A STUDY OF EARLY PHASES OF ACUTE MYOCARDIAL INFARCTION IN ICCU

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CERTIFICATE

This is to certify that the dissertation titled **"A STUDY OF EARLY PHASES OF ACUTE MYOCARDIAL INFARCTION IN ICCU"** is a bonafide work done by **Dr. S. Suresh Kumar.** It is a regular, systematic study done under my guidance and supervision during the period April 2004 - March 2005 and submitted for the ensuing **M.D. BRANCH - I GENERAL MEDICINE EXAMINATIONS**, **September 2006** of the Tamil Nadu Dr. M.G.R Medical University, Chennai.

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DECLARATION

I solemnly declare that the dissertation titled **"A STUDY OF EARLY PHASES OF ACUTE MYOCARDIAL INFARCTION IN ICCU"** was done by me at Coimbatore Medical College and Hospital during the period April 2004 – March 2005 under the guidance and supervision of **PROF. Dr. G. YASODHARA, M.D.**

The dissertation is submitted to the Tamil Nadu Dr. M.G.R Medical University towards the partial fulfillment of the requirement for the award of **M.D. Degree Branch – I in General Medicine**.

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INTRODUCTION

Cardiovascular diseases are one of the leading causes of death. Among the Acquired cardiovascular diseases, Acute Myocardial Infarction is a well recognized and most important cause. Rajeev Gupta et al did an extensive community study, the incidence he found was 1.5% for men for a population aged 40-69 years, 5% for women[75]. It is a world wide problem the relative incidence varies in different countries and was found to be high in developed countries. The incidence varies directly with economic well being. Significant incidence of infarction is found in population taking habitually high calorie diet, saturated fat, cholesterol and refined carbohydrates and low in population where the diet is low in these substances. These statistics also show low incidence of Acute Myocardial Infarction in women especially during child bearing age.

There has been recently change in the incidence of acute myocardial infarction. This probably reflects the changing life style and eating habits.

In patients dying of acute myocardial infarction the initial period seems to be very critical. In a study from Maron[49] reported that of 76%

of all sudden deaths were due to coronary artery disease and those patients dying of coronary artery disease 64% died suddenly; 67% of death occurred within first 2 hours. 67% of all deaths occurred outside the hospital. Green[50] has shown that 40% of the death from acute myocardial infarction is due to arrhythmias, two third due to ventricular fibrillation and one third due to bradycardia, heart block and asystole. The awareness that the earlier period after acute myocardial infarction 24 hours, is critical lead to the development of coronary care units (CCU). The early CCU was started almost simultaneously at Toronto Kansas city, Philadelphia and Sydney[13]. These centers prove to be quite effective in reducing the mortality rates due to primary ventricular fibrillation.

However even then it is seen that the patient is admitted in CCU after a delay of few hours which happens to be the most critical time. This led to the idea of mobile coronary care unit (MCCU). This was first started by Pantridge and his colleagues in Belfast[15]**.** It was then followed in other places e.g. Columbia, Ohio. In Belfast during the first five years of MCCU there was no death due to arrhythmias in MCCU[16]**.** MCCU has decreased the time delay before which the patient gets medical care. Before the establishment of MCCU the delay was around

8 hours which was reduced to around one hour and forty minutes in Belfast.

Even after the beginning of MCCU the incidence of deaths were high in the initial hours. The problem was identified as lack of knowledge about Acute Myocardial Infarction in the patient and the public. National registry of Myocardial Infarction has shown that the mean time interval between attack and arrival to the hospital was 5 hours and 10 minutes[47]. Time lag before asking for medical care was 1 hour and 30 minutes. The most important single factor for high mortality due to Acute Myocardial Infarction is the delay by the patient in seeking medical help. This delay is not significantly reduced by MCCU. A realization that the mortality cannot be significantly reduced without educating the public about immediate measures, has led to rethinking of CCU and MCCU by certain people.

Some claim that ICCU has no advantage in management over ward. Some have pointed out difference in mortality from Myocardial Infarction before and after establishment of CCU is not significant. Mortality rates for Acute Myocardial Infarction vary from 10%-40%.

Heart association (USA) has set a number of standards prepared for CCU[46]. It included among them

1. Priority attention
2. Stand by order for staff to initiate ECG monitoring and therapy for arrhythmias.
3. 24 hours coverage by person trained in recognition and treatment of arrhythmias.
4. Immediate availability of resuscitative equipments and drugs.

This is a voluntary set of codes and many hospitals in USA follow them.

The concept of Intermediate Coronary Care Unit[64] was started as a result of considerable mortality, seen in the later periods after Acute Myocardial Infarction.

It is argued that the role of intermediate coronary care should be to prepare the patient psychologically and physically to meet his daily life and tensions[63].

AIM OF THE STUDY

The aim of this work is to study series of proven cases of Acute Myocardial Infarction in ICCU and conduct careful study of clinical course during stay in hospital and try to assess prognosis as a short term measure.

REVIEW OF LITERATURE

A lot of work has been done in this field in many countries throughout the World.

ESTABLISHMENT AND GROWTH OF ICCU

Acute Myocardial Infarction is recognized to be one of the leading causes of death all over the world. And the incidence seems to be higher in the developed rather than developing countries. The field of Acute Coronary Care has undergone tremendous changes and the development of newer modalities in the management has been there. It was mainly due to the fact and knowledge that most of the deaths after Acute Myocardial Infarction occur in the initial few hours and commonly due to arrhythmias. Moreover it occurs above the age of 40 years. It affects a person at peak of his career and has important social and economical repercussions on the family.

Castilli showed that 67% of deaths due to Acute Myocardial Infarction occur outside the hospital[14]. Meltzer and his associates[29] analyzed the causes of death following acute myocardial infarction, their data states that 40% of deaths are due to arrhythmias, two third of these deaths are due to ventricular fibrillation and one third due to bradycardia, heart block and asystole.

These observations' demonstrating that most of the deaths following Acute Myocardial Infarction occur in the initial hours and these deaths are commonly as a result of arrhythmias has lead to the concept of Intensive Coronary Care Units.

The earlier functioning Intensive Coronary Care Units could bring down the mortality rate only by 20 %. During this period when ICCUs were being established, certain other workers studying the impact of ICCU on the community and the amount came to the conclusion that ICCU does not significantly reduce the mortality rate. This happened when the ICCU was started.

Adverse reports like this did not retard the progress of ICCU. On the contrary reasons for low reduction in mortality were analyzed. It was shown that the reason was the delay in admitting the patient to ICCU. This lead Pantridge and his associates[15] in Belfast to start Mobile Coronary Care Unit. The development of MCCU was followed by a considerable reduction in mortality rate[16].

