Discussion
DISCUSSION

Coronary artery disease has been reported to be on the increase in India in recent times. Various studies indicate that incidence of CAD ranges from 6-20% of all heart disease patients (Sinha 1978).

The prevalence of coronary artery disease is more common in males as compared to females, the ratio being 4:1, but it is not so, at extreme of age when the CAD incidence becomes more or less same in both sexes. In India CAD appears a decade earlier in life as compared to developed countries. Coronary risk factors play a very important role in the development and severity of coronary artery disease.

The present study was conducted in Department of Medicine, M.L.B. Medical College, Jhansi.

Fifty patients were selected for the study. Number of male patients, were 41(82%) and 9(18%) were females.

Maximum number of patients (37) were of the age group 50-70 years. Mean age of patients was 57.2 years.

Most of the patients belonged to middle class, majority of them being from urban areas (70%) and 30% belonged to rural areas.
Most males were of service class, while most females were housewives. In the present study risk factors for CAD were present in good number of cases (70%), while only 30% patients were without any known coronary risk factors. Smoking was the commonest observed risk factor (54%). Tobacco chewers were 38%, hypertension (18%), diabetes mellitus (20%), obesity (24%) elecated cholesterol (12%) and family history of premature CAD in 2%. Glantz SA et al and various other Studies shows that smoking constituents the most important modifiable risk factor for CAD.

**LEFT VENTRICULAR SYSTOLIC FUNCTION**

On interruption of antegrade flow in an epicardial coronary artery the zone of myocardium supplied by that vessels immediately loses its ability to shorten and perform contractile work. Three abnormal contraction pattern developed in sequence Hypokinesia, Akinesia, Dyskinesia. If a sufficient quantity of myocardium undergoes ischemic injury, LV pump function becomes depressed, cardiac output, BP, ejection fraction are decreased and ESV & EDV are increased. The left ventricle undergoes a series of changes in shape, size and thickness in both infarcted an non-infarcted segments. This process is referred to as ‘ventricular remodeling’ and generally precedes the
development of clinically evident CHF. Early dilatation within the 1st to 2 to 3 days after myocardial infarction is attributed to infarct segment expansion, that begins within minutes of the onset of ischemia. This may be a consequence of reduced systolic ejection and increased LV end diastolic volume and pressure, causing increased wall stress. Increased wall stress causes myofibril stretching and thinning and compression of the intercellular space resulting in infarct expansion and it is irreversible at 3hrs\textsuperscript{57}. This early infarct expansion may increase the length of the infarct segment by as much as 65%\textsuperscript{58}. The result of early infarct expansion is an increased LV volume that allows cardiac output to be maintained at a lower LV filling pressure. Patients who do not develop early infarct expansion are unlikely to develop LV dilation subsequently and in some cases infarct segment may shrink as healing occur\textsuperscript{59}. Conversely, patients with early infarct expansion commonly develop global ventricular dilatation later over subsequent months.

Late left ventricular dilation occurs in both the non-infarcted and manufactured segments\textsuperscript{57}. In response to LV dilatation and increased wall stress, compensatory hypertrophy may follow in the non-infarcted segment. Thus
ventricular dilation after MI is due to early stretching of the infarct segment and later generalized ventricular enlargement. Although this adaptation allows initial maintenance of cardiac output, the increase in muscle mass and wall tension promotes further hypertrophy and eventually myocardial failure.

Clinical evidence of heart failure in coronary artery disease (angina/AMI) is associated with increased mortality even if the manifestation of failure resolve within the first 24 hours. LV systolic function after AMI has been extensively studied in relation to the development of heart failure and mortality.

In the present study ejection fraction, fractional shortening and regional wall motion were used to assess systolic function. But, the measurement that most cardiologist demand for global LV systolic function is ejection fraction (EF). The major impetus for presenting systolic function as EF is, the familiarity by clinician with this term and it is the most important indicator of survival after myocardial infarction. Furthermore, the more limited fractional shortening is not truly global if the ventricle is not contracting symmetrically.
In present study the isolated left ventricular systolic dysfunction (defined as an ejection fraction <50% and a normal filling pattern) was found in 9 patients (18%). The combined systolic and diastolic dysfunction was found in 17 patients (34%).

The study by SH Poulson et al (1997)\textsuperscript{51} shows that in patients of acute myocardial infarction the evidence of isolated systolic dysfunction was 21% and of combined systolic and diastolic dysfunction was 38%. While the study by M Schofield et al (1986)\textsuperscript{56} shows that in patients of coronary artery disease the evidence of isolated systolic dysfunction was 25%.

