and clinical significance of atelectasis in critically ill patients are different compared that occurring in patients given general anesthesia (GA). At times, persistent atelectasis may lead to clinically significant hypoxemia, respiratory failure, and affect the outcome. Before discussing pathophysiology and mechanisms of atelectasis, risk factors, and clinical consequences of atelectasis, we will look into physiology of lung expansion.

PHYSIOLOGY OF ALVEOLAR AND BRONCHIOLAR EXPANSION

In a normal lung, various factors play a role in alveolar or bronchiolar expansion and collapse. They can be summarized as follows:

- Forces/stresses acting on terminal bronchioles and alveoli:
  - Pressure applied by gas/liquid on alveolar or bronchial walls resulting in increased transmural pressure
  - Radial stress acting on alveoli or bronchioles by surrounding lung parenchymal extracellular matrix (ECM)
  - Surface tension, which acts radially inwards, by lung surfactant.

Of these three, transmural pressure and radial stress by ECM are outward acting forces (causing lung expansion), whereas surface tension is an inward acting force (causing lung collapse).

PHYSIOLOGY OF PLEURAL PRESSURE AND TRANSPULMONARY PRESSURE

Pleural pressure is the pressure inside pleural cavity. It is usually negative during inspiration in a spontaneously breathing healthy human. There is a regional variation in pleural pressure across pleural space; depending on gravity, the lung weight, and interaction between conformation of lung tissue to the shape of thorax. Pleural pressure changes according to a vertical gradient from top to bottom of the thoracic cavity, being more negative in at the top (nondependent) and less negative at the bottom (dependent) portions of the lung. This gradient of pleural pressure varies with patient positions: Greatest in upright, least in prone, and even distribution in supine position. The less vertical gradient of pleural pressure (because of transfer of mediastinal weight to sternum) results in more even distribution of inspired gas in prone position and contributes to improved oxygenation.

Knowing regional differences in pleural pressure is important to understand the role of transpulmonary pressure (P_L) in regional difference of ventilation. P_L is usually simplified as the difference between pressure at airway opening (which is approximately equal to alveolar pressure at no flow state) and pleural pressure. P_L is the surrogate of elastic recoil pressure of lung and positive P_L is responsible for maintaining alveolar expansion. P_L also shows vertical gradient causing more lung expansion in dependent than apical lung units (regional difference in ventilation **(Fig. 1)**.

PATHOPHYSIOLOGY OF ATELECTASIS

Three basic mechanisms contribute to formation of atelectasis:

1. Lung tissue compression (compression atelectasis)
2. Absorption of alveolar air (absorption atelectasis)
3. Surfactant function impairment.

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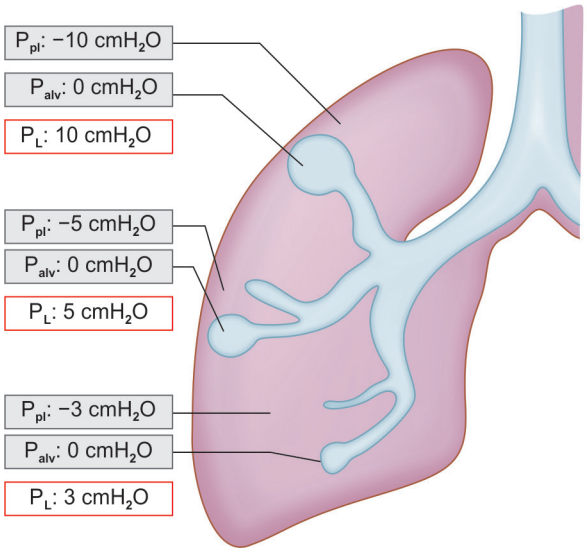


Fig. 1: Pleural and transpulmonary pressure. (P_{alv}: alveolar pressure; P_{pl}: pleural pressure; P_L: Transpulmonary pressure.)

Compression Atelectasis

Pleural Pressure and Alveolar Pressure

The pleural pressure is high in dependent as compared to nondependent lung, causing compression and collapse, i.e., atelectasis of adjacent alveoli in the dependent lung. As shown in **Figure 1**, the pleural pressure is –3 cmH_2O in dependent and –10 cmH_2O in nondependent lungs. The alveolar pressure, below which alveoli would collapse, is called as a critical closing pressure and the minimal pressure needed to keep alveoli open is known as critical opening pressure. Thus, to achieve positive P_L and lung expansion, critical opening pressure would be higher in dependent lung regions needing higher alveolar pressure than pleural pressure.

