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Neuromodulation as a New Strategy in Management of Pulmonary Hypertension

Massive pulmonary embolism, sepsis, and ALI are the other main causes of acute pulmonary arterial hypertension (PAH) in the adult patient population. Preexisting pulmonary vascular and lung diseases, including Interstitial lung disease, chronic obstructive pulmonary disease and obstructive sleep apnea are other leading causes of chronic PAH. Whatever the cause, the management of critically ill patients with hemodynamically significant pulmonary hypertension remains challenging, especially in the field, away from equipped ICU and tertiary medical centers. We developed an innovative therapeutic approach by stimulation vagal nerve (VNS) which can be used in modulation vascular tone. Our study aims was focused to treat acute and chronic PAH in different animal model using VNS approach. A rodent model with PAH induced by hypoxia (FiO₂ 10%) for 3 weeks only (mild form of PAH) or hypoxia and Sugen together (Chronic form of PAH), were used. Continuous monitoring of the vital signs: Heart rate, EKG, oxygen saturation, breathing rate and both right ventricular pressure (RVP) and systemic pressure; were recorded using a computerized hemodynamic recording system. Continuous monitoring/stimulation of vagal nerve activity was established using a stimulation module controlled by the Acknowledge software (Biopac Systems). Stimulation was delivered using different matching stimulation parameters (current intensity Amplitude, pulse width htz, pulsing frequency, and pulsing duration). Our data showed that by using specific electric stimulation with specific parameters, we were able to target specific nerve fiber which can induce smooth muscle relaxation of the pulmonary vascular bed, followed a subsequent drop of RVP. Analysis of the data using beat to beat analysis showed that specific target for certain vagal nerve fibers can induce relaxation of contracted pulmonary vascular tree and significant drop of RVP, without any significant or marked change in systemic pressure, heart rate or breathing pattern (no apnea). Similar data was obtained using different murine animal models with wide range of PH severity and with variable degree of pulmonary vascular remodeling induced by Hypoxia and Sugen. We did also able to define a specific pattern of VNS (with a safe margin), which can trigger the vascular smooth muscle relaxation with none or minimal systemic or unwanted side effects (including hypotension, apnea and bradycardia). In conclusion, VNS is an innovative therapeutic modality which can be used in alleviating pulmonary pressure in animal model with variable degree and severity of PAH. Pre-clinical experiment on large animal model is warranted