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# Impact of Anesthesia on Lung Function: Atelectasis, Shunt, and V/Q Mismatch

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**Abstract**
Anesthesia significantly impacts lung function, primarily through reductions in functional residual capacity (FRC), airway closure, and atelectasis formation. These changes disrupt ventilation-perfusion (V/Q) relationships, impair oxygenation, and contribute to postoperative pulmonary complications. The decrease in FRC is largely due to the loss of respiratory muscle tone, particularly the upward displacement of the diaphragm. Airway closure occurs when extraluminal pressure exceeds intraluminal pressure, predominantly affecting dependent lung regions and leading to V/Q mismatch. Atelectasis, present in approximately 90% of anesthetized patients, is caused by airway closure and gas resorption, and can persist for several days postoperatively. Strategies to prevent atelectasis include positive end-expiratory pressure (PEEP), recruitment maneuvers, and careful management of inspired oxygen concentrations. During anesthesia, ventilation is redistributed from dependent to nondependent lung regions, while perfusion increases progressively from ventral to dorsal regions. Hypoxic pulmonary vasoconstriction (HPV) reduces perfusion in atelectatic regions but is inhibited by inhalational anesthetics. A three-compartment lung model, including normal V/Q, low V/Q, and shunt regions, effectively explains oxygenation impairment during anesthesia. Techniques such as the multiple inert gas elimination technique (MIGET) and single-photon emission computed tomography (SPECT) provide detailed insights into V/Q relationships. Individualized approaches, including continuous positive airway pressure (CPAP) during induction and appropriate PEEP during and after anesthesia, can optimize lung function and minimize postoperative complications.
**Keywords:** anesthesia, Lung Function, respiratory system

**Introduction**

Anesthesia is known to cause respiratory impairment, irrespective of whether the patient is spontaneously breathing or mechanically ventilated. A notable consequence of most anesthetics is a reduction in functional residual capacity (FRC), primarily due to the loss of muscle tone. This decrease in FRC facilitates airway closure and the resorption of gas in regions behind obstructed airways, ultimately resulting in atelectasis (Hedenstierna & Rothen, 2012). To mitigate the risk of hypoxemia, it is a common practice to supplement inspired gas with oxygen, aiming for a fraction of inspired oxygen ($FIO_2$) within the range of 0.3–0.4. Some recommendations even suggest using $FIO_2$ levels as high as 0.8, based on the premise that it could reduce the likelihood of wound infections (Hovaguimian et al., 2013). However, there is no universal consensus on this approach.

Airway and alveolar collapse caused by anesthesia may initiate inflammatory responses in the lung (van Kaam et al., 2004). Additionally, mechanical ventilation itself can exert detrimental effects on the lung by inducing stress and strain, although the severity of these effects may depend on the duration and magnitude of exposure (González-López et al., 2012; Protti et al., 2013). The combined effects of anesthesia and mechanical ventilation can persist into the postoperative period, contributing to pulmonary complications, which are reported to occur in a range varying from a few percent to as high as 40%, depending on factors such as patient characteristics, surgical procedure, and the criteria used to define complications (Serpa Neto et al., 2014). These postoperative complications will be explored in greater depth in a subsequent section. It is essential to note that this discussion predominantly focuses on the respiratory effects of anesthesia during mechanical ventilation, as distinguishing the individual contributions of anesthesia and ventilation can be challenging. While thoracic anesthesia involving one-lung ventilation is outside the scope of this chapter, readers interested in this topic are encouraged to refer to recent comprehensive reviews (Kozian & Schilling, 2014).