A Mobile Coronary Care Unit should have a fast communication system. So that relative, the doctor or the patient himself should be in a position to call for help immediately. Trained medical and paramedical

personnel[48] who know the procedure of defibrillation[11], pacing, intubations, starting IV medication, external cardiac massage etc. The ambulance should carry the following:

1. Battery operated portable defibrillator.
2. A Battery operated pace maker.
3. Essential drugs.
4. Endotracheal tubes.
5. Battery operated suction machine.
6. Oxygen, Ambu bags.
7. Some carry facilities for transmitting the patients ECG to the base hospital and receiving instructions from them.

In 1969 the medium delay in Belfast was reduced to one hour and forty minutes. This was considered as a significant one when compared to a delay of 8 hours before MCCU time. Even after the establishment of MCCU the incidence of mortality during the early hours following acute myocardial infarction was found to be considerable. This was attributed to a) the inability of the patients to recognize the early symptoms and inform the hospital b) time delay in contacting the doctor and in transporting to the hospital. In spite of these factors experience in Seattle have shown the long

term survivors for ventricular fibrillation treated by MCCU have increased from 7.7 % to 20 % [17, 18].

It is now realized that the best way to reduce the mortality further would be by mass scale programme to educate the public about the symptoms of myocardial infarction, first aid and need for early admission.

This led to the concept of Prehospital care of Acute Myocardial Infarction. In a study by Paul.N.Yu[10], in Circulation he concepted an approach regarding the pre-hospital care, which included:

1. Public education with emphasis on early warning symptoms and signs and need and importance of seeking early medical care with special attention directed towards the high risk coronary patients.
2. Professional education.
3. Mechanisms to direct and or bring patients with suspected or proven myocardial infarction promptly to system of medical care with special emphasis on the utilization of a telephone information centre and provision of rapid transportation.
4. Establishment of special life support stations for screening, monitoring and stabilization of cardiac arrhythmias. It can be fixed with emergency department with a precoronary care or mobile units.

5. Prevention of sudden cardiac death which consists of study of mechanism and the clinical environment of sudden cardiac death, development of techniques for early diagnosis and management.

According to W.Moore et al[21], it was found that regarding the pre-hospital coronary care and coronary fatality, the delay between the onset of symptoms and access to specialist coronary care was the most likely critical difference.

Now the concept of chest pain observation units (CPU) has come into vogue. It was shown to be very useful. According to Farkouh et al it was shown that the patients who present to the emergency room with chest pain and who have an intermediate risk of cardiovascular events can be safely cared for with the use of chest pain observation units[20]. The same concept is supported by Gareth Quinn in his study regarding chest pain evaluation units[22].

In the era of evidence based medicine there is also a concept of Evidence Based Coronary Care by Eugene Braunwald and Elliot Antman where they have shown that since Herrick's classic description of Acute Myocardial Infarction was published in 1912, the management of this condition has gone through four phases. The first, "Clinical

Observation Phase" of coronary care, lasted about half a century and consisted of the simple assessments like Vital signs were recorded on the first day after infarction; clinical examinations were done and electrocardiograms were obtained daily for the first few days; and chest roentgenograms were obtained once or twice a week. The infarcted heart was considered to be a wounded organ, the repair of which required the equivalent of the immobilization of a fractured bone. . Patients were usually hospitalized for 5 to 6 weeks. The major debates during this phase revolved around whether ambulation could be started early (1 week) or late (2 to 3 weeks) after admission. There was also considerable controversy about the indications for anticoagulant agents, which were administered primarily to prevent pulmonary thromboembolism, a major complication of bed rest. The in-hospital mortality rate approached 30%; after discharge, patients usually led restricted lives, and 15% died during the remainder of the first year.

Early 1960s in the so-called "Coronary Care Unit Phase"[1]. There was closed-chest cardiac resuscitation, continuous electrocardiographic monitoring and teams of trained physicians and nurses, antiarrhythmic agents (such as lidocaine) and direct-current defibrillators and pacemakers to treat life-threatening arrhythmias. Halving of the in-

hospital mortality rate for acute myocardial infarction but remained susceptible to the late consequences of large infarctions: heart failure and malignant ventricular arrhythmias. This resulted in a continued high incidence of late deaths and serious disability.

The "High-Technology Phase," began in the 1970s new diagnostic and therapeutic methods. Swan-Ganz catheter form bedside assessment of hemodynamics[2]. 24-hour ambulatory (Holter) electrocardiography, followed by electro physiologic testing. Exercise electrocardiography, radionuclide ventriculography, and myocardial perfusion scintigraphy coronary arteriography and myocardial revascularization. Concept of protecting the ischemic myocardium was developed[3]; use of ß-blockers and then to early myocardial reperfusion-thrombolytic therapy and primary coronary angioplasty. Coronary care was based primarily on the rational application of pathophysiologic principles. The clinical outcome of patients with acute myocardial infarction improved further, and the in-hospital mortality rate was reduced to less than 10%. The patients' post discharge prognosis also improved. Because early reperfusion often limited the infarction size, patients were left with more viable myocardium, which reduced the risk for subsequent heart failure and fatal arrhythmias[4].

The "Evidence-Based Coronary Care Phase." The analysis of coronary care by Peterson and colleagues. Performance of a specialized test in a patient with acute myocardial infarction is only justified if the test can provide incremental information that will change the clinician's practice so as to favorably affect clinical outcome. The reasons why many of the tests in the high-technology phase of coronary care had limited clinical value are complex. Chief among these reasons is a progressively lower pretest likelihood of adverse outcome (largely due to improvements in the care of patients with acute myocardial infarction), which produced low positive predictive values for many tests.

The basic strategy described by Peterson and colleagues is 1) to assess risk continuously during the patients' course, especially at the initial presentation, at 24 hours, during the late hospital phase, and before discharge and 2) to drive management at each point according to the results of these assessments.

Many challenges remain in the care of patients with Acute Myocardial Infarction. Even though in-hospital mortality rates have been reduced by approximately two thirds since the end of the clinical observation phase 35 years ago, about 500 000 patients in the United

States still die of acute myocardial infarction each year[5]. What causes these fatal outcomes? Most of the deaths are sudden and occur before the patient reaches the hospital. Most of the remainder results from massive infarctions that cause cardiogenic shock or internal or external cardiac rupture. Although reperfusion therapy (thrombolysis or primary Percutaneous Transluminal coronary angioplasty) has enormously improved the care of patients with acute myocardial infarction and ST-segment elevations who present to the hospital soon after the onset of symptoms, patients with large infarctions and ST-segment depression present a continuing challenge. In the future, an increasing focus on patient behavior will be required. For example, we will have to learn how to identify persons who delay seeking medical assistance after the development of symptoms and how to modify the behavior of these persons so that these delays are shortened[6].

