

Pulmonary Hypertension and Exercise Training: Evidence Based Studies

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Introduction

Pulmonary Hypertension (PH) is regarded as a mean pulmonary artery pressure greater than 25 mm Hg in the setting of normal or reduced cardiac output and a normal pulmonary capillary wedge pressure. A constellation of permissive and provocative factors exists, various mechanisms are activated that lead to vascular constriction, cellular proliferation, and a prothrombotic state in varying degrees, which results in PH and its clinical sequelae [1-6].

Three mechanistic pathways are known in patients with PH. (A) The endothelin (ETn): Big-ETn is converted in endothelial cells to ETn-1 by endothelin-converting enzyme (ECE). ET-1 binds to PASM C ETnA and ETnB receptors, which ultimately leads to PASM C contraction, proliferation, and hypertrophy. ETn-1 binds to endothelial cell Also ETB receptors. (B) The prostacyclin (PGI2): The production of PGI2 is catalyzed by prostacyclin synthase in endothelial cells. In PBMCs, PGI2 stimulates adenylate cyclase, thus increasing production of cAMP from ATP, another second messenger que maintains PASM C relaxation and inhibition of proliferation PASM C. (C) The NO: NO is created in endothelial cells by type III NO synthase , which in turn induces guanylate cyclase (GC) to convert guanosine triphosphate (GTP) to cGMP, the second messenger que constitutively maintains pulmonary artery smooth muscle cell (PASM C) relaxation and inhibition of proliferation PASM C [7]. PH is classified according to Table 1.

1. Pulmonary arterial hypertension (PH)	2. Pulmonary hypertension with left heart disease
1.1. Idiopathic (IPH)	2.1. Left-sided atrial or ventricular heart disease
1.2. Familial (FPH)	2.2. Left-sided valvular heart disease
1.3. Associated with (APH):	3. Pulmonary hypertension associated with lung diseases and/or hypoxemia
1.3.1. Collagen vascular disease	3.1. Chronic obstructive pulmonary disease
1.3.2. Congenital systemic-to-pulmonary shunts	3.2. Interstitial lung disease
1.3.3. Portal hypertension	3.3. Sleep-disordered breathing
1.3.4. HIV infection	3.4. Alveolar hypoventilation disorders
1.3.5. Drugs and toxins	3.5. Chronic exposure to high altitude
1.3.6. Other (thyroid disorders, glycogen storage disease, Gaucher's disease, hereditary hemorrhagic telangiectasia, hemoglobinopathies, myeloproliferative disorders, splenectomy)	3.6. Developmental abnormalities
1.4. Associated with significant venous or capillary involvement	4. Pulmonary hypertension due to chronic thrombotic and/or embolic disease (CTEPH)
1.4.1. Pulmonary veno-occlusive disease (PVOD)	4.1. Thromboembolic obstruction of proximal pulmonary arteries
1.4.2. Pulmonary capillary hemangiomatosis (PCH)	4.2. Thromboembolic obstruction of distal pulmonary arteries
1.5. Persistent pulmonary hypertension of the newborn	4.3. Nonthrombotic pulmonary embolism (tumor, parasites, foreign material)
	5. Miscellaneous

Table 1: WHO Classifications of Pulmonary Hypertension

Pandey and colleagues reported a prospective intervention studies that evaluated the efficacy and safety of exercise training (ET) in patients with PH. Primary outcome of this meta-analysis was a change in six-minute walk distance (6MWD). The authors also assessed the effect of exercise on peak oxygen uptake (VO₂ peak), resting pulmonary arterial systolic pressure (PASP), peak exercise heart rate (HR peak), and quality of life. A total of 16 studies with 434 exercise-training participants were included. ET was associated with significant improvement in 6MWD [Weighted mean difference (WMD): 57.7 meters (95% CI: 42.5 to 72.8)], VO₂ peak [WMD = 1.7 ml/kg/min (95% CI: 1.3 to 2.0)], PASP [WMD = -3.6 mmHg (95% CI = -5.8 to -1.4)], HR peak [WMD = 10.4 beats per min (95% CI: 5.5 to 15.3)], and quality of life as measured on SF-36 questionnaire subscale scores. ET was well tolerated with a low dropout rate and no major adverse events [8].

ET in patients with PH is associated with a significant improvement in exercise capacity, pulmonary arterial pressure and quality of life.

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