**Respiratory Muscle Tone and Lung Volume**

In adult humans, the resting lung volume, represented by FRC, is reduced by approximately 0.8–1.0 liters when transitioning from an upright to a supine position. The induction of general anesthesia further decreases FRC by an additional 0.4–0.5 liters except in cases where ketamine is used as an anesthetic, as will be discussed later. Consequently, end-expiratory lung volume decreases from around 3.5 liters in an awake, upright position to approximately 2 liters in the supine, anesthetized state, a value close to or even equivalent to the residual volume. General anesthesia induces a reduction in FRC even when spontaneous breathing is maintained. Notably, this decrease in FRC occurs irrespective of whether the anesthetic agents are administered via inhalation or intravenously. The introduction of muscle paralysis and the use of mechanical ventilation do not further exacerbate the reduction in FRC.

The diminished FRC contributes to altered ventilation distribution and impaired oxygenation, as will be elaborated upon in subsequent sections. The reduction in FRC is largely attributable to the loss of respiratory muscle tone, which disrupts the balance between the elastic recoil forces of the lung and the outward forces exerted by the chest wall, leading to a lower equilibrium of lung and chest wall volumes. Anesthesia administered with ketamine, which maintains muscle tone, does not result in a reduction in FRC.

Age, body position (upright versus supine), and the effects of anesthesia all influence FRC. FRC tends to increase with age, provided that weight and height remain constant over time, although this may not always be the case. Weight gain, in particular, can counteract the effect of age-related loss of elastic tissue in the lungs by further lowering FRC.

The primary cause of the decrease in FRC during anesthesia is the upward displacement of the diaphragm, with only a minimal contribution from the reduced transverse area of the thorax. The motion of the diaphragm varies depending on whether it is actively functioning as a respiratory muscle or passively serving as a separating membrane between the thoracic and abdominal cavities. During active inspiration, the dorsal portion of the diaphragm moves caudally to a greater extent than the upper regions. This difference can be attributed to two key factors. Firstly, the muscle fibers in the dorsal region are more elongated due to the higher abdominal pressure exerted on dependent fibers, enabling them to develop greater force or undergo more significant shortening during inspiration a phenomenon akin to the Starling law of the heart. Secondly, the dorsal or crural region of the diaphragm contains a greater density of muscle fibers compared to the anterior portion, thereby generating more force.

In contrast, during mechanical ventilation, where the diaphragm remains passive and flaccid, the anterior, nondependent part of the diaphragm experiences greater displacement than the posterior, dependent regions during inspiration. This occurs because the hydrostatic pressure in the abdominal cavity is lower in the anterior regions, making it easier for the upper diaphragm to move as pressure is generated and the lungs expand. These differences in diaphragm motion also influence the distribution of ventilation within the lungs, which will be discussed in subsequent sections.

**Compliance and Resistance**

The static compliance of the total respiratory system ($C_{rs,st}$), which includes both the lungs and the chest wall, is significantly reduced during anesthesia, decreasing from an average of 95 ml/cm $H_2O$ to 60 ml/cm $H_2O$.

Numerous studies have investigated lung compliance under anesthesia, with the majority reporting a decrease in comparison to the awake state. For instance, pooled data from various studies revealed that static lung compliance dropped from a mean value of 187 ml/cm $H_2O$ in the awake state to 149 ml/cm $H_2O$ during anesthesia.

In general, the resistance of the total respiratory system and the lungs increases during anesthesia, whether the patient is spontaneously breathing or mechanically ventilated. However, the interpretation of resistance data has been complicated by varying experimental conditions between the awake and anesthetized states. To date, a study allowing direct comparison of resistance under identical volume and flow conditions remains unavailable. It is possible that the observed increase in lung resistance during anesthesia is a secondary effect of reduced FRC.

**Airway Closure**

Airway closure during expiration is a common physiological phenomenon (Milic-Emili et al., 2007). It becomes more pronounced with advancing age and occurs earlier during expiration. The reduction in FRC associated with anesthesia exacerbates airway closure. Some airways may close transiently during expiration and reopen during the subsequent inspiration, while others may remain continuously closed. Closure occurs when the extraluminal pressure exceeds the intraluminal pressure. Given the higher pleural pressure in dependent lung regions compared to nondependent ones, airway closure predominantly affects the dependent regions. This hinders ventilation, and when perfusion persists, it leads to a ventilation-perfusion mismatch (low V/Q).