The new generation of antiplatelet agents will be of increasing interest. The potent direct glycoprotein IIb/IIIa inhibitors, when used in conjunction with thrombolytic agents, have the potential to establish patency of occluded vessels more rapidly, thereby reducing myocardial damage, enhancing myocardial function, and improving both short- and long-term outcomes. Oral forms of these inhibitors have been

developed, and large-scale testing of these agents in patients who have had acute myocardial infarction is about to begin.

It is likely; therefore, that Evidence-Based Coronary Care will be more than just another phase in the evolution of coronary care.

DURATION OF HOSPITAL STAY:

There is no fixed time limit for patient with myocardial infarction to be discharged from the hospital. The recent trend is distinctly towards early ambulation especially when Acute Myocardial Infarction is not complicated.

A randomized multi centre trial of early ambulation of acute myocardial infarction was done by Goldberg. et al[7]. In this trial patients were divided into two groups .one group was mobilized on day five and another on day ten and they were followed up. There was no significant difference in the mortality in the first year after myocardial infarction. There was unexplained linear increase in mortality of late mobilization group in the second and third year. Studies have set trend for early mobilization.

In a study conducted by National Heart Attack Alert Program Coordinating Committee, and treated acute myocardial infarction along

lines designed to hospitalize the patients for a minimal time and early return to work. Rapid rehabilitation enabled patients to be discharged within a fortnight and 87% within a month. 3% returned to work within four weeks of the onset of myocardial infarction. 56 % within 6 weeks and 77% within 8 weeks. There was no ill effects in hospital or during a 6 month follow up study[6].

In a study by Newby et al[9]. he has shown that, the values and costs of tests and treatments are analyzed rigorously and guidelines are developed in an effort to achieve the most efficient care of patients with acute myocardial infarction. He further showed that the saving of huge amounts of money by discharging patients with uncomplicated acute myocardial infarction at 72 hours.

In a community wide population study regarding the declining the length of hospital stay for acute myocardial infraction and post discharge outcomes by Frederick.A.Spencer[67] et al in a sample of 4551 patients ,they found marked decrease in the length of the stay in the hospital for patients with acute myocardial infarction during the past decade. They found no negative association between declining the length of stay and short term mortality after hospital discharge for acute myocardial infarction.

In another study by Heller and Dobson[66] they have shown that all patients aged less than 70 years with a diagnosis of AMI admitted to the seven public hospitals in the Lower Hunter Region of New South Wales are monitored as part of the WHO MONICA Study. Between August 1984 and December 1985 of 438 hospitalized patients with a 'definite' AMI according to MONICA criteria and a clinical discharge diagnosis of AMI, 386 (88%) patients were discharged alive from hospital. Four patients had lengths of stay between 46 and 77 days and have been omitted from further analysis. The mean length of hospital stay was 13.6 days; 74% of all patients stayed in hospital for more than ten days. The mean length of stay in the Intensive Coronary Care Unit (ICCU) was 4.5 days with 60% staying longer than three days. Mean hospital stay varied from 10.5 to 17.4 days among the seven hospitals, although most of this variation was accounted for by three hospitals with few patients. Restricting analysis to the four hospitals with 90% of all the patients, multiple regression analysis showed that the CK enzyme levels, the evolution of Q waves on ECG, the presence of an anterior AMI and the use of nitrates and digoxin during hospitalization were all associated with increased length of stay in hospital.

COMMON PROBLEMS IN ICCU:

Arrhythmias:

As said before arrhythmias are one of the commonest causes of death after acute myocardial infarction. The major advantage of treatment in CCU is the early detection and correction of arrhythmias.

Genesis:

In acute myocardial infarction there are metabolic changes including impaired glucose utilization, increase free fatty acids, secretion of catecholamine and increase in the blood lactate. Apart from them, myocardial ischemia produces abrupt increase in extracellular potassium causing localized hyperkalemia. There is also increase in cyclic AMP all leading to alteration in electrophysiological properties and production of various cardiac arrhythmias[19].

TACHYARRHYTHMIAS:

Supraventricular TachyArrhythmias:

This includes all arrhythmias whose site of impulse formation or reentry site is above the bifurcation of common bundle of His.

The incidence varies in different centers which depends on admission policy and time delay. The incidence of arrhythmias has demonstrated by Metlzer.L.E.et al[12] are as follows

a. Atrial arrhythmias including atrial premature beats are seen in 50% and in some it is a sign of atrial infarction
b. Supraventricular tachycardia is 4-20%
c. Atrial flutter 5%
d. Atrial fibrillation 7-16%

Of these the minor arrhythmias are Atrial premature beats and sinus tachycardia and they usually do not require treatment. Other arrhythmias are considered major and require treatment though not as urgently as ventricular tachycardia.

In trying to explain the mechanism behind these arrhythmias various theories had been put forward. Circus movement theory is accepted for Atrial flutter only. Allessie first put forward the theory of multiple Atrial foci[29] Moe G.K[68] suggested multiple reentry circuits. Allessie also suggested multiple reentry circuits.

The clinical features depend on the ventricular response to the arrhythmias, presence or absence of the cardiac failure and cardiogenic shock associated with acute myocardial infarction.

Zoni[69] suggested that many Supraventricular Tachyarrhythmia during the first 48 hours are transient and cause little hemodynamic

embarrassment but they may be recurrent. Due to recurrent episodes it is common for them to develop failure which requires diuretics.

Ventricular Tachyarrhythymias:

They have mores serious implications. The two most common are extra systolic ventricular tachycardia and ventricular fibrillation. Continuous monitoring, Holter monitoring, 24 hours magnetic tape recorders and online computers are the ways to detect.

Ventricular tachycardia defined as three or more beats occurring successively. The incidence of Atrial arrhythmias increases with age but not with ventricular tachycardia.

Ventricular tachycardia represents the physiological fragmentation pf the ventricles into a complex out of phase state; refractoriness, excitation and responsiveness. The development of this state is favored by two events a). Uneven recovery of the biventricular chamber. b). Stimulation of the biventricular chamber by a very premature ventricular ectopics or a rapid ventricular tachycardia, before activation or recovery is complete. Ventricular fibrillation occurring in Acute Myocardial Infarction is classified as being primary or secondary, depending on the presence or absence of complicating

factors like congestive cardiac failure, cardiogenic shock as suggested by Bigger[24]. Sometimes it has been reported to be induced by pacemaker.