Role of Diaphragm and Chest Wall Dynamics

Diaphragm, an elliptical cylindroid with a dome-shaped cap, is a muscular septum which separates abdominal and thoracic cavities. It is the primary muscle of ventilation and it also decreases transmission of abdominal pressure to lungs. It can be considered as having the lower, dependent part and an upper, nondependent part, which shortens on inspiration leading to increased transverse diameter of thorax. The lower, dependent portion of diaphragm shows more displacement as compared to its upper, nondependent part in supine, spontaneously breathing humans. This leads to more lung expansion and more ventilation in the alveoli adjacent to dependent portion of diaphragm.

Induction of anesthesia causes reduction or loss of tone of diaphragm, which leads to transmission of abdominal pressure to lungs, a cranial shift of diaphragm with resultant decreased transverse diameter of the thorax, and reduced preferential downward displacement of the dependent portion of the diaphragm. Muscle paralysis worsens all the above effects. All of these lead to compression of dependent lung units and atelectasis. These effects are not fully counterbalanced by positive pressure ventilation as positive pressure preferential displaces upper, nondependent portion of diaphragm. These changes contribute to decrease in functional residual capacity (FRC) and decreased compliance. In addition to altered diaphragm dynamics, increased lung weight by pulmonary edema, compression of dependent lung units by nondependent lung leads to increased pleural pressure and decreased P_L along vertical axis and results in dependent lung atelectasis. Also, mediastinal weight, pleural effusion, and conditions with increased abdominal pressure can result in compression atelectasis.

Absorption Atelectasis

Two mechanisms are described which lead to absorption atelectasis. One is associated with complete airway occlusion and other without airway occlusion. Airway occlusion due to either increased airway resistance with airway collapse or airway obstruction due to mucus plug or secretions creates a gas pocket distal to occlusion. With preserved perfusion to lung unit, this gas pocket will collapse as gas uptake from lung unit into the blood will continue. This is also called as "obstructive atelectasis."

In other cases, absorption of alveolar air can occur without airway occlusion. Few lung units have low ventilation compared to perfusion (low V_A/Q ratio) and high fraction of inspired oxygen. In such case, high fraction of inspired oxygen will facilitate oxygen outflow to capillary blood which combined with low ventilation will result in alveolar collapse. Such lung units with low V_A/Q ratio are present in dependent lung regions leading to basal/dependent atelectasis. This concept is called as "absorption atelectasis."

Impaired Surfactant Function

General anesthesia, positive pressure ventilation, and high inspired oxygen concentration lead to impaired surfactant production and its function through inflammation, alveolar, and type II pneumocyte damage and thus contributes to development of atelectasis.

RISK FACTORS FOR PERIOPERATIVE ATELECTASIS

Risk factors for atelectasis in the perioperative period can be categorized as being related to patient, anesthesia, and surgery **(Tables 1 to 3)**. Positioning of the patient irrespective of the association of risk factors is an additional concern to develop atelectasis **(Table 4)**. GA is responsible for nearly instantaneous diaphragmatic cephalad displacement, fall in FRC, and compliance. These changes are exaggerated in obese patients, and laparoscopic, upper abdominal, and cardiothoracic surgeries. Additionally, the atelectasis in

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TABLE 1: Patient-related risk factors.

Factors	*Mechanism of atelectasis*
Obesity BMI > 25 kg/m^2	Increased compressive forces due to obesity, more cephalad displacement of diaphragm, rightward shift of the respiratory system pressure–volume curve, higher pleural pressure, lower respiratory system compliance, and reduced FRC
Age-dependent small airway closure	Risk of atelectasis increases as the age advances up to 50 years. After this age, there is age-dependent small airway closure, which causes delay in denitrogenation and alveolar collapse at the time of preoxygenation
Smoking	Irritation of airway mucosa, increased airway secretions, bronchiolar obstruction, and collapse
Diaphragm dysfunction/ neuromuscular disease	Higher risk of atelectasis, especially in upper abdominal surgery and cardiothoracic surgery. Reduced diaphragmatic excursions (if <10 mm: Higher incidence of atelectasis)
Increased intra-abdominal pressure	Ileus, ascites, compartment syndrome, pregnancy, intra-abdominal hypertension cause rise in pleural pressure, and loss of diaphragmatic tone
Acute lung inflammation	Aspiration, pulmonary edema lead to increase in lung weight, and impairment of surfactant more chances of atelectasis

(BMI: body mass index; FRC: functional residual capacity)

TABLE 2: Anesthesia-related risk factors.

