

Conclusion

General anesthesia produces a profound effect on overall ventilation, pulmonary perfusion, and the relationship each has to the other. Even patients who are able to breathe spontaneously during general anesthesia have an increase in dead-space ventilation in the non-dependant areas of the lung as well as increases in shunt in the dependant areas of the lung.

Diaphragmatic travel is altered during general anesthesia and is altered even more if the clinical situation calls for neuromuscular blocking agents. Such agents may result in an increase in the Aa gradient of up to 15 mmHg.

The application of pressure ventilation, either PCV for those patients who cannot or will not breathe spontaneously or PSV for those able to breathe spontaneously can be employed to potentially improve ventilation and decrease the ventilation/perfusion mismatch produced by general anesthesia.

Additional reading:

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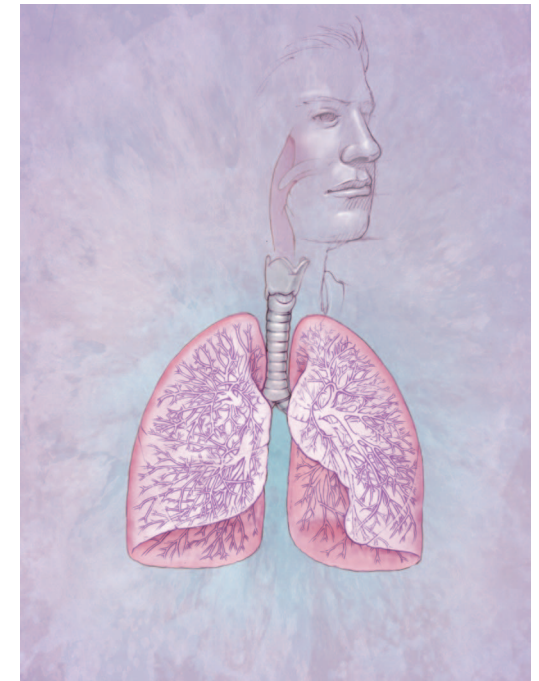
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Ventilation and Perfusion during Mechanical Ventilation

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From the Ventilation Series

Ventilation and perfusion during mechanical ventilation

Though modern anesthesia practice and ventilation have improved dramatically in the past decades some challenges remain. Among these challenges is how to address the impact that mechanical ventilation has on both ventilation and perfusion. The human lung was designed for breathing, not for being stet ventilated. This **Clinical Focus**, produced by the Department of Clinical Affairs, will discuss ventilation and perfusion during mechanical ventilation.

In an awake, erect person, the lungs can be divided into three zones as described by West. The upper third, Zone 1, represents an area with more ventilation than perfusion. In this zone, the alveolar pressure (P_A) exceeds the pulmonary arterial pressure (P_{Pa}) and the P_{Pa} exceeds the pulmonary venous pressure (P_{Pv}). This ventilation/perfusion profile, $P_A > P_{Pa} > P_{Pv}$, produces more dead-space ventilation than the other two zones. In Zone 1, alveoli are well ventilated but poorly perfused. The middle third, Zone 2, represents a slightly different ventilation/perfusion profile in which dead-space ventilation is diminished in favor of greater perfusion. In this area $P_{Pa} > P_A > P_{Pv}$ and the relationship between ventilation and perfusion is almost equal. The final third, Zone 3, is defined as the area where $P_{Pa} > P_{Pv} > P_A$. This zone has the greatest amount of perfusion and the least amount of ventilation. This profile represents the area where pulmonary shunt is greatest.

While these regional differences are minimal in the awake, standing person the changes induced by general anesthesia, skeletal muscle relaxation

and mechanical ventilation may be quite profound. With assumption of the supine position the traditional location of these zones is rotated. They are no longer arranged from apex to base but rather ventral to dorsal. Other changes in ventilatory dynamics also occur in the supine position. The diaphragmatic travel and the rotation of the ribs are affected. The changes in diaphragmatic travel produce significant changes to the ventilation/perfusion profile of the intact lung. During quiet breathing in the supine position, the diaphragm is pulled caudally with the dorsal portion traveling farther than the ventral portion. In the anesthetized patient, even during spontaneous breathing, the ventral portion of the diaphragm is retracted caudally and the dorsal portion retracts very little. This change effectively increases dead-space ventilation in the ventral zone of the lung and increases shunt in the dorsal zone. Such a ventilation/perfusion mismatch clearly suggests the need to assist ventilation even in the anesthetized patient who is able to breathe spontaneously. If clinical conditions require the addition of neuromuscular blocking agents, the effects are even more profound and may result in an arterial/alveolar (Aa) carbon-dioxide gradient as high as 12-15 mmHg. Under normal circumstances, the standing spontaneously breathing person will have an Aa gradient of 4 mm Hg. During general anesthesia even with an end-tidal CO_2 ($P_{et}CO_2$) value of 36 mmHg, the patient's arterial CO_2 value may be as high as 50 mmHg effectively producing an acidotic state even with what would appear to be adequate ventilation as reported by the $P_{et}CO_2$ monitor.

What strategies exist to overcome such ventilation/perfusion mismatches?

Among the various ventilation strategies that attempt to address the increase in both dead-space ventilation and in pulmonary shunt caused by general anesthesia are Pressure Control Ventilation (PCV) for the patient who must be actively ventilated, and Pressure Support Ventilation (PSV) for patients who are able to breathe spontaneously.

With either form of pressure ventilation, there is also a change in the duration that inhalation gases are present in the lungs and available for diffusion across the alveolar/capillary membrane. This is the result of the gas flow pattern present in both forms of ventilation, a pattern that delivers the majority of gases to the alveoli early during the inspiration time. This pattern is called a decelerating flow pattern. Effectively, this pattern overcomes much of the shunt produced in the dependant portion of the lung without preferentially ventilating the non-dependant portions more susceptible to dead-space ventilation.

The decelerating flow pattern also accounts for the commonly noted differences between Volume Control and Pressure Control Ventilation modes when the rates and tidal volumes are identical. In Pressure Mode, more of the gases are present in the alveoli longer during the inspiratory period which may improve ventilation.