The heart rate immediately preceding ventricular fibrillation and their mortality was studied by Rajagopalan et al[28]. 52 patients in their series were studied 2 (3.8%) had heart rate below 60 per minute with one death (2.9 %). 30 patients (58%) had heart rate between 91- 94 with 5 deaths (18%). And 20 (38%) above 100 per min with 2 deaths (4.11 %). Pantridge et al[27] documented autonomic disturbances in the early phase stage one of infarction. Those with sinus tachycardia with or without hypertension were considered to show sympathetic over activity, while those with heart rate below 60 per min had increased parasympathetic activity .Ventricular fibrillation may occur following spontaneous increase in heart rate. The ideal heart rate after acute myocardial infarction is around 90- 100 per min. Primary ventricular fibrillation could be a fatal complication in conduction disorder either due to change in ventricular function resulting ventricular ectopics. Ruberman showed that the slow rate of complete heart block has high risk of ventricular tachycardia of ventricular fibrillation[23].

In an analysis of ventricular fibrillation in 52 patients[23], mortality was 60% in age group more than 70 years, mainly due to atherosclerosis.

There is slight difference in the incidence of ventricular fibrillation with infarction in different areas. The incidence was slightly higher in anterior wall infarction (36 out of 1246) than in inferior wall infarction (7 out of 667) and extensive anterior wall infarction (9 out of 315). This shows there is no direct correlation between the size of infarct and ventricular fibrillation.

The incidence of ventricular fibrillation among the Acute Myocardial Infarction that dies within one hour is 90% as shown by Campbell R.W[70]. Muscle necrosis is not necessarily a pre requisite for ventricular fibrillation, even ischemia or reperfusion itself causes electrical instability and lead to ventricular fibrillation.

The tempo of clinical response to ventricular arrhythmias depends on variety of consideration but the functional consequence depends on two factors. They are adequate perfusion of vital organs and the risks and natural history.

In general rapid ventricular rate may end in left ventricular failure. Various arrhythmias may act as a warning for the impending arrhythmias. There is no good correlation between the occurrence of two types of arrhythmias and subsequent ventricular tachycardia. The arrhythmias are:

1. Combination of frequent multifocal and paired ectopics.
2. Combination of R on T with consecutive ectopics.

There is about 35 % chance of ventricular tachycardia developing within half an hour of occurrence of these arrhythmias[29]. The prognostic significance of ventricular tachycardia is related to:

1. The ectopic ventricular rate.
2. Sinus rate immediately preceding ventricular tachycardia.
3. Associated conduction disorders.
4. The state of underlying myocardium.

BRADY ARRHYTHMIAS:

Brady arrhythmias contribute significantly to mortality following acute myocardial infarction. They are potentially lethal and treatable complication. Adgey and his associates[30] feel that the incidence of vagally induced Brady arrhythmias is more. Brady arrhythmia occurs in 40 % within four hours of 294 patients of acute myocardial infarction. They were more common in posterior wall infarction.

Sinus bradycardia is probably is only second to ventricular extra systole in occurrence[12]. Pantridge et al[27] reported that 28 developed sinus bradycardia within one hour. Some found it was more with Inferior wall infarction[30].

The incidence of sinus arrhythmias is reported to be 8.1 % as shown by Dener which more in inferior wall myocardial infarction. In a series reported by Dener [31] this arrhythmias was observed only one case out of 100 consecutive patients. This arrhythmia was always observed in Inferior wall infarction.

AV CONDUCTION DISTURBANCES:

First degree block:

In a series as shown by Dener first degree block was very frequent[31]. Its quite transient Adgey et al[30] feel that progression of block is common with inferior wall infarction.

Second degree and third degree block:

The incidence of these varies between 2.5 % to 8 % as shown by Behar occurs within first few hours almost invariably within the first week[33]. Its incidence between 7 to 27 % with inferior wall infarction1 to 5 % with anterior wall infarction[33].

Second degree wenckebach also more common with inferior wall infarction. A complete heart block developing after first week is quite unusual according to Goldberg[32].

SHOCK IN ACUTE MYOCARDIAL INFARCTION:

This defined as hypotension i.e. with a reduction of 30mmHg from baseline systolic level or systolic blood pressure lower than 90mmHg. Clinically manifesting as hypoperfusion. The manifestations commonly are oliguria, cold clammy mottled skin and altered

sensorium. It is important both hypotension and clinical manifestations be present to warrant the diagnosis of cardiogenic shock.

In patients dying of cardiogenic shock studies revealed that 40 % or greater of the left ventricular muscle wall are infarcted these findings suggest that there is critical quantity of myocardial necrosis, beyond which cardiogenic shock will develop. The progress in these patients is predictably is dismal, the mortality ranging from 80 -90%. Though the clinical recognition is straight forward there is however a group of patients with acute myocardial infarction in advanced stages of pump failure that may have normal blood pressure and no clinically apparent evidence of hypoperfusion in an analysis done by Cercek[34].

There is phase of preshock syndrome which identifies people with high risk of developing shock. Ideally hemodynamic monitoring includes the use of right heart balloon catheter, pulmonary arterial pressure and cardiac output. They measure right heart function, left heart function, CVP monitoring reflects the right side pressure changes accurately.

Arrhythmias precipitate as well as exacerbate cardiogenic shock. Where a compensatory was expected a heart rate, below 70per min may

be considered as an indication for intervention treatment with isoprenaline is associated with increased with infract size and should normally be avoided unless facilities for pacing is not available. Arrhythmias have three important effects on infarcted heart particularly in cardiogenic shock by a study by Schrieber[35].

- Loss of Atrial booster pump and diastolic filling period resulting in decreased stroke volume.
- Shortening of diastolic also leads to decrease in coronary blood flow.
- Tachycardia causes an increase in myocardial oxygen consumption.

MITRAL REGURGITATION:

This may be due to papillary muscle dysfunction or frank rupture of papillary muscle. Mitral regurgitation is severe and most cases died within 48 hours according to Wei[36].

Rupture of interventricular septum may be suggested by development of systolic murmur and a thrill. Right heart balloon catheterization reveals an oxygen saturation step up in the right ventricle indicating ventricle septal defect.

Rupture of free wall of the left ventricle initially produces a picture of cardiac tamponade. One of sudden catastrophic event is electromechanical dissociation.

