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Opinion Article

Assessment of Cardiopulmonary Exercise in Pulmonary Arterial Hypertension

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DESCRIPTION

A range of pathophysiological disorders with various etiologies are together referred to as Pulmonary Hypertension (PH). It is characterized by remodeling of the pulmonary arteries, which causes the resistance of the pulmonary arteries to rise gradually, resulting in right heart failure and death. Dyspnoea on exercise is a common complaint among pulmonary hypertension patients and is a non-specific symptom that is often neglected or misdiagnosed. 85% of individuals with high-risk PH are already in an advanced stage of the disease when they are diagnosed. As a result, it is critical to screen for and promptly identify people who may have PH. The standard for the diagnosis of PH is Right Heart Catheterization (RHC) however it can potentially lead to problems. The most used screening procedure for PH in clinical settings is echocardiography.

Cardiopulmonary exercise testing is a valuable method for determining the underlying pathophysiologic processes of exercise intolerance and allows for the evaluation of the integrated cardiopulmonary response to exercise. Because pulmonary hypertension is a rare condition and has vague symptoms such dyspnea, tiredness, and exercise intolerance, patients may experience a significant delay in diagnosis.

Cardiopulmonary exercise testing in individuals with indications of both circulatory impairment and Ventilatory inefficiency may be predictive of pulmonary hypertension. The intense dyspnea that many pulmonary hypertension patients experience during exercise is caused by a number of other reasons, including peripheral muscle weakness and mechanical Ventilatory restrictions from dynamic hyperinflation.

Cardiopulmonary Exercise Testing (CPET) is a thorough approach with roles in diagnosis, therapy response, and prognosis in Pulmonary Arterial Hypertension (PAH). A useful method for identifying aberrant hemodynamics linked to exercise-induced and resting pulmonary hypertension as well as right ventricular dysfunction is the analysis of submaximal and maximal exercise data. Increased CPET granularity may aid in further risk stratifying patients to assist better individualize treatment plans and provide prognosis information.

On a cyclo-ergometer (Ergometrics Lode Medical Technology-Corival, Groningen, The Netherlands), incremental CPET was carried out using a ramp protocol that was customized with the aim of having each patient attain their maximal exercise within 8 to 10 minutes. After 60 seconds of unloaded pedaling at a speed of 60 revolutions per minute, work was gradually raised from 0 W to 4-10 W/min. Breath-by-breath study of ventilation and expiratory gases were conducted in each instance (Vmax Spectra 29S, Sensor Medics, Yorba Linda, CA). The V-slope analysis from the plot of VCO_2 *vs.* VO_2 on equal scales was used to estimate AT. Ventilatory equivalents and CO_2 and O_2 end-tidal pressures were used to validate the AT value. The AT was deemed to be unidentified if no consensus could be reached. Throughout the whole exercise, the relationship between VO_2 and work rate was assessed. The slope of the linear connection between VE and VCO_2 from 1 min after the start of loaded exercise to the completion of the isocapnic buffering period was determined to be the VE/VCO_2 slope. Additionally recorded was peak exercise ventilation as a percentage of an anticipated value (VE %). Peak systolic blood pressure divided by minute ventilation-CO_2 generation was used to calculate VP. Continuous measurements of the Electrocardiogram (ECG), blood pressure, and heart rate were made.

RHC used a Swan-Ganz pulmonary artery catheter from Edwards Life sciences in Irvine, California, to examine the patient's hemodynamics. At the conclusion of a peaceful breathing cycle, the pulmonary arterial (systolic, diastolic, and mean), right atrial, and Pulmonary Capillary Wedge Pressures (PWP) were measured. Then the oxygen saturations in the pulmonary artery, femoral artery, inferior vena cava, superior vena cava, and inferior vena cava were measured. The assumption for pulmonary vein saturation was 98%. Utilizing table-derived oxygen consumption estimates and computed oxygen contents at the appropriate various places, pulmonary and systemic flows were determined using the Fick principle. The difference between the mean pulmonary arterial pressure and the mean pulmonary capillary wedge was used to establish the trans-pulmonary pressure gradient. This led to the reporting of the Diastolic Pulmonary Gradient (DPG), which is defined as diastolic PAP minus mean Pulmonary Arterial Wedge Pressure

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(PAWP). The standard formula was used to get the indices of pulmonary and systemic vascular resistance. Pre-capillary PAH could not have been diagnosed with a PAWP>15 mmHg. When predicting PAH at RHC in individuals with suspected PAH, Ventricular Paced (VP) evaluated at CPET may offer further insight. In order to distinguish between individuals with mixed post-capillary and pre-capillary PH and those with isolated post-capillary PH, correlations with PVR and DPG may be useful.