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Review Article

Atelectasis: Causes, Consequences, Comorbidities, Pathophysiology, Prevention, and Treatment

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Abstract

Impeded compliance in the respiratory system and diminished oxygenation are signs of atelectasis. Although not thoroughly explained, several mechanisms resulting in atelectasis are commonly held: compression, alveolar gas resorption, and surfactant impairment. Various and diverse factors precipitate atelectasis, such as introducing FiO2, obesity of the patient, general and intubated anesthesia, chronic obstructive pulmonary disease (COPD), the patient's age, surgery, and type of surgery. Atelectasis results in compliance reduction, oxygenation deterioration, and exacerbation of lung impairment. Comorbidities of atelectasis include cardiomyopathy, type 2 diabetes, hypertension, and stroke.

Many patients who undergo a surgical procedure with general anesthesia experience atelectasis. Hence, it is critical to identify the underlying etiology and mechanism of atelectasis, applying a specific therapeutic approach and eliminating or reducing perioperative complications in the lungs and alveoli. Atelectasis' prevention and treatment range from spontaneous ventilation, preoxygenation, CPAP or PEEP, and recruitment maneuvers. Properly applied recruitment maneuvers improve respiratory mechanisms and gaseous transfer in patients who manifest atelectasis under general anesthesia. Nevertheless, more studies regarding the administration and verifying the effectiveness of such procedures on oxygenation and lung parameters would further confirm, to a greater or lesser degree, their beneficial and uniform contribution to the improved prognoses of impaired patients.

Keywords: *Alveoli; COPD; General Anesthesia; Intubation; Lung Impairment; Respiratory*

Abbreviations

ARDS: Acute Respiratory Distress Syndrome; COPD: Chronic Obstructive Pulmonary Disease; CPAP: Continuous Positive Airway Pressure; T: Computed Tomography; EIT: Electric Impedance Tomography; FiO_2: Fraction of Inspired Oxygen; MRI: Magnetic Resonance Imaging; PEEP: Positive End Expiratory Pressure; QoL: Quality of Life

Introduction

Atelectasis is a significant clinical condition which is frequently an antecedent of or a contributor to other postoperative pulmonary complications that are generally complicated. Atelectasis involves alveolar collapse, which is reversible. Atelectasis occurs from an obstruction in the airway that serves affected alveoli involved in respiratory gas exchange [1].

Historical perspective

For several decades, surgeons and anesthesiologists have recognized that patients with otherwise healthy lungs can experience significant respiratory compromise after the administration of general anesthetics [2].

Atelectasis' symptoms include diminished compliance in the respiratory system and accompanied by reduced oxygenation. Atelectasis was initially presumed to be a cause of impaired oxygenation during general anesthesia. Bendixen., et al. (1963) hypothesized that ventilating patients spontaneously, deprived of deep episodic breathing, might result in advanced atelectasis complicated by augmented shunting and reduced pulmonary compliance. However, these changes were reversible by hyperinflating the lungs [3].

Due to conventional radiology limitations at that time, it had been problematic to confirm this hypothesis until the advent of enhanced imaging technologies. More contemporary and useful images techniques include computed tomography (CT), magnetic resonance imaging (MRI), electric impedance tomography (EIT), ultrasonography, and—the most current procedure—intravital microscopy for diagnosing atelectasis [4,5]. The use of these newer imaging procedures has revealed that about 90% of patients who undergo general anesthesia develop atelectasis and in some cases, resulting in aveolar damage. Severe atelectasis can lead to respiratory failure and death [2,5,6].

The following summarizes the causes, consequences, comorbidities, pathophysiology, prevention, and treatment of atelectasis, and calls for further research into the effectiveness of specific treatment procedures.

Discussion

Causes and pathophysiology of atelectasis

The degree of susceptibility for the attack of atelectasis is the greatest for patients under anesthesia. Although the precise mechanisms underlying atelectasis remain unsettled, three mechanisms contributing to intraoperative atelectasis are recognized worldwide [7]. These mechanisms are compression, alveolar gas resorption, and surfactant impairment [8].

