Hypoxic Pulmonary Vasoconstriction

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Introduction

Hypoxic pulmonary vasoconstriction (HPV) is characterized by a local reaction to hypoxic areas of the lung. HPV is a reflexive contraction of vascular smooth muscle in the pulmonary circulation. This reflex contraction is in response to low partial pressure of oxygen (Lamb & Slinger, 2015).

This vasoconstriction is seen in the vasculature of the pulmonary system and is the major difference which separates it from the systemic circulation, which usually vasodilates in response to hypoxia (Lamb & Slinger, 2015).

My intent of this research project is to explain the pathophysiological processes of HPV and understand the implications of anoxia related to it.

Through this research I will be able to provide a safe and personalized plan of care for any patient, while taking into account the risks of HPV.

Characteristics & Symptoms

- The result of only one portion of the lung being affected, is that blood is shunted from the hypoxic region to the well ventilated portion of the lung (Arturio & Yao, 2012).
- If the entire lung is affected by alveolar hypoxia however, then there is widespread pulmonary vasoconstriction, leading to pulmonary hypertension (Arturio & Yao, 2012).
- The symptoms of HPV depend of the degree of hypoxic lung tissue.
- HPV is a local reaction occurring in hypoxic areas of the lung.
- It may be localized due to various causes including regional anesthetics. It can also be diffuse, affecting both lungs, as seen in high altitude pulmonary edema.
- HPV is triggered by alveolar hypoxia, not arterial hypoxemia as one may assume.
- The peak effect of alveolar hypoxia is seen within 15 minutes.
- Many elements can affect HPV including cardiac output, medications, PIH and Prez.

Pathophysiology

- HPV is the autoregulatory mechanism which prevents the ventilation to perfusion mismatch caused by alveolar hypoxia in order to improve gas exchange and arterial oxygenation.
- HPV is active in utero, reducing the hypoxic pulmonary vasoconstriction (Arturio & Yao, 2012).
- Excessive tidal volume worsens the ventilation to perfusion mismatch (Lamb & Slinger, 2015).
- The cellular mechanism involves a redox based oxygen sensor in the smooth muscle cells of the pulmonary arteries. Hypoxia then reduces production of activated oxygen species and they act on second messengers from the oxygen sensors. Outflow is then reduced leading to inhibition of voltage dependent potassium channels further resulting in an influx of extracellular calcium which causes vasoconstriction (Nagelhout & Plaus, 2014).
- If the alveolar hypoxia persists it can lead to permanent pulmonary hypertension, which will eventually lead to our pulmonary alveoli (Arturio & Yao, 2012).

Hypoxic pulmonary vasoconstriction. The left frame shows normal alveolar ventilation and perfusion. In the right frame, reduced ventilation (thus O2 tension) in the alveoli (green) leads to a reduced perfusion because of the hypoxic pulmonary vasoconstriction mechanism.

Factors that Reduce Effectiveness of HPV

- Alkalosis
- Hypothermia
- Hypocapnia
- Excessive tidal volume
- Excessive PEEP
- Vasodilators
- Calcium channel blockers
- Phosphodiesterase inhibitors
- Hypervolemia

Anesthetic Implications

- During mechanical ventilation it is very important to match ventilation and perfusion in order to optimize gas exchange in the mechanically ventilated patient.
- Studies have shown that an occlusion of this artery can very quickly lead to a reduction of blood flow to that region of the lung by about 50% (Lamb & Slinger, 2015).
- In both asthma and chronic obstructive pulmonary disease (COPD), giving 100% oxygen worsens the ventilation to perfusion (V/Q) matching ratio.
- Many of the drugs used during anesthesia have some sort of effect on HPV. Many anesthetic drugs inhibit HPV, however, none commonly used augment it (Lamb & Slinger, 2015).
- Any drug that is a vasodilator may inhibit HPV, and all current anesthetic agents can inhibit HPV but are dose dependent (Lamb & Slinger, 2015).
- The effects of nitrous oxide on HPV are unclear at this time.

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References


Advantages

- Arteriovascular theoretical questions.
- No ventilation/vasodilatory lung to single arterial myocyte.
- Ventilated/perfused models.
- Human and animal pulmonary vascular mechanisms of acute hypoxic pulmonary vasoconstriction.
- O2 delivery to single arterial myocyte. Ventilated/perfused models.


Additional Sources


References


References


