Summary

chronic heart failure (CHF) is associated with secondary hypertension, impaired vascular reactivity and permeability, and reduced alveolar-capillary membrane conductance .

Various mechanisms are thought to be involved in the pathogenesis of "reactive" pulmonary hypertension in CHF, but an initial step possibly involves passive increase of pulmonary venous pressure secondary to left ventricular dysfunction.

Secondary structural changes occur in the vascular bed, including functional changes at the pulmonary endothelial cell level, leading to reduced release of NO and increased production of endothelin-1.

Sildenafil is a potent and selective inhibitor of cGMP specific phosphodiesterase type 5(PDE5) which is responsible for degradation of cGMP, Stabilization of CGMP results in increasing No at the tissue level leading to pulmonary vessel vasodilatation**.**

This study was conducted at Benha University Hospital during the period from october 2008 till May 2009.

It included 30 patients of chronic heart failure who had received standard medical therapy for CHF.

These patients were divided into two groups:-

The first group [I] was the control group which consisted of 15 patients and had received the standard therapy .

The second one [II] was the active group and consisted of 15 patients who received a single dose of sildenafil 50 mg in addition to the standard therapy.

The inclusion criteria for the patients to be involved in the study were to had chronic left ventricular systolic dysfunction (left ventricular ejection fraction 30 - 50%) and are receiving standard medical therapy for CHF, their ages more than 20 and to be stable for at least to months before involvement and to be able to exercise .

The exclusion criteria was hypotension , concomitant use of nitrates and intolerance for sildenafil and advanced function class.

All patients were subjected to careful history taking , complete general and local examination . they were examined by echocardiography for PASP and CPET before and two hour after the use of the drugs.

The ages of the patients were between 36 to 72 years, 11of them were above 60 years (36%) .

In the current study, the mean PASP before giving the sildenafil was 58.4 mmHg and after two hour of use sildenafil it became 51.7 mmHg.

In the current study, patients were assessed after one hour of sildenafil intake and significant improvement in VE peak,VO_2 Peak, VCo2 peak, VE/VCO_2 slope, **T** ½ VE, T-1/2 Vo_2 (min) and T-1/2 VCO_2 (min) from ٤٩,٩٣, 19.2 , ١٧,٥٣, 39.85 , ٢,١9 , 2.01 and ٢,٠ to ٥١,٥٣ , ٢٠,٨٦ ,١٦,٢ , ٣٦,٤٨, ١,٩٩ , ١,٨7 and ١,٨٢ respectively ($p < 0.05$).

So sildenafil is safe in stable CHF as it reduces pulmonary hypertension , improve exercise performance, ventilatory efficiency and oxygen uptake kinetics.