RIGHT VENTRICULAR INFARCTION:

Right ventricular infarction is recognized with increased frequency in acute myocardial infarction. It occurs in 5 % of all acute myocardial infarction and is invariably associated with inferior wall and posterior wall infarction. A definite diagnosis can be made by 99mTc pyrophosphate uptake study. According to Setaro[37] it may contribute to cardiogenic shock with hemodynamic picture mimicking cardiac tamponade. The patient will have hypotension, raised jugular venous pressure but no sings of pulmonary congestion.

SIGNIFICANCE OF FASCICULAR BLOCK:

This incidence is higher in Anteroseptal myocardial infarction probably reflecting the involvement of ventricular septum secondary to obstructive diseases of the anterior descending branch of left coronary artery according to Dener[31].

Left anterior Hemi Block:

It is the most common interventricular conduction defect and the incidence ranging from 4 to 15.2 %. It does not progress to complete heart block and remains as an isolated finding. It has no influence on the incidence of arrhythmias, cardiac failure or survival[39].

Left posterior Hemi Block:

Isolated event is very rare with an incidence of 0.22%-1.5% of all acute myocardial infarction[40]. The reasons for rarity are blood supply to this is from both right and left coronary artery, a relatively favorable anatomical location. The fact that the posterior division is the broadest of the fascicles. It does not proceed to complete heart block but usually denotes widespread damage. The main cause of death is being pump failure according to Domenighetti[41].

Right bundle branch block

The incidence varies from 4-6% in acute myocardial infarction according to Girks. Isolated RBBB does not mask the acute myocardial infarction. It doers not progress to higher grades of AV block but is said to carry a 44% of the hospital mortality according to Girks. They generally are younger and have anterior myocardial infarction according to Depasquale It is usually associated with left anterior hemi

block or posterior hemi block. And this combination carries a bad prognosis[71].

Bifascicular block

A block in the left main bundle or its anterior or posterior fascicle together results in left bundle branch block. Mortality is around 40%. Incidence of LBBB is more in anterior myocardial infarction and it ranges from 3-5% according to Hindmann[39]. Majority of patients with rate dependent LBBB have bradycardia dependant LBBB and more with inferior wall infarction. Reasons for this association is the fact that right coronary artery occlusion which produces inferior wall myocardial infarction also causes sinus slowing allowing bradycardia dependent LBBB.

The combination of RBBB with LAHB carries a grave prognosis according to Depasquale[38]. Mortality in RBBB with LAHB complicating acute myocardial infarction is between 50-60%[38], while those RBBB with LAHB is 1%. The incidence of complete heart block is 50% and the mortality ranges from 75-100% in a study by Goldberg[32].

Trifascicular block

Two major sites for AV block is demonstrated by His Bundle studies in patients with acute myocardial infarction, proximal to N (nodal) and distal to H, i.e. trifascicular block .The prognosis, mortality in patients with this block in myocardial infarction is more than 90% according to Karton[43].

MATERIALS AND METHODS

Patients admitted consecutively for a period of one year from April 2004 to March 2005 in our ICCU were chosen for the study. For the diagnosis of infarction the WHO criteria was followed.

This criteria states that infarction is said to have occurred if atleast two of three following criterias are present.

1. Typical history and clinical picture
2. Unequivocal ECG changes.
3. Raised CPK-MB.

The serum studies in our series were done in all the patients. But main emphasis being on typical history and clinical and ECG changes. Patients not satisfying these criteria were not chosen. Patients' history in relation to time of onset of symptoms, associated features and time delay before admission were taken with care. Immediately on admission patient had a 12 lead ECG and usually monitored. Patients' clinical status was assessed using pulse, blood pressure, presence of gallop, murmur, pericardial rub, muffling of heart sounds and features of congestive cardiac failure. The patients were grouped according to cardiac function on admission based on Killips classification.

KILLIPS CLASSIFICATION:

Class I : No signs of pulmonary or venous congestion.

Class II : Moderate heart failure as evidenced by crepitations at lung bases. S 3 Gallop, tachypnea, (or) signs of right sided heart failure, including hepatic and Venous congestion.

Class III : Severe heart failure, pulmonary edema.

Class IV : Shock with systolic BP less than 90 mmHg and evidence of peripheral vasoconstriction, mental confusion, decreased urinary output.

The patients were followed up during there hospital stay. They were watched for occurrence of arrhythmias, thromboembolism, reinfarction, cardiac failure, cardiogenic shock, conduction disturbances and other complications that might arise. The causes for deaths were analyzed. Histories in relation to risk factors smoking, hypertension, previous anginal episodes, diabetes and history of ischemic heart disease were gone into detail. The ECGs were repeated on the 3^{rd} day and frequently on the 10^{th} and 21^{st} day of hospital stay. ECG s of these were analyzed to asses the severity of the infarction. The extent of ST segment deviation was used to decide the severity of infarction and the TP segment was used as isoelectric.

The ST deviation was measured in mm to the nearest millimeter.

In anterior wall MI the ST segment deviation of > 5 mm was considered to be major and rest minor.

In Inferior wall MI the ST deviation of > 2 mm was considered to be major and the rest minor.

An attempt was made to correlate the clinical condition of the patient as defined by Killips classification, degree of ST segment deviation, and the incidence and type of arrhythmias and mortality.

ANALYSIS OF DATA

The highest incidence of infarction in the study was recorded in the age group between 51 – 60 yrs, in which the number of infarction was 32 out of the 70 recorded. The patients were divided into 6 groups with an age difference of 10 between 20years to 70years.

The incidence in the age group is tabulated:

Age group	No of cases
21-30 yrs	5
31-40yrs	7
41 – 50yrs	14
51- 60 yrs	32
61 – 70 yrs	8
> 70 yrs	4

The lowest incidence occurred in the age group of more than 70 years in which the number of infarction was 4 out of 70 cases. The males outnumbered females. The number in the youngest age group was 5 out of 70 cases. The Number of infarcts among males was 61 and females 9 in the ratio of 6.9:1 .The age of incidence of female patients among the 9, 5 occurred in the age group of 51 – 60 yrs. And 4 in the age group of 61 – 70 yrs. All the transmural infarcts were classified according to the area of infarction. 10 Patients showed a combination of

inferior and anterior wall infarction or extensive Anterior wall infarction, 10 patients with Anteroseptal infarction, 17 patients with inferior wall Infarction, 21 anterior wall infarction, 6 antero lateral infarction and 6 high lateral infarction.

