DISCUSSION
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Hypertension is an important public health problem. Although easily detectable, asymptomatic and usually easily treatable, yet, it leads to lethal complication as in majority it is either untreated or is inadequately treated.

Diastolic and systolic dysfunctions have been observed early in the course hypertension and either or both may lead to heart failure. Left ventricular hypertrophy (LVH) is also common cardiac abnormality in hypertension and is found in about 30% of untreated hypertensive patients on echocardiography. The presence of LVH is consistently and strongly related to subsequent cardiovascular morbidity and mortality and its reversal leads to improved survival.

Richard B Deveruex proposed that different anti hypertensives despite reduction in BP of similar degree differ in their ability to regress the left ventricular hypertrophy. As the Angiotensin II and NE play an important role in the pathophysiology of left ventricular hypertrophy. ACE Inhibitor and Beta blockers have a define role in the regression of left ventricular hypertrophy.

Present study was conducted in Department of Cardiology, M.L.B. Medical College, Jhansi. A total of 52 patients were included in the study who had hypertension, and on echocardiography there is either, systolic, diastolic dysfunction or LVH and not taking any antihypertensive medication for last 3 months. They were randomly assorted into 2 groups. Group A consist of 22 people with age of 54.5
years and out of which 86% were male. Out of this group 23% had stage I hypertension and 77% had stage II hypertension according to JNC VII classification. This group received ACE inhibitor Rampril to achieve the target blood pressure $\leq 140/90\text{mm}$ after initial echocardiography study.

Group B consist of 20 persons with a mean age of 56.4 years and out of which 80% were male.

In group B, 35% patients had stage I hypertension and 65% had stage II hypertension according to JNC VII classification this group received Atenolol as selective B blocker to achieve the target blood pressure $\leq 140/90\text{mm}$, these patients were followed up at regular interval of 3 weeks to ensure the optimal blood pressure control. During follow up period a total 10 patients lost.

After 6 months of treatment repeat echocardiography was performed and the finding was analysed in context to initial echocardiography at beginning of therapy.

In both group there is improvement in the ventricular relaxation pattern (measured from of E/A ratio) in group A (receiving ACE inhibitor ) the mean E/A ratio was improved from 0.8% to 1.02. In group B receiving the B blockers the E/A ratio improve from 0.82 to 1.22 and this finding is statistically significant (p value $<0.032$). This finding is in agreement with previous finding of Gosse P. et al, Hevi Y et al, Salcedo A et al.

Ventricular relaxation is energy dependent is active process and involves the activation of SERCA-2 (Sarco endo plasmic reticulum Ca$^{2+}$ ATP are) through phosphorylation of phospholamban. Increased ventricular mass increase the oxygen demand and
ischemia at cellular level result in poor relaxation pattern. A reduction in ventricular mass improves the energy management in myocardium and improve the ventricular relaxation pattern.

Giovdano F.S. et al, and Brounwald E opined that mechanical strain and several agonists such as NE down regulate the expression of SERCA-2 particularly in hypertrophied myocardium. This explains the improvement in ventricular relaxation pattern with use of anti hypertensive drugs particularly B blockers which enhance the re expression of SERCA-2 and thus reuptake of free Ca²⁺ in endoplasmic reticulum.

Pretreatment the ejection fraction in group A (treated with ACE) was 54.4% and post treatment it was 54.15, there was decline of 0.46%. Though in other studies there was an improvement of ejection fraction particularly if there was suppression of ejection fraction at beginning of therapy like CONSENSUS and SOLVD M Eng J Med (1992).

As ventricular hypertrophy is a compensatory response to increase after load imposed by hypertension and a decrease in LVH lead to poor contraction in initial phase, though in long term there is improvement in EF. As this study was under taken for 6 month period and mean EF was calculated from both, patient with heart failure and those with normal EF. It imprudent to derive any conclusion.

In group B the pretreatment EF was 62% and post treatment it was 56.4% a negative change of 6.4%. This finding is consistent with normal physiological response to B blockers as sympathomimetic effect leads to positive ionotropic action and B blockers prevent this
action. But various studies using B blocker in heart failure whether associated with LVH or not shown an improvement in mortality despite an early reduction of EF (MCD).

In Group A pretreatment mean left ventricular mass index was 120gm/m2 and after 6 month of treatment the mean LVMI was 105.6 gm/m2. There was 12% decrease is the LVMI. Angiotensin II plays an important role in the development of LVH. Eichhoron EJ, et al and Pfeffers in his elegant study proved that prevention of angiotensin generation has in important role in the management of LVH and HF (Pfeffers MA et al). Opie L.H. et al also proposed that angiotensin II also promotes the proto oncogenes thus ventricular hypertrophy through protein Kinase C.

In group B pretreatment LVMI was 122.5gm/m and after treatment with Atenolol it was 117.5gm/m, showing a decrease of 4% in LVMI. This finding is significant (p < 0.043). These finding are in agreement with the studies of other authors like Agabiti Rosei E, et al and Salcedo A et al and R. B. Devereux et al. The probable cause for LVMI reduction with B blockers is blockade of neurohormonal changes responsible for myocardial hypertrophy and fibrosis.