Pathophysiology and Prevention of Intraoperative Atelectasis: A Review of the Literature

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Atelectasis is a common problem in the perioperative setting, affecting a significant number of surgical patients receiving general anesthesia. Absorption, compression, and reduced surfactant are the three mechanisms implicated in the etiology of atelectasis. Interventions designed to minimize the risk of intraoperative atelectasis such as positioning, positive end-expiratory pressure, and administration of the least amount of fraction of inspired oxygen can be used to maintain patency of small airways and ultimately improve gas exchange in the surgical patient.

Keywords: *intraoperative atelectasis, compression, absorption, PEEP, recruitment, continuing education.*

OBJECTIVES—(1). DEFINE THREE major mechanisms implicated in intraoperative airway collapse; (2). Identify hemodynamic changes occurring with increased intrapleural pressures; (3). Identify reasons why CO_2 absorption may be variable between laparoscopic patients; (4). Identify care strategies implemented to improve gas exchange immediately postanesthesia.

Atelectasis is a condition of the lung in which air is not able to fully expand certain branches of alveoli. When there is closure of airways, a patient is at increased risk of acquiring pneumonia as well as hypoxemia.[1] In the perioperative setting, it is fundamental for the nurse anesthetist and the postanesthesia care unit (PACU) nurse to understand the pathophysiology and prevention of atelectasis. With this understanding, a more efficacious action plan for the hypoxemic patient in the operating room and the PACU can be applied.

Understanding atelectasis is essential as it deteriorates gas exchange. About 90% of the surgery patients are affected by perioperative atelectasis.[2] Minimizing atelectasis with the use of positive end-expiratory pressure (PEEP) and recruitment can help stabilize hemodynamics and reduce respiratory problems intraoperatively.[3] General anesthesia reduces functional residual capacity (FRC) with an immediate and universal development of atelectasis in dependent regions of the lung.[4] In addition, intraoperative pulmonary dysfunction can trigger postoperative complications prolonging the recovery phase of care.[5,6] The incidence of pulmonary complications found postoperatively in noncardiac versus cardiac surgeries is 2.7% versus 2.5%, respectively, all associated with increased morbidity and mortality.[2,7]

This review was conducted to initiate discourse for the purposes of educating nurse anesthetists, PACU nurses, and other perioperative experts such as critical care and postsurgical unit nurses and respiratory therapists about the pathophysiology and prevention of intraoperative atelectasis.

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Intraoperative atelectasis is a common problem. Prevention is essential to reduce future complications such as postoperative pulmonary complications, length of recovery, and hospital costs.[8]

Pathophysiology

While under anesthesia, a patient is extremely susceptible to atelectasis, although the exact pathophysiologic mechanisms are not completely clear.[9] Three of the most agreed on mechanisms of intraoperative atelectasis are absorption, compression, and reduced surfactant.[10]

Absorption

Absorption atelectasis occurs more often in anesthetized patients because of two factors, namely hyperoxygenation and a low ventilation-perfusion (V/Q) ratio. When a patient is undergoing anesthesia, they will often be placed on 100% oxygen before placement of an artificial airway.[11] Under normal circumstances, the alveolar walls remain expanded because the air we breathe is not 100% oxygen. Room air contains a mixture of nitrogen, oxygen, and other gases. Because the capillary beds only absorb oxygen, the nitrogen in the alveoli keeps the lungs from collapsing. When a patient is breathing pure oxygen, the nitrogen in the lung is pushed out. Oxygen is absorbed by the capillaries leaving no air inside the alveoli. Alveoli are not supported by any cartilage and therefore will collapse on exhalation if there is an absence of remaining air. The second mechanism of absorption atelectasis is a low V/Q ratio. Essentially, the capillary beds are absorbing the oxygen faster than the bronchioles are providing the air. This can also occur more easily in restricted or blocked airways. Anesthesia exacerbates this problem by lowering the amount of air left in the lungs after expiration. This is known as the FRC. Intraoperatively reduced muscle tone lowers FRC by allowing more air to escape on exhalation. The FRC is further decreased on assuming a supine position.[2]

Compression

Compression atelectasis occurs when the pleural pressures of the thorax become greater than the intrapulmonary pressures, and the alveoli are no longer able to remain open. The anesthetized patient is particularly susceptible to this type of atelectasis for several reasons. In the supine position, an anesthetized patient has an increased pleural pressure owing to the weight of the organs in the abdomen against the diaphragm[12] and the weight of the patient's chest,[2] both significant mechanisms contributing to compression atelectasis. The diaphragm needs to be a rigid wall to balance the pressures of the lungs and the abdomen.[13] Furthermore, negative abdominal pressure is significant in reducing airway collapse.[14] Laparoscopic procedures are exceedingly common and are challenging from a ventilation standpoint. Carbon dioxide is used to facilitate visualization during laparoscopic procedures. The resultant pneumoperitoneum from abdominal insufflation can contribute to significant atelectasis. With increasing intra-abdominal pressure, a shift or mechanical displacement of blood flow occurs. This increases intrapleural pressure, which may alter outcomes in the operative patient who is burdened with comorbidities such as chronic obstructive pulmonary disease or heart failure. Hemodynamic changes from increased intra-abdominal pressure are shown in Table 1, adapted from Berridge.[15]

The rate of reabsorption of CO_2 would seem to have a significant influence on atelectasis resolution. Postoperatively, retained CO_2 leftover from the laparoscopic procedure can cause abdominal pain and nausea. Adequate pain management becomes a challenge.[16,17] With the exception of ketamine, all anesthetics produce respiratory muscle relaxation,[18] intensifying the effects of compression on the alveoli, squeezing out what is left of FRC during expiration. Specific disease processes can also cause compression atelectasis. For example, sepsis can cause a surplus of fluid in the lungs owing to inflammation; a heavier

Table 1. Hemodynamic Changes at Differing Intra-abdominal Pressures During Laparoscopic Cholecystectomy

IAP	HR	SV	CO	CVP	MAP	SVR
7	↑	↑	↑	Unknown	↑	↑
15	↑↑	↓↓	↓	↓	↓	↓

IAP, intra-abdominal pressure; HR, heart rate; SV, stroke volume; CO, cardiac output; CVP, central venous pressure; MAP, mean arterial pressure; SVR, systemic vascular resistance.

Adapted from Berridge.[15]