Myocardial infarction (MI) is one of the dangerous manifestations of coronary artery disease and is one of the most common diagnoses in hospitalized patients of Western countries. The determination of location, extent of infarcted myocardium is of vital importance for the prognosis as well as management of the patients. Therefore the present study is conducted to correlate the minimum level of serum magnesium with the enzymatic estimate of infarct size i.e. by measuring peak Serum Glutamate Oxaloacetate Transaminase (SGOT) and also with Electrocardiographic estimate of infarct size. Minimum serum magnesium was measured on day 1 of admission to the ICU of 53 patients of acute myocardial infarction. Also 12 lead ECG was recorded from these patients and serum SGOT was measured on for first three days after admission. Statistical analysis for correlation between peak SGOT activity and minimum serum magnesium was done using Pearson correlation coefficient test. We found statistically significant decrease in serum magnesium and increase in SGOT activity in patients with widespread myocardial infarction.

INTRODUCTION
Cardiovascular diseases are a major cause of mortality, morbidity and disability in developed and developing countries. In physiological study of major inorganic constituents of heart muscle cells, role of magnesium was neglected till now. There were several reasons for this neglect. Chemical analysis for magnesium was found to be very difficult for many years, the ionized magnesium (Mg +2) in cardiac cells could not be directly determined and the rate at which magnesium is transferred into and out of the cell is so much slower than rate of transfer of potassium, sodium, calcium and chloride that electrophysiological measurement of transport in the in-vitro preparation of heart muscle becomes difficult.

Therefore the present study was undertaken to correlate the serum magnesium levels with peak SGOT (enzymatic estimate of infarct size) and ECG (electrocardiographic estimate of infarct size) in patients of acute myocardial infarction.

AIMS AND OBJECTIVES:
1. To study the correlation between minimum level serum magnesium with Peak SGOT (An enzymatic index of infarct size)
2. To study the correlation between serum magnesium with Electrocardiogram (Electrocardiograph estimate of infarct size)

MATERIALS AND METHODS
The present study was conducted in Intensive Cardiac Care Unit (ICCU) of Sir JJ Hospital and Grant Medical College, Mumbai 8. Three ml of venous blood collected from antecubital vein with aseptic precautions. Serum magnesium and SGOT was measured in Department of Biochemistry in JJ Hospital, Mumbai 8.

Also ECG is recorded using 12 lead ECG machine with standard procedure.

Mean, standard deviation were calculated for serum magnesium, SGOT in Microsoft excel sheet.

Pearson’s correlation coefficient test was used to study the correlation between peak SGOT and minimum serum magnesium levels.

The approval from Institutional Ethical Committee (IEC) was obtained before commencement of study. Also the written informed consent of the subjects were taken.

INCLUSION CRITERIA
Out of 58 cases of acute transmural myocardial infarction, 5 cases died during ICCU stay before completion of study protocol. So, remaining 53 acute myocardial infarction study cases which were finally included for statistical analysis out of which 46 were males and 7 were females with mean age was 49.32 ± 7.41 years. Diagnosis of myocardial infarction was done using clinical history and standard guidelines laid down by WHO criteria.

Out of these 53 patients of acute myocardial infarction were divided into 3 groups.

Group A (Anterior): This group included patients with anterior, anteroseptal, inferolateral, inferoposterior wall myocardial infarction cases.

Group B (Inferior): This group included patients with inferior, inferolateral, inferoposterior wall infarction.

Group C (Combined): This group included patients of fresh infarction in both anterior and inferior wall.

Table 1: cases in study group

<table>
<thead>
<tr>
<th>SR NO</th>
<th>GROUP</th>
<th>NO OF CASES</th>
<th>PERCENTAGE</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Group A (Anterior)</td>
<td>34</td>
<td>64 %</td>
</tr>
<tr>
<td>2</td>
<td>Group B (Inferior)</td>
<td>14</td>
<td>26 %</td>
</tr>
<tr>
<td>3</td>
<td>Group C (Combined)</td>
<td>5</td>
<td>10 %</td>
</tr>
<tr>
<td>TOTAL</td>
<td></td>
<td>53</td>
<td>100 %</td>
</tr>
</tbody>
</table>

EXCLUSION CRITERIA
1. Patients with previous history of acute myocardial infarction.
2. Any other cardiac changes on electrocardiogram.
3. Non cardiac causes of increase in levels of SGOT. E.g cerebrovascular accidents, intramuscular injections, hepatitis, pancreatitis, uraemia, trauma etc.
4. Any illness causing reduction in serum magnesium levels like hepatic failure, renal failure, neoplasia.

Graph no 1 shows the percentage distribution of study groups.
with combined anterior and inferior wall infarction in which there was a significant increase in peak SGOT in patients of study group. We found highly significant increase in peak SGOT in patients of study group, SGOT which is used as enzymatic index of severity of MI. It shows greater is the size of infarct , greater is the fall in serum magnesium levels. Thus this can be used as a electrocardiographic estimate of myocardial infarction.

The size of infarct correlates well with the levels of serum magnesium. The size of infarct was more in group C as compared with group A and group B individually and this was associated with maximum fall in serum magnesium level in group C as compared to group A and group B. From this data it was concluded that serum magnesium was very much reduced when the size of infarct was more. Similar findings were observed by Kedarnath and Prasad et al.  

The peak SGOT was determined in 53 cases of study group, SGOT estimation was done on the day of admission and thereafter daily in the morning for 3 consecutive days to determine peak SGOT in each study groups. We found highly significant increase in peak SGOT in patients with combined anterior and inferior wall infarction in which there was maximum fall in serum magnesium levels.

MECHANISM OF HYPOMAGNESEMIA:
The exact mechanism for hypomagnesemia is not well known but several hypothesis has been put forth.

Prasad et al (3) suggested that serum magnesium may be utilized to check the increased adhesiveness of platelets following MI in order to prevent thrombosis.

Nath et al (4) suggested that magnesium is mopped up at the site of infarction to check the hycroagulability of blood.

Land Mark K, Uardal P (7) stated that fall in serum magnesium can be due to increased beta 2 adrenergic receptors.

Kedarnath and Prasad et al (5) have studied correlation between serum magnesium and SGOT levels at various duration after acute MI. They concluded that significant fall in level of serum magnesium as early as 2 hours following acute MI without significant change in SGOT in those hours can be taken as reliable diagnostic index in case of suspected myocardial infarction.

MECHANISM OF ELEVATED SGOT IN PATIENTS OF ACUTE MI:
Any cause of tissue breakdown like trauma, ischemia, infarction may result in elevated serum enzymes presumably by the release of enzymes from the damaged tissue. As these enzymes are present in sufficiently high content in myocardial tissues, sodeath of a relatively small amount of tissue results in substantial increase in measured enzyme activity in serum.

CONCLUSIONS:
There is significant fall in serum magnesium levels following acute myocardial infarction. This is directly related to the size of infarct. Greater is the size of infarct, greater is the fall in serum magnesium levels. Thus this can be used as a electrocardiographic estimate of myocardial infarction.

Also, we found inverse relation between minimum serum magnesium levels with peak SGOT in patients with acute myocardial infarction. Thus this parameter can be used for enzymic estimate of myocardial infarction.

However, currently many other reliable and rapid diagnostic criteria are available for early diagnosis and treatment of the patients like CPK-MB, troponin –t test etc.

REFERENCES.