Compression

The distinctive effect of general anesthesia on alveoli is mechanical compression. Compression atelectasis occurs when the thorax's pleural pressure exceeds intrapulmonary pressure, leaving the alveoli unable to persist in an open state [9,10]. When a patient under anesthesia is placed in a supine position, heightened pleural pressure is encountered due to various organs' weight against the diaphragm, contributing to compression atelectasis [11].

The diaphragm equilibrates pressure between the lungs and abdomen [12]. A change in blood pressure can increase intrapleural pressure, further adversely affecting patients with specific commorbidities [11–13].

Alveolar gas resorption

Aveolar gas resorption is affected by two processes. The first process involves oxygen tension during oxygen supplementation. Under normal conditions, the lungs' low-ventilation regions compared to perfused regions exibit decreased alveolar oxygen tension when the amount of inspired oxygen is low. During general anesthesia, when the inspired oxygen's fraction is enhanced with supplemental oxygen, a proportionate increase in the partial pressure of arterial oxygen occurs. Consequently, gaseous exchange between the alveolus and the capillary is enhanced. Besides, the alveolar nitrogen tension decreases as the aveolar volume is reduced due to nitrogen loss and a corresponding increase in oxygen absorption [14].

The second process is triggered by an overall collapse of small airways. Under this scenario, gas pockets form, which become trapped in alveoli distal to the obstruction. These gas pockets progressively collapse as oxygen uptake proceeds according to the diffusion gradient [9].

Surfactant impairment

Pulmonary surfactant, a surface-active phospholipid, decreases the surface tension of the alveoli. Its presence enhances stability of alveoli, preventing alveolar collapse. Anesthesia adversely affects surfactant, hindering its stabilizing properties. The repetitive opening and closing of alveoli during general anesthesia (with mechanical ventilation) results in diminished levels of surfactant, leading to increased surface tension and a significant reduction in or loss of functional residual capacity [15].

Causes of atelectasis

Fraction of inspired oxygen (FiO2)

During mechanical ventilation, the introduction of high FiO2 contributes to the development of atelectasis, impairing respiratory gaseous exchange [16,17].

Ventilation parameters and tidal volume

The setting of ventilation parameters and tidal volume is especially critical in acute respiratory distress syndrome (ARDS) patients. These patients express low tidal volumes. Positive end-expiratory pressure should be maintained (being mindful of existing pulmonary lesions) [18].

Obesity in the patient

A slight association has been observed between of atelectasis and body mass index [19]. However, a low residual functional capacity and more significant abdominal pressure are observed in obese patients [20]. Together, these factors contribute to alveolar collapse. The prevalence of atelectasis is more significant in the obese group than the non-obese group of patients undergoing surgery [21].

Anesthesia

Atelectasis may be observed in a patient after the initiation of general anesthesia [22]. Anesthetics cause respiratory depression and impairment of the neurological control of the respiratory muscles [23].

Chronic obstructive pulmonary disease (COPD)

COPD patients develop mild atelectasis and slight shunt. Moreover, a worsening of the V/Q ratio is encountered [22].

The preventive mechanism for atelectasis in COPD patients has not been wholly explained [24]. Hyperinsufflation circumvents compressive alveolar collapse, reducing the drop in residual functional capacity, although promoting absorption atelectasis [22]. Also, equilibrium can be augmented by the slight flexible recoil of the thoracic wall and lung, decreasing lung volume [24].

Age of the patient

A patient's age is not the most crucial factor contributing to atelectasis. However, children are prone to atelectasis owing to lower residual capacity. Greater pliability of the thoracic cage (rib cage) and related paradoxical movements of the diaphragm also predispose children to atelectasis [25]. The application of 5 cm H2O effectively twarts atelectasis in this population [24.25].

