

Intra-Operative Hypoxemia: Causes

Anesthetic Pearls: Anesthetic Implications and Management of Intra-Op Hypoxemia

1. Mechanical failure of anesthesia apparatus to deliver oxygen to the patient

Causes include: disconnection of the patient from oxygen supply, empty oxygen cylinder, substitution of non-oxygen cylinder at oxygen yoke, failure of gas pressure in a piped oxygen system, fractured or sticking flowmeters, transposition rotameter tubes, fresh gas line disconnection from machine to in-line hosing.

2. Mechanical failure of endotracheal tube (main stem bronchus intubation)

Main stem intubation results in absence of ventilation of contra-lateral lung causing increase in shunting and decrease in PaO₂. A well positioned ETT in the trachea may enter a bronchus after the patient or the head of the patient is turned or moved into a new position. Flexion of the head causes the ETT to move caudad and extension of the head causes it to move cephalad. Positioning the patient in steep Trendelenberg (> 30°) causes cephalad shift of the carina and may cause a previously fixed ETT to become lodged in a main stem bronchus.

3. Hypoventilation

Patients under general anesthesia have reduced spontaneous tidal volume that results in atelectasis and FRC reduction. Decreased minute ventilation decreases the overall V/Q ratio of the lung leading to a decrease in PaO₂.

4. Decrease in FRC

Induction of general anesthesia causes decrease in FRC (~ 0.4 L) which leads to a decrease in compliance. Change from the upright to the supine surgical position causes an additional decrease in FRC (0.5 - 1.0 L). The use of muscle relaxants causes paralysis of the diaphragm, which results in a cephalad shift of the diaphragm and decreased FRC.

5. Inhibition of HPV (Hypoxic Pulmonary Vasoconstriction)

The pulmonary circulation has inherently poor smooth muscle surrounding it and any condition that increases the pressure against which the vessels constrict will reduce HPV. Conditions that increase the pulmonary artery transmural pressure include mitral stenosis, volume overload, low FIO₂, thromboembolism, hypothermia, and vasoactive drugs.

6. Hyperventilation

Hypocapnic alkalosis causes a decrease in cardiac output, increased O₂ consumption, left shift of the oxy-Hb curve, decreased HPV, and increased airway resistance / decreased compliance. Each of these factors promote a decrease in PaO₂.

7. Supine position, immobility, and excessive IV fluid administration

During surgery, patients are often kept supine and immobile for prolonged time periods. This results in some of the lung being continually dependent and below the left atrium, causing it to be maintained in West Zone 3 or 4 characteristics. The dependent position also predisposes the lung to fluid accumulation. Combined with excessive fluid administration, these conditions are sufficient to promote transudation of fluid into the lung and result in pulmonary edema and a decreased FRC (leading to PaO₂ reduction).

8. Absorption atelectasis due to high FIO₂

Atelectatic shunting during high FIO₂ breathing is caused by a large increase in O₂ uptake by lung units with low V/Q ratios, converting these areas into shunt units. Absorption atelectasis is most likely to occur when the FIO₂ is high, V/Q ratio is low, long exposure time, and low mixed venous blood content.

9. Decreased cardiac output

A decreased CO in the presence of a constant O₂ consumption, or an increased O₂ consumption in the presence of a constant CO, or a decreased CO and an increased O₂ consumption all result in a lower mixed venous O₂ content which leads to a lower O₂ content of arterial blood.