The patients presenting with the following ECG changes :

Types of Infarction	**Number of Patients**
Anterior wall infarction	21
Inferior wall infarction	17
Extensive anterior wall infarction	10
Anteroseptal infarction	10
Antero lateral infarction	6
High lateral infarction	6

In analyzing the risk factors 12 among them had previous infarcts 7 had previous angina, and 8 were under the treatment for diabetes. The hypertensive were 19. The smokers were 37 in this group who were all male members. Patients were considered as smokers if they smoke 15 beedies or cigarettes per day for atleast 5 yrs previous to infarct. Time delay before admission influenced the prognosis and mortality rate to great extent. With this point in view of the time delay our patients were analyzed.

On an average the patients were brought to this hospital 3 to 5 hours after the onset of symptoms. Number of patients who were brought before one hour was only 8 while after one hour it was only 11, those after 24 hrs was 7 only. Interestingly among the 10 patients who died ,4 were brought earlier than the average time i.e. Within 3 hrs . 2 were brought between 5 – 8 hrs and 1 within 1 hr.

The patients were grouped into 4 divisions according to their in Killips classification.

In group I: There were 50 patients. These are the patients who showed no signs of pulmonary congestion clinically and were not tachypnoeic.

In group II: There were 12 patients who showed evidence of moderate heart failure i.e. Rales in lung bases, s 3 gallop, tachypnea,and signs of right heart failure including raised JVP and hepatic congestion.

In group III: Patients numbering 6 consists of those with severe heart failure and pulmonary edema.

In group IV: 2 in number had frank cardiogenic shock with patients systolic BP less than 90 mm Hg, peripheral vasoconstriction, mental confusion and decreased urinary output.

The severity of infarct was assessed by measuring the degree of ST segment deviation. In Anterior wall infarct the deviation of greater than 5 mm and in Inferior wall infarction more than 2mm divisions were taken as major infarcts.

There were 12 patients with major anterior wall infarction and 7 in inferior wall infarction. One in major inferior wall infarct died. Arrhythmias were recorded in 19 patients in our study. Though many had occasional ventricular ectopics they were not considered here. The majority of the patients who showed arrhythmias had anterior wall infarction (14). 4 had inferior wall myocardial infarction and one had lateral wall myocardial infarction.

The majority of the patients developed arrhythmias on the second day after the onset of symptoms. They were 10 out of 19. The longest time interval was 25 days after the onset of symptoms where the pt developed VF. Frequent VPCs were seen in patients who were admitted 15 mins after the onset of symptoms.

The occurrence pf arrythmias in our series were :

Sinus Bradycardia	6
Sinus Tachycardia	4
Atrial Fibrillation	3
Atrial Flutter	1
Junctional Ectopics	1
Junctional Rhythm	3
AV	2
Multiple VPC s	11
I st degree block	6
RBBB	4
LBBB	3
LAHB	7
VT	5
VF	3
Electro Mechanical Dissociation	1

The complications other than arrhythmias noted were :

Acute LVF	:	12
Hemi paresis	:	2
DKA	:	2
Hypotension leading to shock	:	6
Hemoptysis complicating heparin therapy	:	2
Hematemesis complicating heparin therapy	:	1

Deaths:

Out of the 70 patients 12 died.

6 due to acute LVF.

4 due to ventricular fibrillation.

1 died due to reinfarction on the ward on the fourth day .

1 died due to persistent VT.

DISCUSSION

AGE AND SEX FACTORS:

The lesions of Coronary Atherosclerosis are more frequent and more extensive in older individuals. They are more common in males than in females and the females it is less in women of child bearing age group. In certain families there seems to be a very high incidence of infarction. This has been attributed by Wilson[44] to an unknown risk factor running in families not necessarily to a Familial hypercholesterolemia. Though there is progression of Atherosclerosis with aging and an increase incidence of myocardial infarction, this association need not necessarily mean cause. The highest in our study was in the age group 51 – 60 yrs. This data agrees with the general principle that incidence of infarction increases with age. The lesser number of infarction above 60yrs may be attributed to the population itself being low in India. Aila M.Rissanen and associates are of the opinion that familial aggregates for MI occurring in younger age groups is due to familial occurrence of hypertension and hyperlipidemia[45].

Recent study have indeed confirmed that individuals with parents affected by the disease prior to the age of 50 yrs have a greater risk of developing Coronary atherosclerosis developing at a younger age

group. In cases the relative risk may be as high as 5:1[44]. It has been noted that 1 out of 150 Children at birth have hyperlipoproteinemia of type II predisposing them to early coronary atherosclerotic diseases according to Grudy[72].

It is widely accepted that men are prone to coronary atherosclerosis than women of child bearing age .After menopause there is rapid narrowing of this diff. of the many reasons presented for this diff. in Susceptibility to atherosclerosis, a possible protective effect of estrogen, diff in lipid levels and hematocrit, reduced risk of cigarette smoking and a more sheltered life have been proposed. A modest effect of estrogen is observed on alpha and beta lipoprotein[52]. The incidence of females to that of males in our study was 1: 6.9. The total number of females with infract in our study was 9, the incidence of MI in the population aged 40 – 69 to be 1.5 for men and 0.5 for women for the first attack[48].

TIME FACTOR EFFECT ON MORTALITY:

The most important single factor for high mortality rate in AMI is the delay by the patient in seeking medical help according to Simon[47]. This is not significantly reduced by mobile coronary care units. In patients dying of Acute Myocardial Infarction the initial period

seems to very critical as shown by Bousma and Deckers[8]. 76% of all sudden deaths were due to coronary artery disease according to Maron[49] and of those patients dying of coronary artery disease 65% died suddenly and 67% deaths occurred within 2 hrs and 76% of all deaths outside the hospital. Meltzer et al have shown that 40 % of all deaths from AMI are due to arrhythmias and of these ventricular fibrillation accounted for 2/3 and the rest were due to bradycardia, heart block and asystole[12]. The varying mortality rates in different ICCU units are to a large extent influenced by the time delay before the pt is admitted. In those places where the patients are picked up by mobile coronary care units were naturally higher. Those units were the time delay was longer, the pt had already withered the storm and naturally the prognosis is better, brought the mortality in ICCU. The mortality in varying ICU s varies from 10 – 40%. The average time interval between the onset of symptoms and admission calculated in a community study was 5 hrs and 23 minutes according to Goldberg[7]. This study was done in a population in UK. The time delay was attributed to the ignorance of the pt in realizing the seriousness of the symptoms, difficulty in contacting doctor, and bottle necks in the transport. The time elapse before sending for medical help was calculated to be 1hr and 30mins.