With an increase in age, the incidence of premature closure of the small airways ensues with a corresponding rise in regions of low ventilation perfusion ratios. This scenario discourages compression atelectasis, but may promote absorption atelectasis [26].

Surgery and the type of surgery

Regardless of the type of surgery, atelectasis is predominant during the intial phase of anesthesia [27]. Surgical trauma can trigger reflexes in the phrenic nerve, affecting the respiratory muscles. These surgically-induced reflex-disruptions result in respiratory impairment, causing constriction. These factors combined produce hypoventilation and a concomitant decrease in residual function capacity, contributing to atelectasis [28].

In laparocscopic procedures, pneumoperithoneum insufflation pressure is factorial in promoting or discouraging atatelectasis [29].

In thoracic surgery, lung compression is accompanied by secretion-excess and airway reactivity. In cardiac surgery (pulmonary bypass), shunt and hypoxiemia develop with atelectasis—facilitated by increased capillary permeability and aveolar edema, resulting in increased lung weight and extravascular pulmonary fluid [30].

Consequences of atelectasis

Compliance reduction

Compliance reduction results from diminished lung volume, impairing pulmonary function [31].

Oxygenation deterioration

During surgery under general anesthesia, the shunt's persistence corresponds to the degree of atelectasis [32]. Small airway closures and atelectasis contribute significantly to hypoxemia in patients undergoing surgery, resulting in hypovolemia, anemia, hypoventilation, and disturbances in the ventilation-perfusion ratio [31,32].

Aggravation of lung impairment

As the alveoli open and close repetitively, pulmonary damage occurs in the atelectatic region and throughout the lungs. The amount of lung tissue affected by atelectasis is inversely proportional to the amount of lung tissue that needs to adapt to the tidal volume applied [18,33].

Postoperative infections

In the experimental setting (as opposed to the clinical setting), a correlation between respiratory infection progression and perioperative atelectasis has been established [34]. Atelectasis interferes with antibiotic agents' absorption and perfusion into the lungs, disrupting the precise and efficacious drug concentrations needed to effectively control pathogens, resulting in persisent infection or delayed recovery [35].

Comorbidities and sequelae of atelectasis

Comorbidities of atelectasis include cardiomyopathy, diabetes mellitus (Type-II), hypertension, and stroke [20,25].

Prevention and treatment of atelectasis

Preparations should be made in the prevention of or rapid response to atelectasis during any phase of the perioperative period, bearing in mind that the intial introduction of the anesthesia is the most vulnerable period.

Spontaneous ventilation

Spontaneous ventilation contributes to lung conscription, compensating for the loss of diaphragmatic tone and a reduced gradient of arterial aveolar oxygen [27,33].

Preoxygenation

Preoxygenation (using a FiO2 less than 1 before inducing anesthesia) is reached as the patient inhales gas with FiO2 equal to 1. Such levels of oxygen concentration and denitrogenation induce atelectasis [16,17,36].

Continuous positive airway pressure (CPAP) or positive end-expiratory pressure (PEEP)

The application of CPAP at 5–6 cm H2O during anesthetic initiation prior to orotracheal intubation, inhibits intraoperative atelectasis by restoring oxygenation, increasing oxygen reserves, and restoring lung volume in dead spaces [36]. Pressure of about 10 cm of H2O is needed by obese patients [37]. Lung overdistension may appear at PEEP H2O levels over 10 cm to 15 cm, which is avoided at lower levels [36,38].

Optimum ventilation parameters

Low tidal volume with moderate to high PEEP is more potent in and in keeping with collapse avoidance than high tidal volume and low PEEP [39]. When ventilating one lung, 8 ml/kg tidal volume, a PEEP of 4–10 cm H2O, and 35 cm H2O of plateau pressure-assisted level reduce the occurrence of atelectasis post-surgically [40].

