Introduction

Despite multiple pharmacologic and non-pharmacologic strategies for the management of chronic heart failure (CHF), most patients report some limitation in their exercise capacity during the natural course of the syndrome. *(Gullestad* ***et al., 2001).***

In fact, exercise intolerance dominates the clinical presentation of moderate to severe CHF and is a major determinant of overall prognosis. ***(Mancini.2004).***

Multiple mechanisms seem to interfere with exercise performance in CHF, including central (cardiopulmonary) and peripheral(vascular)components. In particular, pulmonary hypertension is an important predictor of functional disability in CHF and may reflect both left ventricular dysfunction and congestion. ***(Moraes. 2000).***

Endothelial dysfunction is also a well-recognized feature of CHF that has been implicated in both its clinical presentation and prognosis.***(Katz et al., 2005)***

Underlying mechanisms integrating vascular endothelium changes in CHF include increased systemic vascular resistance, contributing to reduced cardiac performance, and altered pulmonary hemodynamics, contributing to pulmonary hypertension. Irrespective of which vascular tree is affected, endothelial dysfunction in CHF is associated with reduced vascular nitric oxide (NO) release.***(Moraes.2000).***

Inhibition of 5′-phosphodiesterase by sildenafil has proven to be beneficial in different scenarios where endothelial function and vascular tone can be positively influenced. **(*Halcoxet al., 2002).***

These beneficial effects are mediated in part by increases of NO availability to the vascular bed. ***(Burnett.2005).***

Moreover, a growing body of evidence shows that sildenafil consistently improves pulmonary hypertension caused by conditions other than CHF. ***(Galie et al., 2005).***

Recently, Lewis and coworkers showed that a 12-week protocol with sildenafil improved functional capacity and decreased pulmonary pressure in patients with CHF. ***(Lewis et al., 2007).***