Out of 26 patients with LV systolic dysfunction, 15 (30%) had mild LV dysfunction (EF 49-40%), 9 (18%) moderate LV dysfunction (EF 39-30%) and 2 (4%) had severe LV dysfunction (EF <30%).

The LV fractional shortening was reduced to below normal (<25%) only in 8 patients (16%).

AV Goldersis M et al (1992)\textsuperscript{31} in their study showed that, in CAD, evidence of reduced fractional shortening in men and women was 18.4% and 10.9% respectively.
LEFT VENTRICULAR DIASTOLIC FUNCTION

The previous combined haemodynamic doppler color studies have demonstrated the utility of mitral flow velocities in the assessment of LV relaxation, compliance and filling pressure\(^1\). Among several mitral flow parameters, early diastolic (E), late Atrial (A) peak velocity, their ratio (E/A ratio), the isovolumetric relaxation time (IVRT) and mitral early deceleration time (DT) are most commonly used to evaluate the LV diastolic filling and they are shown best to correlate with LV pressure.

In present study transmitral flow velocities were used to assess LV diastolic function after onset of symptoms in patients with angina and myocardial infarction. Both myocardial relaxation and compliance are affected by ischemia, but the predominantly diastolic abnormality is an impairment in relaxation. Abnormal relaxation is also seen in aged and hypertrophied hearts, whereas decreased compliance is seen most often in patient with advanced dilated cardiomyopathy, and restrictive and cardiac amyloidosis.

Abnormal cardiac relaxation has been produced by balloon inflation during percutaneous transluminal coronary
angiography and is also reported to be present in subacute phase of a myocardial infarction\textsuperscript{61}. Decrease LV compliance (or its chamber stiffness) has recently been described in patients 1-2 days after an acute MI\textsuperscript{62}.

**Normal values of mitral flow velocity curves:**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>IVRT</td>
<td>70-100 msec</td>
</tr>
<tr>
<td>E wave velocity</td>
<td>76±13 cm/sec</td>
</tr>
<tr>
<td>A wave velocity</td>
<td>56±13 cm/sec</td>
</tr>
<tr>
<td>DT of E wave</td>
<td>&lt;220 (140-240) msec</td>
</tr>
<tr>
<td>E/A ratio</td>
<td>1.6 (&gt;1)</td>
</tr>
</tbody>
</table>

Types of diastolic dysfunction

1. **Abnormal relaxation pattern**
   
<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>IVRT</td>
<td>&gt;100 msec</td>
</tr>
<tr>
<td>D-time</td>
<td>&gt;220 msec</td>
</tr>
<tr>
<td>E/A ratio</td>
<td>&lt;1 cm/sec</td>
</tr>
</tbody>
</table>

2. **Pseudonormalized pattern**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>IVRT</td>
<td>&lt;100 msec</td>
</tr>
<tr>
<td>D-time</td>
<td>&lt;150 msec</td>
</tr>
<tr>
<td>E/A ratio</td>
<td>1-2 cm/sec</td>
</tr>
</tbody>
</table>
3. Restricted filling pattern

IVRT <60msec
D-time <150msec
E/A ration ≥2.0cm/sec

In present study the diastolic filling was found normal in 20 patients (40%). Isolated LV diastolic dysfunction (defined as impaired relaxation or pseudonormalized or restrictive filling pattern) and a normal systolic function was found in 13 patients (26%) and combined systolic and diastolic dysfunction was found in 17 patients (34%). In patients with diastolic dysfunction, abnormal relaxation was found in 19(38%), pseudonormalized in 5(10%) and restrictive filling pattern was found in 6(12%) cases.

SH poulson et al (1997)$^{55}$ found normal LV filling in 38% patients, isolated diastolic dysfunction in 24% and combined systolic and diastolic dysfunction in 38%. The evidence of impaired relaxation was 37% and pseudonormalized or restrictive filling pattern was found in 25% cases. Similarly Jacob E et al (2000)$^{53}$ found normal LV filling in 30% patients, impaired relaxation in 30%, pseudonormalized in 18% and restrictive filling pattern in 21% of patients.
Nijland et al (1997)$^{54}$ found restrictive filling pattern in 13% cases of CAD. Patients with impaired LV relaxation are often asymptomatic or display mild functional impairment at exercise, and often have normal or near normal filling pressure. In contrast, patients with restrictive filling characteristics due to decreased compliance have moderate to severe functional impairment and are associated with elevated filling pressures$^{63}$.