In our study the medium delay was 6.3 hrs and our conditions were the hospital serves surrounding rural areas and where transport is an overwhelming problem, this time delay can be taken as being reasonable. Inspite of the median time delay being 6.3hrs majority of the patients were admitted between 1-5 hours. This compares favorably with the study done by Goldberg[7]. There is an attempt at further reducing the time delay by the introduction of mobile coronary care units. Now the consensus is that mortality due to AMI cannot be further unless the public are trained in first aid like external cardiac massage and to recognize initial symptoms.

In our study 4 patients came after 24 hrs, among them one came on the third day. The reason given by them was that they lived in far of places and the trouble of coming all the way was not worth it as they thought the initial symptoms showed a minor disorder.

HYPERTENSION:

Elevated blood pressure is a risk factor of prime importance and of established association with coronary atherosclerosis[45]. The prevalence of coronary artery disease is significantly higher in hypertensives that in normotensive according to Kennel[51]. However

epidemiologically an experimental study suggests that hypertension accelerates atherosclerosis only if hyperlipidemia is present and that hyperlipidemia is present effect on hypertension is related to the degree of abnormality[52]. The beneficial effects of reducing blood pressure in coronary atherosclerotic heart disease remains to be established although veterans' administrative study suggest a beneficial effect in regard to cerebral atherosclerosis. In our study there were 19 patients with hypertension with a diastolic blood pressure of more than 100 mmHg. And retrospective and prospective study has shown a definite association between hypertension and coronary atherosclerotic heart disease according to Van den Hoogen[53].

ABNORMAL GLUCOSE TOLERANCE:

Patients with diabetes mellitus have been found in retrospective studies to have greater a prevalence of the disease, more extensive lesions and earlier onset of the disease according to Gerstein[54]. It is difficult however to isolate diabetes mellitus as a single factor since it is well recognized that obesity, hypertension and hyperlipidemia are also frequent with impaired glucose tolerance. There is some evidence that high levels of circulating insulin may have a role in development of coronary atherosclerosis and that arterial wall is an insulin sensitive

tissue according to Despres[55]. Exposure of arterial wall tissue to insulin results in proliferation of smooth muscle cells, inhibition of glycolysis and synthesis of cholesterol, phospholipids and triglyceride. In our study the number of diabetic patients were 12 out of 70.all were type 2 diabetes mellitus and two of them DKA. There was no definite history of hypoglycemia s a cause of precipitation of AMI.

SMOKING:

Statistical evidence associating cigarette smoking with an increase incidence of coronary atherosclerosis in impressive according to Castelli[14]. In general the risk of developing coronary atherosclerotic heart disease is 2-5 times higher than that of the non smokers. The risk appears to be directly proportional to number of cigarette smoked per day. Ample statistical evidence supports mean increase of about 70% in death rates and 3-5 fold increase in risk of ischemic heart disease, in men who smoke 1 packet per day when compared to non smokers. According to Wilhelmson[57], the risk of coronary artery disease due to smoking increases with age. In women the relationship with smoking was less than in men. However there is impressive accentuation of ischemic heart disease mortality in women taking oral contraceptives, in addition to smoking. In more atherosclerosis prone populations such

as patients maintained on long term haemodialysis, cigarette smoking interacts with other risk factors, resulting in enhancement of mortality due to ischemia heart disease. Such interactions are also likely for diabetic and hypertensive patients. This association of cigarette smoking increased IHD remains unexplained. Pipe and cigar smokers have a lesser risk presumably because lesser smoke is inhaled. Smokers dying of causes other than IHD have been found at autopsy to have more coronary atherosclerosis than non smokers. The major influence by smoking is on the incidence of sudden death. Those who stop smoking show a prompt decline in the risk and may reach the risk level of non smokers as early as one yr. there were 37 smokers in our series. All of them have been smoking atleast 15 cigarettes or beedies per day for the past 5 yrs. There were no cigar or pipe smokers and patients who had angina previously prior to AMI were all smokers, they were 7 of them. The risk due to smoking decreases with age.

AN ANALYSIS OF PROGNOSTIC FACTORS:

In our study, patients were grouped according to Killips classification as said earlier in an attempt to predict the mortality rate. Among the 12 patients who died 6 of them under class III, 2 under class IV, 2 under class II and the rest 2 were in class I.

Clinical status of patients on admission – Killips classification:

Class I	50 patients
Class II	12 patients
Class III	6 patients
Class IV	2 patients

Death in relation to clinical status on admission:

Class I	2 patients
Class II	2 patients
Class III	6 patients
Class IV	2 patients

This classification shows that of those who died in our study were due to acute left ventricular failure following acute myocardial infarction. Those patients who were assessed to be in class I on admission and later succumbed, the mortality was due to be an reinfarction in the ward after transfer from ICCU in one patient and another patient had persistent Ventricular Tachycardia who later died in a private nursing home after transfer from ICCU, cause of death probably being Ventricular Tachycardia generating into Ventricular Fibrillation.

A comparison between two tabular columns clearly shows a high mortality rate of patients in class III and class IV. The percentage of death in our study in relation to their place in Killips classification is as follows:

Class I	2.25%
Class II	6.67%
Class III	100%
Class IV	100%

The mortality rates established by other workers for patients classified according to Killips are:

Class I	0.5%
Class II	10-20%
Class III	35-45%
Class IV	85-95%

The factors that should be taken into consideration in studying this distribution of mortality in Killips classification is the fact that the patients were analyzed at the time of admission and were assigned to a class . Some of them later deteriorated due to causes like reinfarction. This apparently raised the mortality for class I. In our study the mortality rates for classes I, II, III were uniformly higher than the predicted values.

ST SEGMENT DEVIATION – INDEX OF SEVERITY OF AMI :

Engelen and his co workers have studied and evaluated the value of ECG in estimating infarct size[58]. They have come to the conclusion that all waves in ECG are useful in assessing the infarct size. He further studied and correlated the ST segment deviation in transmural and sub endocardial infarcts in pathological studies. The study involved 80 patients who died of acute myocardial infarction who had showed QRS, ST segment changes. Changes in the QRS complex occurred in 9 out of 15 cases of sub endocardial infarction which later confirmed pathological studies. 5 of these had definite ST segment depression occurred most frequently with sub endocardial infarction but 43% of Tran mural infarcts also show this finding. In 17 % of Trans mural infarct, this was the only ECG finding mimicking sub endocardial infarct. They have concluded that subendocardial infarct cannot always be differentiated from transmural on the basis of QRS changes alone.