Ventilation in the "airway closure" zone is reduced relative to perfusion, which explains the mismatch. Moreover, persistently closed airways can lead to resorption atelectasis, a topic that will be discussed in detail later. In anesthetized, normal-weight individuals, airways typically begin to close at an expiratory pressure of approximately 6 cm $H_2O$, which represents the threshold needed to keep the airways open. In obese individuals, higher pressures are required to maintain airway patency.

The prevalence of airway closure increases with age, although the relationship is biphasic rather than linear. Airway closure is more common in young children compared to teenagers. From around age six, when such measurements become feasible, a linear correlation emerges between closing volume as a fraction of vital capacity or closing capacity as a fraction of FRC and increasing age up to approximately 20 years. By late adolescence, even a maximal expiratory effort seldom triggers airway closure. This suggests that the optimum period for open airways and maximal alveolar gas exchange is around age 20. The biphasic relationship can be attributed to the growth of the lungs, where the structural support for narrower airways is weaker in young children compared to fully developed lungs at 20 years. After this age, the gradual loss of elastic tissue in the lungs increases the susceptibility of airways to collapse.

Airway closure significantly impairs oxygenation during anesthesia, with its impact being inversely proportional to the fraction of inspired oxygen ($FIO_2$). At high $FIO_2$ levels, even poorly ventilated lung regions are capable of oxygenating pulmonary capillary blood to near-normal levels, although this is slightly less efficient due to elevated alveolar $PCO_2$. However, high $FIO_2$ accelerates gas resorption, thereby promoting atelectasis and increasing shunt formation.

**Atelectasis**

In their seminal work, Bendixen and colleagues introduced "a concept of atelectasis" as a contributing factor to impaired oxygenation during anesthesia. However, conventional chest X-rays failed to detect atelectasis. The advent of computed tomography (CT) imaging provided clarity, revealing densities in dependent lung regions of anesthetized pediatric and adult patients. Subsequent morphological studies in various animal models confirmed these densities as atelectasis.

Atelectasis occurs in approximately 90% of patients undergoing anesthesia, irrespective of whether spontaneous or mechanical ventilation is employed or whether the anesthetics are intravenous or inhalational (Hedenstierna & Edmark, 2010). On CT scans, the atelectatic regions near the diaphragm typically occupy 3–4% of the total lung area but can exceed 15–20%. The volume of collapsed lung tissue is even greater, as atelectatic areas mainly consist of lung tissue, while aerated regions include both air and tissue. Consequently, 10–20% of the lung commonly collapses at the lung base during routine anesthesia, prior to surgical intervention. Abdominal surgery has minimal additional impact on atelectasis, but the lung collapse can persist for several days postoperatively. Following thoracic surgery and cardiopulmonary bypass, more than 50% of the lung may remain collapsed for several hours after surgery. The extent of atelectasis decreases toward the apex of the lung, which is generally spared and remains fully aerated. Atelectasis is thought to act as a focal point for infection and may contribute to pulmonary complications.

Extensive research involving patients aged 20–70 years has shown that atelectasis formation does not increase with age. Limited studies conducted on infants and pediatric patients using CT imaging during anesthesia suggest that these younger groups exhibit equal or even greater percentages of atelectasis relative to transverse thoracic area compared to other age groups.

A notable condition that promotes greater atelectasis formation is pneumoperitoneum. Paradoxically, despite the increase in atelectasis, arterial oxygenation often improves during $CO_2$ pneumoperitoneum due to a reduction in

shunt. This paradox is explained by an efficient redistribution of blood flow away from collapsed lung regions, likely mediated by enhanced hypoxic pulmonary vasoconstriction (HPV) triggered by $CO_2$. Experimental studies using single-photon emission computed tomography (SPECT) in a porcine model demonstrated a more effective shift of perfusion away from dependent juxtadiaphragmatic lung regions than the corresponding reduction in ventilation (Strang et al., 2010). However, this effect was not observed when air, rather than $CO_2$, was used to inflate the abdomen (Strang et al., 2013).