Recruitment maneuvers

Recruitment maneuvers are applied once atelectasis is confirmed. The maneuvers' aim is to provide a constant increase of transpulmonary pressure, reversing the collapse of aveoli [41]. Recruitments stimulate surfactant release, reinstate alveolar stability, and lessen any damage caused by mechanical ventilation.

The efficacy of the maneuvers is subject to various factors, such as ventilator adjustments, the patient's pulmonary condition, and the patient's threshold in amplified thoracic pressure [41]. The maneuvers typically take place laterally and until maximum results are achieved [39,42]. Hence, recruitment maneuvers should be chosen to match the patient's particular state and status.

The transpulmonary pressure necessary for distention is dependent on pleural pressure. Consequently, transpulmonary pressure must be higher than pleural pressure in cases of compliance-reduction in the thoracic cage [39]. Higher pressures of up to 60–70 cm H2O are required for patients experiencing acute respiratory distress syndrome due to more significant surfactant impairment and alveolar edema [43,44].

The pressure required to avoid alveolar collapse is comparatively lower than that required to inflate the alveolus. The pulmonary components are stabilized and re-occlusion is avoided when PEEP is applied after recruitment. Also, when recruitments are applied devoid of PEEP, a transitory effect is observed. Hence, after re-occlusion, PEEP is employed to avoid the reoccurrence of atelectasis. Optimum PEEP accomplishes alveolar opening while avoiding hyper-distension of ventilated areas [32,42,44].

The recruitments are generally applied to patients experiencing impairment in gaseous exchange while under anesthesia—when a high fraction of inspired oxygen is applied or due to a low PEEP [32,45].

There are several parameters for evaluating the efficacy of the recruitment maneuvers, such as the increase in the ratio of the partial pressure of oxygen to the fraction of inspired oxygen, the decrease in alveolar-capillary gradient for oxygen tension, the outcome of lungcompliance and end-expiratory lung volume. The combination of an increase in residual functional capacity and dead space reduction generates maximum effect in normalizing the aveoli while avoiding lung hyperdistension [2,40].

Recruitment maneuvers have been shown to diminish the shunt or atelectasis in various conditions and surgical procedures. Claxton., et al. (2003) reported on the success of one-lung ventilation—in patients after cardiopulmonary bypass and in a bariatric surgery group of patients—in avoiding shunt and atelectasis [31].

Frequently after recruitment, a transitory reduction in oxygenation is seen. This reduction occurs due to the hemodynamic worsening and the blood flow shunt to highly insufflated regions [45]. Experiemental models have demonstrated that specific recruitment maneuvers can have lethal results in cases of pheumonmia and sepsis [4].

Moreover, recruitment maneuvers are contraindicated for intracranial hypertensive patients as they are prone to barotrauma. These patients must also be monitored precisely as high transpulmonary pressure, such as those observed in particular intensive recruitment maneuvers, has been associated with the breakdown of the alveolocapillary barrier—causing the release of cytokines into the interstitial pulmonary edema and blood [45].

Types of recruitment maneuvers

Two types of recruitment maneuvers can be applied to non-pulmonary disease patients undergoing anesthesia and surgery. The first is the vital capacity maneuver, which provides comprehensive lung distension that is not pathological, ameliorating atelectasis that might occur after cardiopulmonary bypass or induction [44].

The second maneuver is applied during pressure-regulated ventilation. This procedure involves an incremental increase in inspiratory pressure and positive end-expiratory pressure for every two to three respiratory cycles. This procedure maintains a sustained differential pressure of 20 cm to 25 cm H2O until a peak inspiratory pressure of 40 cm H2O and a PEEP of 20 cm H2O is attained. This state is regulated for about a minute, followed by a decrease until an optimum PEEP is achieved [30]. This maneuver seems related to low hemodynamic decline compared to its vital capacity complement. Levels are reached at respiratory frequencies of 10 to 12 cycles per minute and inspiration- expiration ratios from 1:1 to 1:1.5 [32].

