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Despite multiple pharmacologic and nonpharmacologic strategies for the management of chronic heart failure (CHF), most patients will experience 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. **Halcox, et 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).**

Similarly, acute administration of sildenafil has been shown to improve parameters of endothelial, pulmonary, and cardiac function in patients with CHF**. Katz, (2000).**

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).**

Whether the acute beneficial effects of sildenafil on hemodynamic parameters, ventilatory efficiency, exercise performance, and endothelial function could be maintained for a longer period of time, thus mediating clinical and functional improvements, remained relatively unexplored.

Aim of the work

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The aim of the present study is to investigate whether a single oral dose 50 mg of sildenafil could improve exercise performance, ventilatory efficiency, oxygen uptake kinetics, pulmonary hypertension after two hours in outpatients with stable CHF.