**Prevention of Atelectasis**

The primary cause of atelectasis during anesthesia is airway closure, a crucial factor to consider when devising strategies to prevent atelectasis or reopen collapsed lung tissue. While lung compression might be suspected as a significant contributor, evidence suggests otherwise. Airway closure typically precedes alveolar collapse when lung volume decreases. A secondary factor necessary for atelectasis formation is the resorption of gas trapped behind closed airways. The rate of gas resorption, and consequently atelectasis formation, increases with higher oxygen concentrations. For instance, the relative contributions of lung compression versus gas resorption in acute respiratory distress syndrome (ARDS) patients remain an area of inquiry. However, in the context of relatively short anesthetic durations, both a decrease in FRC and high oxygen concentration are requisite for alveolar collapse.

Positive end-expiratory pressure (PEEP) is a straightforward method to augment lung volume and airway dimensions. Depending on the magnitude of PEEP applied, airways can be reopened; however, it is less certain whether a given PEEP level is sufficient to recruit collapsed alveoli. For instance, airway closure in a normal-weight, anesthetized individual occurs at a transpulmonary pressure approximately 6 cm $H_2O$ higher than baseline and the threshold is likely higher in obese individuals. A practical, though untested, guideline suggests using a PEEP of 6 cm $H_2O$ for individuals with a body mass index (BMI) below 25 kg/m², increasing to 8 cm $H_2O$ for a BMI up to 30 kg/m², with higher levels required for more obese patients to maintain airway patency. Applying PEEP before atelectasis develops may help prevent its formation.

Interestingly, a PEEP of 10 cm $H_2O$ has been shown to consistently reopen collapsed lung tissue. This process occurs within minutes, though it may be attributed more to increased inspiratory airway pressure than PEEP itself. Nevertheless, not all previously collapsed tissue is reopened, even with prolonged application. Moreover, the improvement in arterial oxygenation is often disproportionate to the reduction in atelectasis, likely due to the redistribution of blood flow to more dependent, atelectatic lung regions. PEEP levels exceeding 10 cm $H_2O$ may also cause hemodynamic disturbances ("High versus Low Positive End-Expiratory Pressure during General Anaesthesia for Open Abdominal Surgery (PROVHILO Trial)," 2014). This does not negate the use of PEEP but highlights the need for an optimal, individualized approach that balances recruitment and circulatory effects. Techniques to estimate changes in resting lung volume induced by PEEP have been explored (Grivans et al., 2011).

A "sigh," characterized as a double tidal volume breath, has been proposed to reopen collapsed lung tissue and enhance gas exchange in both intubated and non-intubated patients. However, normal tidal breathing or a "sigh" with an airway pressure of up to 20 cm $H_2O$ does not reduce the extent of atelectasis. A sustained inflation of the lungs at an airway pressure of 30 cm $H_2O$ decreases atelectasis to approximately half its initial size, but further inflations at the same pressure yield only minimal additional lung tissue recruitment. In anesthetized adults with healthy lungs, complete reopening of collapsed lung tissue requires an airway pressure (recruitment pressure) of 40 cm $H_2O$. In contrast, morbidly obese patients with increased chest-wall elastance require higher pressures to achieve equivalent transpulmonary pressures. For instance, airway pressures as high as 55 cm $H_2O$, maintained for 10 seconds, have successfully recruited lung tissue in patients with a BMI >45 kg/m² (Reinius et al., 2009).

The interaction between time and pressure during recruitment maneuvers is complex, and the optimal time frame may vary depending on the recruitment pressures applied. As previously mentioned, PEEP of 10 cm $H_2O$ may reopen collapsed lung tissue, and this relationship should be considered.