Special considerations in children

Compared to adults—children have smaller aveolar diameter, airways, and a flexible ribcage. Nevertheless during anesthesia, the atelectasis is dispersed in children similar to adults. However, the inspiratory pressure required for children is less. than adults [4].

Efficacy of non-invasive mechanical ventilation

CPAP at approximately 10 cm H2O can reestablish RFC, diminishing atelectasis and hypoxemia by improving intrathoracic pressure and reducing respiratory exertion. The use of CPAP in patients who experience hypoxemia after abdominal surgery reduces the frequency of atelectasis, the requirements for reintubation, and the frequency of respiratory infections. The use of non-invasive ventilation with pressure-assistance seems to reduce the occurrence of atelectasis, regarding the application of continuous positive pressure [46].

Application of analgesia

Appropriate analgesia provides a reduction of atelectasis owing to an upsurge in lung volume and vital capacity. It also improves diaphragm activity indexes [40,41]. Local epidural methods, in combination with local anesthetics, may be advantageous. Experimental data regarding these methods are inconclusive: 1) outcomes are inconsistent concerning how pulmonary complications are cleared; 2) how a specific type of analgesia could hinder abdominal and intercostal muscles that assist ventilation. Systemic opiates can raise the pressure produced by the abdominal muscles, decreasing lung volume, while epidural analgesia can cause the condition [47].

Atelectasis secondary to pleural effusion (caused by pneumonia and or pleurisy)

One of the most significant risk factors for atelectasis is pleural effusion [48], an extreme buildup of fluid in the pleural space. Pleural effusion occurs due to an imbalance in the production and removal of fluids. Although pleural fluid may not indicate a specific disease, it impies a causal pathology—pleural effusions accompany several lung, pleural. and systemic disorders [49].

Mechanism of atelectasis secondary to pleural effusion

Probable mechanisms of pleural fluid buildup are as follows:

- Interstitial fluid in the lungs secondary to increased pulmonary capillary pressure (as in heart failure or penetrability as in pneumonia).
- Reduced intrapleural pressure.
- Diminished oncotic plasma pressure (as in hypoalbuminemia).
- Pleural membrane penetrability and lymphatic flow obstruction (as in pleural malignancy or infection).
- Diaphragmatic disorders (as in hepatic hydrothorax).
- Rupture of the thoracic duct (as in chylothorax).

Although several diseases cause pleural effusion, pneumonia is recognized as the foremost cause in adults and children [50]. Extensive pleural effusions may not only give rise to compressive atelectasis but may also cause complete lung collapse, accompanied by a contralateral shift of the mediastinum [51].

Signs and symptoms of atelectasis secondary to pleural effusion

Pleural effusion interrupts the respiratory mechanism of atelectasis patients, which can progress to restrictive respiratory failure. Respiratory parameters include total lung capacity, forced vital capacity, and functional capacity. A disparity in ventricular diastolic col-

lapse and ventilation-perfusion may arise, depending on the degree of atelectasis caused by effusion. Consequently, the most commonly observed symptoms are mild, nonproductive cough and dyspnea [52].

Diagnosis of atelectasis secondary to pleural effusion

The following diagnostic application of bronchoscopy is recommended in suspected atelectasis, central lung lesion, or massive pleural effusion [52]. Doyle and Lawler (1984) described seven criteria for diagnosing the rounded atelectasis [53] (Table 1).

1.	A rounded, peripheral lung form that is never surrounded entirely by the lung
2.	A form that is considerably dense at its periphery
3.	A form that creates an acute angle with the pleurae
4.	Thickening of adjacent pleurae
5.	Bronchi and vessels uniting in the direction of the mass
6.	A blurred centrally focused edge
7.	The existence of an air bronchogram

Table 1: *Seven criteria for diagnosing the rounded atelectasis, adapted from Doyle and Lawler (1984) [53].*

Radiologic diagnosis—chiefly the chest radiograph—is employed when signs and symptoms are suspected of having been caused by pleural effusion (as in atelectasis consequent to pleural effusion). Generally, the bilateral decubitus chest radiograph is performed to evaluate the underlying atelectasis [52]. Cytological analysis of pleural fluid is also utilized in diagnosing atelectasis resultant to pleural effusion [54].

