Journal of Clinical Respiratory: Open Access

Mechanism Involved in Hypoxic Vasoconstriction in Fetus and Adults

Kawasahi Shin*

Department of Respiratory Medicine, Madda Walabu University, Robe, Ethiopia

Opinion Article

Received: 01-Mar-2023, Manuscript No. JCROA-23-93882; **Editor assigned:** 03-Mar-2023, Pre QC No. JCROA-23-93882 (PQ); **Reviewed:** 17-Mar-2023, QC No. JCROA-23-93882; **Revised:** 24-Mar-2023, Manuscript No. JCROA-23-93882 (R); **Published:** 31-Mar-2023, DOI: 10.4172/jclinresp.5.1.008

***For Correspondence:**
Dr. Kawasahi Shin, Department of Respiratory Medicine, Madda Walabu University, Robe, Ethiopia

E-mail: shin@gmail.com

Citation: Shin K. Mechanism Involved in Hypoxic Vasoconstriction in Fetus and Adults. J Clin Res. 2023;5:008.

ABOUT THE STUDY

In the developing fetus, the pulmonary vascular resistance is high. The oxygenated blood from the placenta flows through the foramen ovale and ductus arteriosus, largely bypassing the lungs. If the oxygen level in the fetus is raised by increasing maternal oxygenation, the fetal pulmonary vascular resistance falls, demonstrating that normally in the fetus active hypoxia induced vasoconstriction contributes to the high resistance. At birth, lung expansion and rising oxygen levels cause a rapid drip in pulmonary vascular resistance and subsequent remodelling of the resistance pulmonary arteries. Thus from the ontogenetic point of view HPV can also be regarded as the other side of the coin to normoxic pulmonary vasodilation. Once out of the uterus the lung displays HPV in response to alvoelar hypoxia. In humans, hypoxic vasoconstriction can be defined as rapid, monophasic reversible increase in pulmonary vascular resistance due to the contraction of small muscular pulmonary arteries with an internal diameter of approximately 200 to 600 micrometers and in response to physiological levels of hypoxia.

The considerable individual variability in the magnitude of HPV between individuals might be based on genetics and adaptive mechanisms as well as in reptiles. Hypoxic vasoconstriction depends largely on the alveolar and not on the mixed venous pO_2 and starts within seconds of the onset of the airway hypoxia, by a decrease in alveolar oxygen tension below a threshold fractional inspired oxygen concentration of 10%. Subsequently a fall in the alveolar oxygen partial pressure below 50 mm Hg doubles pulmonary vascular resistance. In intact dogs, rabbits, the isolated ferret lungs, rabbit lungs as well as the isolated pulmonary arteries, the response to alveolar hypoxia shows a biphasic character with an early pulmonary arterial pressure peak and a more protracted secondary [pressure elevation. The acute initial transient vasoconstrictor phase 1 is independent of the endothelium. For the slowly developing increase in vascular tone both the endothelium and calcium sensitization possibly via activation of Rho-kinases are required. Hypoxia appear to activate independent of blood borne factors or influences that require the central nervous system as HPV can be demonstrated in isolated perfused lungs and isolated pulmonary arteries. According to current knowledge neither angiotension, prostaglandins thromboxane nor arachdonic acid lipoxygenase products directly mediate the mechanism of acute HPV. In contrast, hypoxic vasoconstriction is blocked by calcium antagonists, reduced by endothelin antagonists and alpha-adrenergic antagonists and modulated by serotonin, suggesting not only involvement of voltage dependent calcium channels and extracellular calcium but also the participation of the serotonin and endothelin pathways and possibly the contribution of catecholamines. The administration of the exogenous vasodilators such as NO and prostacyclins are able to override HPV which does not necessarily suggest an endogenous role of these substances in the mechanism. If only a small region of the lung is hypoxic HPV can occur without an significant effect on pulmonary arterial pressure. However if the critical mass of the lung becomes hypoxic, as seen in many lung diseases and in high altitude exposure the subsequent pulmonary vasoconstriction contributes to pulmonary hypertension heart failure and death.