**Oxygen and Atelectasis During Induction of Anesthesia**

Preoxygenation is a standard procedure employed to prevent hypoxemia in the event of a difficult intubation, ensuring maximum safety for the anesthetist. However, it is important to consider the potential for atelectasis formation during this process, as it may shorten the "apnea tolerance time," defined as the duration before hypoxemia develops.

Avoiding preoxygenation and using ventilation with 30% oxygen instead of 100% oxygen prevents atelectasis formation during the induction and subsequent anesthesia. Studies have shown that atelectasis develops in all patients preoxygenated with 100% oxygen, is significantly smaller with 80% oxygen, and is nearly absent with 60% oxygen. However, the reduced atelectasis associated with lower oxygen concentrations during induction is only temporary. For example, patients ventilated with 80% oxygen and PEEP ≤3 cm $H_2O$ during induction had comparable levels of atelectasis to those ventilated with 100% oxygen after 30 minutes (Edmark et al., 2011). This occurs because the gas trapped behind closed airways consists of 80% oxygen and is resorbed over time, ultimately resulting in atelectasis. Recruitment maneuvers using lower oxygen concentrations, such as 40%, can

replenish closed regions with less oxygen-rich gas, thereby slowing resorption atelectasis for the remainder of the anesthesia period.

Inducing anesthesia while maintaining continuous positive airway pressure (CPAP) may prevent the reduction in functional residual capacity (FRC) and subsequent atelectasis formation. Using CPAP allows full oxygen utilization while increasing lung volume, creating a larger oxygen reservoir and providing additional safety during complicated intubations. While the typical duration of tracheal intubation at atmospheric pressure is too brief to cause significant lung collapse, prolonged or difficult intubations may lead to collapse. In such cases, prior CPAP use is beneficial, as it extends the time to hypoxemia (Bouvet et al., 2014).

**Oxygen and Atelectasis During Anesthesia**

Ventilating the lungs with pure oxygen following a vital capacity maneuver, which reopens collapsed lung tissue, results in rapid reformation of atelectasis. In contrast, using 40% oxygen in nitrogen for ventilation slows atelectasis formation, with only 20% of the initial atelectasis reappearing 40 minutes after the maneuver. Therefore, maintaining moderate $FIO_2$ levels during anesthesia can prevent atelectasis. If higher oxygen concentrations are necessary, they should be administered alongside PEEP ventilation.

**Oxygen and Atelectasis During Emergence from Anesthesia**

High oxygen concentrations are often used during the emergence phase of anesthesia to mitigate hypoxemia risks during wake-up. This is commonly combined with airway suctioning to remove secretions. However, this combination of oxygenation and suctioning significantly contributes to atelectasis formation, with no other maneuver comparable in its atelectasis-inducing potential.

These findings have prompted research into employing recruitment maneuvers at the conclusion of surgery and anesthesia. Inspired oxygen levels play a critical role in this context. For example, performing a recruitment maneuver followed by ventilation with 100% oxygen—common in routine anesthesia—leads to new atelectasis, as demonstrated in postoperative CT scans. Conversely, ventilation with lower $FIO_2$ levels after recruitment prevents new atelectasis formation. Another strategy to minimize postoperative atelectasis is to maintain PEEP until extubation and then continue with CPAP for a brief period, during which inspired $FIO_2$ is reduced to 30% or ambient air. A small study employing this technique reported a reduction in atelectasis to approximately 40% compared to controls without PEEP or CPAP, as evaluated by CT 25 minutes post-wake-up (Edmark et al., 2014).

**Ventilation**

**Dead Space**

It is widely believed that dead space increases during anesthesia with mechanical ventilation. However, this perception may not be entirely accurate. Anatomical dead space decreases due to airway intubation, while apparatus dead space, primarily from tubing, increases. Alveolar dead space may appear elevated, but this is more likely a result of reduced blood flow relative to ventilation in nondependent upper lung regions. Despite this, $CO_2$ elimination is impaired during anesthesia.