Treatment of atelectasis secondary to pleural effusion

The selection of conservative treatment is based primarily on the specific cause and etiology, fluid drainage, and pleurodesis. Surgical intervention is employed for more severe or life-threatening cases. Specific treatment aimed at the primary cause resolves most transudative effusions, including atelectasis.

Pleurodesis is the most suitable treatment choice for repeated accumulative effusions. This procedure entails inserting a chest tube and introducing sclerosing chemical agents into the pleural cavity, creating bonds between the chest wall's outer and inner surfaces, to avoid air or fluid accumulation in the pleural cavity. This method is the most efficacious and minimally invasive procedure available to resolve pleural effusion [55].

The drainage process is accomplished by intercostal tubes or pigtail catheters, allowing the patient to remain ambulatory [56]. The urosac applied as a chest drainage bag has been shown to be safe, effective, and economical in several studies [57]. The surgical management of atelectasis secondary to a pleural effusion includes pleuropneumonectomy, decortication, window operation, pleurectomy, closure of bronchopleural fistula with or without grafting, thoracostomy and thoracoplasty, and fenestration [58].

Consequences of untreated atelectasis (or failed atelectasis treatment) secondary to pleural effusion

Patient presentation of pleural effusion accompanied by atelectasis is frequently observed in emergency departments and intensive care units. The determination of the extent of the pleural effusion and the degree of atelectasis is essential. Large-area pleural effusion poses a risk of compressive atelectasis, collapsing a lung (with a mediastinum contralateral shift) [51].

Atelectasis can arise as a complication in the perioperative period, contributing to morbidity and mortality and the progression of pneumonia and acute respiratory failure [59].

The pleural effusion diagnosis is challenging as the pleura is an inner cavity, lacking direct access. Several noninvasive diagnostic procedures are limited for definitive diagnosis. Treatment choice is based on any underlying disease, disease distribution in the pleural cavity, and the physician's experience. Delays or setbacks in a timely and accurate diagnosis can result in significant adverse consequences, including diminishing the patient's quality of life (QoL) [60].

Conclusion

Diminished compliance in the respiratory system and reduced oxygenation are hallmarks of atelectasis. Although not entirely understood, several mechanisms resulting in atelectasis are generally accepted: compression, alveolar gas resorption, and surfactant impairment. Numerous and varied factors contribute to or directly cause atelectasis, such as introducing FiO2, obesity in the patient, general anesthesia, COPD, the patient's age, surgery, and type of surgery. Atelectasis results in compliance reduction, oxygenation deterioration, and aggravation of lung impairment. Comorbidities of atelectasis include cardiomyopathy, type 2 diabetes, hypertension, and stroke.

Many patients who undergo a surgical procedure under general anesthesia experience atelectasis. Thus, it is vital to recognize the underlying causes and mechanisms of atelectasis in order to select and apply a specific therapeutic approach, eliminating or minimizing perioperative complications in the lungs and alveoli. The prevention and treatment of atelectasis range from spontaneous ventilation, preoxygenation, CPAP or PEEP, and recruitment maneuvers. Appropriately applied recruitment maneuvers enhance respiratory echanisms and gaseous exchange in patients who develop atelectasis under general anesthesia. Nevertheless, further studies regarding the application and overall efficacy of such procedures on oxygenation and lung parameters would help confirm, to a greater or lesser degree, their beneficial and consistent contribution to the improved prognoses of affected patients.

Conflict of Interest Statement

The authors declare that this paper was written in the absence of any commercial or financial relationship that could be construed as a potential conflict of interest.

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