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 PASMC ETnA and ETnB receptors, which ultimately leads to PASMC contraction, proliferation, and hypertrophy. ET-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 PASMC relaxation and inhibition of proliferation PASMC. (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 guanylate cyclase (GC) to convert guanosine triphosphate (GTP) to cGMP, the second messenger que constitutively maintains pulmonary arte...