An additional mechanism contributing to elevated arterial $CO_2$ levels is the pulmonary shunt. This process allows mixed venous blood, containing higher $CO_2$ concentrations, to bypass the lungs and enter the arterial circulation, thereby increasing $PaCO_2$ and widening the arterial-to-end-tidal $CO_2$ difference ($PaCO_2$-$PECO_2$). Although often referred to as “shunt dead space,” this phenomenon is unrelated to physiological dead space.

**Distribution of Ventilation**

In anesthetized and mechanically ventilated supine individuals, a redistribution of inspired gas from dependent to nondependent lung regions has been observed. In awake individuals (upper right inset), both ventilation and perfusion increase progressively toward the base of the lung. However, during anesthesia and mechanical ventilation, ventilation is predominantly directed to nondependent lung regions, with a gradual reduction in regional ventilation toward the lower portions of the lung. In the most dependent areas, ventilation is absent, corresponding to regions of atelectasis, as confirmed by concurrent CT imaging.

Application of PEEP increases ventilation in dependent lung regions in anesthetized individuals in the lateral position, resulting in a ventilation pattern similar to that of awake individuals. Comparable findings of a more balanced ventilation distribution between nondependent (upper) and dependent (lower) lung regions have been reported in supine, anesthetized individuals following a prior inflation of the lungs. This is likely due to the recruitment of atelectatic lung units in dependent regions and the reopening of closed airways in the lower lung regions. Additionally, further expansion of upper lung regions reduces their compliance, redirecting ventilation toward the dependent regions.

**Distribution of Lung Blood Flow**

In anesthetized, mechanically ventilated patients, perfusion increases progressively from ventral to dorsal lung regions, with a slight decrease in blood flow in the most dependent areas. These lowermost regions are often collapsed (atelectatic), as confirmed by simultaneous CT imaging, yet they remain perfused. These findings align with early lung perfusion models described in the 1960s.

As previously discussed, PEEP redistributes blood flow toward dependent lung regions thereby increasing the pulmonary shunt. Conversely, perfusion in nondependent regions may be diminished, leading to a dead space-

like effect. PEEP also reduces venous return to the right heart, thereby decreasing cardiac output. While PEEP may influence pulmonary vascular resistance, its effect on cardiac output is generally less pronounced.

**Hypoxic Pulmonary Vasoconstriction (HPV)**

HPV serves to reduce perfusion in hypoxic (atelectatic) lung regions, with vessel compression and kinking playing only minor roles. Inhalational anesthetics inhibit HPV in isolated lung models; however, intravenous anesthetics such as barbiturates do not demonstrate this effect. Findings from human studies vary, likely due to the experimental complexity and the concurrent influence of multiple variables. Changes in myocardial contractility, cardiac output, vascular tone, blood volume distribution, blood pH, $CO_2$ tension, and lung mechanics can obscure the HPV response.

In studies where cardiac output remains relatively stable, inhalational anesthetics such as isoflurane and halothane have been shown to depress the HPV response by approximately 50% at two MAC (minimum alveolar concentration). Desflurane is expected to have a similar effect (Soares et al., 2012). Additionally, HPV is influenced by other factors, including alkalosis, hypothermia, and the use of vasodilators such as nitroprusside and calcium channel antagonists (Nagendran et al., 2006).

**Ventilation–Perfusion Relationship**

A relationship between the extent of atelectasis and pulmonary shunt, as determined by the multiple inert gas elimination technique (MIGET), has been established in several studies. A regression equation derived from data on 45 patients undergoing inhalational anesthesia quantifies this correlation, calculated as follow:

Shunt = 0.8 atelectasis + 1.7

$r = 0.81$, $p < 0.01$;

where shunt is expressed as a percentage of cardiac output, and atelectasis, assessed via CT, is expressed as a percentage of the transverse lung area near the diaphragm.

Through the combination of CT scanning and SPECT imaging, the localization of shunt within atelectatic areas has been further corroborated.