Anesthesia	*Mechanism of atelectasis*
Lower tidal volume (without PEEP) and muscle paralysis	• Low TV ventilation "without PEEP" exaggerates the atelectasis • Use of muscle relaxants aggravates the damage
Anesthetic agents	• Direct correlation of intravenous and inhalational anesthetic agents (except ketamine), their duration, and atelectasis • Ventilatory effects of regional anesthesia depend on type and extent of motor blockade. Thoracic epidural and high spinal blocks can compromise with breathing and forced vital capacity • Nitrous oxide has higher lipid solubility than nitrogen, promotes alveolar gas absorption and risk of postoperative atelectasis • Opioids or sedatives reduces FRC, hypoxemia, respiratory muscle dysfunction, and atelectasis
High FiO_2	Promotes absorption atelectasis
Neuromuscular block, inadequate reversal, and phrenic nerve block	Aggravates compressive effect of the abdomen, changes in chest wall cross section lead to atelectasis
Transfusion-related acute lung injury	Evidence supports perioperative blood transfusion and atelectasis

(FiO_2: fractional inspired concentration of oxygen; FRC: functional residual capacity; PEEP: positive end-expiratory pressure; TV: tidal volume)

these patients may persist for a longer period. One should identify these risk factors and anticipate and implement preventive measures in advance.

CONSEQUENCES AND CLINICAL IMPACT OF ATELECTASIS

Atelectasis commonly occurs in dependent regions such as behind the diaphragm, which may not be evident clinically and on chest radiography. It is best detected and quantified with computed tomography. Clinical presentation depends on how rapidly it develops and its extent. Small and slowly developing areas of collapse may be asymptomatic but rapidly developing large-scale atelectasis can present with hypoxia and respiratory failure. The consequences of atelectasis on the respiratory mechanics as well as gas exchange are given below in brief.

- *Decreased compliance:* Atelectasis results in decreased compliance of both lung and respiratory systems causing increased work of breathing. Higher driving pressure ($P_{plateau}$ – PEEP = tidal volume/compliance) is associated with low respiratory system compliance. Evidence suggests that a decrease in FRC caused by anatomical and anesthetic factors does not correlate with the magnitude of atelectasis suggesting role of other factors (such as increased intrathoracic blood volume) in lung collapse and atelectasis.
- *Oxygenation impairment:* As atelectasis is associated with low V_A/Q ratios and intrapulmonary right-to-left shunt, atelectasis causes impaired oxygenation and hypoxemia. This impaired oxygenation correlates well with degree of atelectasis.
- *Effect of pulmonary vascular resistance (PVR):* Studies have shown that PVR is minimal at FRC. Atelectasis and therefore low FRC can lead to compression of extra-alveolar vessels and rise in PVR. Recruitment of alveoli and reduction in atelectatic lung areas reduce PVR. Factors associated with atelectasis such as pneumonia, acute respiratory distress syndrome (ARDS), endotoxemia, sepsis, and few volatile anesthetic agents (isoflurane, sevoflurane, desflurane above 1 minimum alveolar concentration) also impair hypoxic pulmonary vasoconstriction, thereby making hypoxemia more prominent.

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TABLE 3: Surgery-related risk factors.

Surgery	*Mechanism of atelectasis*
Type and duration of surgery	Cardiothoracic, upper abdominal, laparoscopy more proximity to diaphragm directly relates with atelectasis
Increased intra-abdominal pressure	• Promotes the cephalad displacement of the diaphragm • Peritoneal insufflation independently reduces end-expiratory lung volume (approximately 35% in the nonobese and approximately 15% in the obese) • Reduction in respiratory system compliance and end-expiratory transpulmonary pressure
One-lung ventilation	• Lung isolation exposes the collapsed lung to various insults; inflammation ischemia and re-expansion • Direct surgical trauma and mechanical ventilation injury • Compressive atelectasis during lateral decubitus
Cardiopulmonary bypass (CBP)	• More risk of alveolocapillary membrane injury, surfactant impairment, and mucociliary dysfunction • Sternotomy, pleural violation compromises with chest wall function, impair lung expansion, and diaphragmatic dysfunction in intra- and postoperative periods
Inadequate pain relief	Poor coughing, poor expiratory muscle strength, and ineffective mobilization
Body position	See **Table 4**

TABLE 4: Anatomical and physiological changes in lung due to positioning.

Body position	*Anatomical changes*	*Physiological changes*
Supine and lithotomy	Cephalad shift of diaphragm by compression of intra-abdominal organs	Almost 30% decrease in functional residual capacity (FRC) compared to sitting
Trende-lenburg	More lung compression than supine position, due to more cephalad shift of diaphragm	• Additional decrease in FRC (~10%) from supine • Increased end-expiratory transpulmonary pressure reduction in regional lung ventilation—atelectasis
Reverse Trende-lenburg	Release of compression of lung by intra-abdominal contents	Improved FRC compared to Trendelenburg and homogenous regional ventilation
Prone	• Cephalad shift of diaphragm • Released lung compression by mediastinal structures • Better matching of lung and chest wall shapes	• In awake, spontaneously breathing—reduction in FRC compared to sitting • In anesthetized patient with free abdominal movement—almost 50% increase in FRC compared to supine—favorable effects on homogenous ventilation, better V_A/Q matching
Lateral decubitus	Dependent lung—compressed from weight of mediastinum, nondependent lung and abdominal organs—cephalad shift of diaphragm	• Dependent lung—reduced FRC • Atelectasis almost exclusive to dependent lung • But, larger global FRC than supine position due to nondependent lung as nondependent lung volume exposed to lower compression forces and larger transpulmonary pressures