**Congestive Heart Failure**

The risk of developing physical signs and symptoms of left ventricular failure increases proportionally to increasing area of abnormal wall motion. Clinical heart failure accompanies area of abnormal contraction exceeding 25%, and cardiogenic shock, often fatal, accompanies loss of more than 40% of LV myocardium$^{66}$.

In present study, 35 patients (70%) had no clinical sign of heart failure (group A) while 15 patients (30%) were in Killip class II-IV (group B).

SH Poulson et al (1997)$^{51}$ in their study showed that in patients of AMI, the evidence of congestive heart failure was 34%.
The left ventricular systolic function was significantly decreased in patients with CHF, compared to those without CHF (EF=41±10 vs 58±10, EDV=79±24 vs 67±19, ESV=47±19 vs 34±18).

Silvio Romano MD et al (2000)\textsuperscript{29} studied 192 patients of AMI. RWM, EF, EDV, & ESV were evaluated. In 35 (18.5\%) patients death, recurrent angina, reinfarction or severe heart failure occurred during in hospital phase, whereas the remaining patients had good outcome. Patients with poor prognosis were older (68±6 vs 59±5 yrs), had a worse LV function (EF 50±10 vs 58±8, ESV 54±25 vs 38±12 ml/m\textsuperscript{2}).

However, 3 patients out of CCF group (20\%) had a normal ejection fraction but an abnormal diastolic filling pattern. A similar incidence of CHF and a normal ejection fraction have previously been reported among patients with clinical heart failure.

Grossman W et al (1993)\textsuperscript{44} recognized that in approximately 30\% of patients with CHF, the CHF is secondary to LV diastolic dysfunction.

SE Jensen et al (1997)\textsuperscript{51} assessed 65 consecutive patients with first MI within one hour of arrival in CCU and
concluded that, most patients with heart failure had early systolic dysfunction with decreased EF, but 23% had an abnormal filling pattern with preserved ejection fraction.

Moreover, no patients with normal filling pattern develop CHF, only patients with impaired relaxation, pseudonormal or restrictive filling pattern developed clinical heart failure. Elevated pressure is thought to increases LA pressure, producing shortness of breath and sign of pulmonary congestion.

It is remarkable that among patients who developed CHF, the LV ejection fraction was similarly impaired in the subgroup with impaired relaxation and in patients with restrictive or pseudonormal filling. However, the latter group had a significantly higher Killip classification. This findings is in accordance with studies in chronic heart failure where the restrictive filling pattern, with a shortened mitral E deceleration and isovolumetric relaxation time, is associated with more impaired functional status.64

Pseudonormal or restrictive filling may reflect primary changes in the infarcted and ischemic myocardium. In this study, these patients demonstrated early dilatation and
significantly larger end-systolic volumes, compared to patients with impaired ventricular relaxation.

This finding might impart, be compensatory to normalize the elevated filling pressure as indicated by the shortened mitral deceleration time. In present study, age and mitral E deceleration time <150msec independently identified patients at risk of developing congestive heart failure during the first week after first MI. mitral deceleration time has been shown to be independent of heart rate\textsuperscript{65}. This is useful in patients with pseudonormal transmirtal flow and is shown to posses prognostic information\textsuperscript{64}.

Bodi Peris V et al (1997)\textsuperscript{56} showed that the E-wave deceleration time DT predict LV diastolic pressure, in patients of AMI with systolic dysfunction. In this study 89 patients of AMI were divided into two groups according to ejection fraction. Group I (n=17) with EF<45% and group 2 (n=72) with EF>45% when patients from group 2 were analysed, no correlation was found between LVEDP and either E/A ratio or E-DT. However, in patients from group 1, LVEDP strongly correlated with both EDT (r=0.83, p=0.0001) and E/A ratio (r=0.70, p=0.003). Moreover, the sensitivity and specificity of an E-DT of less than 150ms in predicting a LVEDP>20mmHg was 100%. This conclude
that after AMI, E-DT provides a non-invasive and useful parameter for estimating LVEDP in patients with systolic dysfunction.

Thus early assessment of diastolic function may be helpful in selecting patients who might benefit from early intervention with ACE-inhibition following AMI. Further studies are needed to demonstrate the importance of diastolic function and the effect of early intervention with ACE-inhibitors on LV diastolic function in patients with AMI.

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