Interestingly, "true" shunt values, as measured by MIGET, do not exhibit an age-related increase. However, the standard "shunt" equation, which utilizes arterial and mixed venous oxygen content alongside alveolar gas oxygen concentration, demonstrates an age dependency of shunt or venous admixture. This distinction arises because venous admixture includes perfusion of "low V/Q" lung units, potentially caused by airway closure during respiration. The degree to which venous admixture accounts for low V/Q regions depends on the inspired oxygen fraction (FIO2). Higher FIO2 reduces the impact of low V/Q regions on venous admixture calculations, but these regions may collapse due to gas resorption, converting them to shunt regions. A robust correlation has been identified between age and the combined measure of "true" shunt and low V/Q perfusion (venous admixture). Overall, approximately 75% of oxygenation impairment during anesthesia is attributable to atelectasis and airway closure combined.

A three-compartment lung model effectively explains oxygenation impairment during anesthesia. This model includes a compartment with normal ventilation and perfusion, a compartment with airway closure that reduces ventilation (low V/Q), and a compartment with collapsed lung (atelectasis) that receives no ventilation (shunt).

Detailed insights into ventilation–perfusion (V/Q) relationships can be obtained using MIGET and SPECT. Comparisons of these techniques in anesthetized individuals show good correlations, with MIGET providing higher resolution of V/Q distributions, while SPECT offers spatial visualization of ventilation and perfusion.

Numerous studies employing MIGET have demonstrated key findings, such as increased V/Q dispersion and the development of shunt during anesthesia. Increased V/Q dispersion reflects uneven ventilation relative to perfusion due to airway narrowing and closure (secondary to a decrease in FRC) and perfusion redistribution caused by increased intrathoracic pressure. Shunt, on the other hand, corresponds to atelectasis. Although V/Q ratio inhomogeneity increases with age, shunt and atelectasis do not. Notably, anesthesia exacerbates perfusion inhomogeneity to an extent equivalent to approximately 20 years of aging, although this effect is expected to reverse postoperatively.

As previously mentioned, an FIO2 of around 0.4 is commonly used during general anesthesia with mechanical ventilation. In a study by Anjou-Lindskog et al., anesthesia was induced with air (FIO2 = 0.21) in middle-aged to elderly patients undergoing intravenous anesthesia before elective lung surgery. Minimal shunt (1–2%) was observed, despite an increase in perfusion inhomogeneity. These findings align with earlier observations that low FIO2 during anesthesia induction leads to negligible atelectasis. When FIO2 was increased to 0.5 in the same study, shunt rose to 3–4%.

Similarly, another study on elderly patients receiving halothane anesthesia found that increasing FIO2 from 0.53 to 0.85 resulted in a shunt increase from 7% to 10% of cardiac output. This suggests that ventilation–perfusion distribution is partially dependent on FIO2, potentially due to the attenuation of the HPV response at higher FIO2 or the progression of atelectasis and shunt formation in lung units with low V/Q ratios.

**Conclusion**

Anesthesia profoundly impacts respiratory physiology, primarily through reductions in functional residual capacity (FRC), airway closure, and atelectasis formation. These changes disrupt ventilation–perfusion (V/Q)

relationships, impair oxygenation, and contribute to postoperative pulmonary complications. Mechanisms such as pulmonary shunt and the redistribution of ventilation to nondependent lung regions further exacerbate these issues.

Understanding the interactions between airway mechanics, lung compliance, and perfusion is crucial in mitigating the adverse effects of anesthesia. Techniques like positive end-expiratory pressure (PEEP), recruitment maneuvers, and careful management of inspired oxygen concentrations can minimize atelectasis and improve V/Q matching. Moreover, individualized approaches, including the use of continuous positive airway pressure (CPAP) during induction and appropriate PEEP during and after anesthesia, are effective in optimizing lung function.

Future research should focus on refining strategies to balance oxygenation with minimizing pulmonary complications, ensuring safer anesthesia practices across diverse patient populations. The integration of advanced imaging techniques like CT and SPECT enhances our understanding of V/Q dynamics, paving the way for improved clinical interventions.

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