- *Inflammatory response:* There is altered cytokine and immune response due to atelectasis adding to the systemic inflammatory state.
- *Structural dysfunction and lung injury:* Repeated closing and opening of alveoli contributes shear stress and adds to lung injury. Also, atelectatic alveoli show structural damage to actin fibers and impaired barrier function due to immobility. Regional atelectasis diverts the inspiratory tidal volume toward normally aerated alveoli causing overstretching of these alveoli and lung injury.
- Atelectasis has also been associated with development of *pneumonia* and is one of the causative factors of surfactant dysfunction.

CONCLUSION

Atelectasis results from the partial or complete, reversible collapse of the small airways leading to an impaired exchange of CO_2 and O_2. It is one of the most common respiratory complications in the perioperative period. If allowed to persist, it can cause significant morbidity and mortality, including the development of pneumonia and acute respiratory failure. Therefore modifiable risk factors should be identified and preventive measures should be taken.

FURTHER READING

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Atul Prabhakar Kulkarni MD (Anesthesiology) FISCCM PGDHHM FICCM is currently Professor and Head, Division of Critical Care, Department of Anesthesiology, Critical Care and Pain at the Tata Memorial Hospital, Homi Bhabha National Institute, Mumbai, Maharashtra, India. A recognized postgraduate teacher for MD (Anesthesiology), he is now a Teacher for DM (Critical Care Medicine). A postgraduate diploma holder in Postgraduate Diploma in Health and Healthcare Management, he has received training in Intensive Care at St George Hospital, Sydney in 2002–2003. He has participated in many conferences on critical care and anesthesiology at international, national, and state levels. He was the Organizing Secretary for Anesthesia Review Course since 2004 to 2018. He was the Treasurer of Indian Society of Critical Care Medicine during 2004–2010. He was later General Secretary of Indian Society of Critical Care Medicine (2010–2014) before he took over as the President of Indian Society of Critical Care Medicine (2015–2017) and the Chancellor, Indian College of Critical Care Medicine. He was a Member of the Editorial Board of Indian Journal of Critical Care Medicine (IJCCM) and Reviewer for IJCCM and Indian Journal of Anesthesia (IJA). He is also the Past President of Association of South Asian Association for Regional Cooperation (SAARC) Critical Care Societies and Secretary General. After serving as the Honorary Secretary General of the Asia Pacific Association of Critical Care Medicine, he took over as the President of the prestigious organization. Currently, he is the Editor-in-Chief of the Indian Journal of Critical Care Medicine.

Shilpushp Jagannath Bhosale DM (Critical Care Medicine) Fellowship Pediatric Critical Care Medicine (University of Toronto) is currently Professor, Division of Critical Care, Department of Anesthesiology, Critical Care and Pain at the Tata Memorial Hospital, Homi Bhabha National Institute, Mumbai, Maharashtra, India. He completed his Fellowship in Pediatric Critical Care Medicine at the Sick Kids Hospital, University of Toronto, Canada in 2015. He is recognized Postgraduate Teacher and Examiner for DNB and DM (Critical Care Medicine) examinations. He has been the Treasurer of Indian Society of Critical Care Medicine in 2019–2020 and Joint Secretary in 2016. He has also been the Member of the Indian Society of Critical Care Medicine, Mumbai Branch in 2018. He has participated in many conferences on critical care medicine and anesthesiology at international, national, and state levels. He is also the Organizing Secretary for Case Based Review in Critical Care Medicine for the Examinee (BRITE) course that is conducted in Tata Memorial Hospital yearly.

Vandana Agarwal MD (Anesthesiology) FRCA is Professor, Department of Anesthesiology, Critical Care and Pain at the Tata Memorial Hospital, Homi Bhabha National Institute, Mumbai, Maharashtra, India. After postgraduation, she trained in the UK and became a Fellow of Royal College of Anaesthesia by examinations. She is a recognized Postgraduate Teacher. She is Clinical Lead for Hepato-Pancreato-Biliary anesthesia. She has been Secretary of ISCCM (2014–2016 and 2018–2019) and Treasurer (2016–2017). She is currently the Chairperson of Mumbai Branch of Society of Onco-anaesthesia and Perioperative Care. She has over 40 publications in national and international journals and many book chapters to her credit in anesthesia and intensive care. She has delivered many lectures on various topics in anesthesia, and intensive care in several national and state level conferences.

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