Summary

Pulmonary hypertension has been defined as an increase in mean pulmonary arterial pressure (PAP)> 25 mmHg at rest as assessed by right heart catheterization.

The goals of therapy for pulmonary arterial hypertension include promoting vaso relaxation, suppressing cellular proliferation, and inducing apoptosis within the pulmonary artery wall.

The rationale for the use of phosphodiesterase type 5 inhibitors in pulmonary arterial hypertension is augmentation of the cyclic guanosine monophosphate pathway. By inhibiting the hydrolysis of cyclic guanosine monophosphate,agents in this class increase its levels, with consequent vasodilatory, antiproliferative, and proapoptotic effects that may reverse pulmonaryartery remodeling**.**

Our study search for assessment of the short term effects of sildenafil citrate therapy for secondary pylmonary hypertension

This study carried out on fourty patients known to have symptomatic secondary pulmonary hypertension due to valvular heart disease,chronic thromboembolic, C O P D, I P F and non I D C M. twenty patients recevied sildenafil and other group recived placebo ,each group divided to fife equal subgroup acording to the etiology of secondary pulmonary hypertension ,follwed up for six weeks.

After six weeks all patients reassessed for hemodynamic changes, functional class progress, pulmonary artery systolic pressure improvement, ejection fraction changes.

The finding showed that sildenafil group had a significant decrease in PASP and significant improvement in NYHA class, and trend toward increase in ejection fraction.

Subgroup analysis showed all sidenafil group had good prognosis rather than placebo group, all sildenafil group significant decrease in PASP and significant improvement in NYHA class, and trend toward increase in ejection fraction.