The presence of Q waves in leads in which they are not normally found need not necessarily denote infarction. It was shown by mickelgreenspan[59] and his co workers that exercise induced Q waves appeared in normal persons. Some persons have tried to correlate the presence of pericarditis as shown by the presence of pericardial rub

after AMI, to subsequent mortality, 313 patients was studied by Kent and workers according to Baumann[60]. They found pericardial rub in 67(21%). Of these having Anterolateral MI was associated with pericarditis, is 22 out of 26. (85%). Lateral wall infarction 4 out of 4. Inferolateral MI 2 out of 5.anterior 16 out of 36 (15%). 10 persons with pericarditis died. 7 had shock, 2 were due to rupture of ventricle and both of them had inferior wall MI. They have concluded that pericarditis is limited in the early course of myocardial infarction and most prevalent in lateral wall infarction. Persistence or recurrence of pericarditis in 18 patients was associated with extensive infarct or reinfarct.

Bent.Lyuger Nelson[111] have attempted to correlate the degree of ST segment deviation and the prognosis. They classified myocardial infarction into major and minor depending on the degree of ST segment deviation.

In anterior wall infarction more than five divisions was considered as major.

In inferior wall infarction more than 2 divisions was considered as major.

The TP segment was used as an isoelectric line and deviations are measured to the nearest millimeter. They have come to the conclusion that patients with major deviation had greater incidence of cardiac arrest, cardiac failure, death, atrial fibrillation, shock, heart blocks, ventricular tachycardia and ventricular premature complexes[23].

In our study out of the 70 patients of acute myocardial infarction number of patients who could be classified as major infarcts according to Bent.Lyuger criteria were:

Anterior wall infarction : 12

Inferior wall infarction : 7

All the others i.e. 51 out of 70 had minor infarctions. Number of deaths in patients having major infarcts was 33% while the mortality rate with minor infarction was 20.4%.

The difference in our study is not significant as far as the mortality rate is concerned. This is probably due to the limited number of patients who died in my study.

All the arrhythmias recorded in my study occurred mostly in minor infarcts. Though the death rate of the patients with major infarct

(33%) was higher compared to minor infarct with low number of patients being in major infarct this difference is not significant.

TIME DELAY:

The person who died in our study was admitted much later than the patients who survived. This clearly shoes the importance of time of time factor. This was clearly showed by Lakartidningen[65] where he states that **"Time is a heart muscle"** Quicker management and reperfusion save more patients with ST-segment elevation infarction.

Findings from the Worcester Heart Attack Study[7] show that patients who delayed for more than 6 hours were 6.5 times less likely to receive thrombolytic agents compared to patients who arrived within 1 hour of the onset of AMI.

The expression “aborted infarction” was first used to describe the patients treated very early in the Myocardial Infarction and Triage Intervention (MITI) trial[73]. It was found that 40% of all patients treated within 3 hours of onset of symptoms had no evidence of infarction as measured by thallium scan at 30 days follow-up. Minimal infarct size of less than 10% was noted in additional 35% patients.

A meta-analysis of 22 trials, including more than 50000 patients, showed maximal effectiveness of thrombolytic therapy within the first hour of symptom onset (the golden hour), whereas the benefit was reduced by nearly 50% in the subsequent hour (the Boersma's curve).

An estimated 65, 37, 26 and 29 lives are saved per 1000 patients when treated with thrombolytic therapy within 0-1, 1-2, 2-3 and 3-6 hours respectively[4]. If the patients of AMI can be identified and treated very early after the onset of symptoms, the infarction process can essentially be aborted.

PROGNOSTIC INDICES :

Immediate risk stratification of patients with myocardial infarction in the emergency department (ED) at the time of initial presentation is important for their optimal emergency treatment. Current risk scores for predicting mortality following acute myocardial infarction (AMI) are potentially flawed, having been derived from clinical trials with highly selective patient enrolment and requiring data not readily available in the ED. These scores may not accurately represent the spectrum of patients in clinical practice and may lead to inappropriate decision making.

In an extensive study at the mayo clinic that involved 1212 patients who were admitted between 1988 and 2000 were studied. A risk score model was developed for predicting. The risk score included age, sex, systolic blood pressure, admission serum creatinine, extent of ST segment depression, QRS duration, Killips class, and infarct location. The predictive ability of the model in the validation set was strong[8].

Various people have correlated presence of different arrhythmias with prognosis with considerable success as pointed out before.

B.L. Chapman and C.H.Gray have formed their prognostic indices with the following factors[74]. They have used a scoring system.

1. Cardiogenic Shock

Systolic blood pressure less than 80mmHg with signs of reduced perfusion to brain.

2. Oliguria

Less than 500ml per 24 hours following myocardial infarction.

This scoring system of Chapman ideally suits our needs as it does not involve elaborate studies and is based mainly on clinical judgment.

The prognostic indices published by Norris considered the following factors viz. age, position of infarct, admission systolic BP, heart size, lung fields and previous ischemia. In this study Chapman Gray index predicted correctly the outcome for 88% of patients with a cut off point of 20 and for 91% with a cut off point of 80.

ARRHYTHMIAS AND THEIR IMPORTANCE :

The largest number of deaths in infarction is due to arrhythmias occurring early according to Green[50]. Among the arrhythmias ventricular fibrillation is the commonest cause of death. He further showed that Ventricular fibrillation occurring early during myocardial infarction without any other complications is termed primary while that occurring later and with complications is termed secondary. The former responds well to defibrillation and recurrence rate is low while the later is refractory to defibrillation and mortality is high.

Ventricular premature complexes stand out as the commonest. Numerous studies have been conducted to asses their importance[23]. If it occurs in a malignant form, treatment should be initiated immediately.

SUPRAVENTRICULAR TACHYARRHYTHMIAS

Regarding supraventricular tachyarrhythmia, it is mainly due to enhancement of sympathetic tone in response to baroreceptor reflexes and anxiety state, alteration in the electrophysiology of pacemaker, conduction system, hypoxia caused by impaired circulation, shock state and depressed respiration, release of potassium from injured cells, changes in the catecholamine levels, free fatty acid levels and electrolyte disturbances.

The changes in hemodynamics vary from momentary changes to depression of circulation. Rapid coordinated contraction of atria and ventricle usually produce decrease in cardiac output. Patients usually tolerate supraventricular arrhythmias well. But in some cases it decreases the cardiac reserve and rapidly produces cardiac failure or shock.

The incidence of supraventricular arrhythmias demonstrated in a study of patients with infarction is as follows according to Meltzer.

a. Atrial arrhythmias including atrial premature beats as seen in 50% and in some it is a sign of atrial infarct.

b. Supraventricular arrhythmias in 4 to 20%

c. atrial flutter in 2 to 5 %

d. atrial fibrillation in 7 to 16 %.

In our study of 70 patients who were not continuously monitored the incidence was

a. Sinus Tachycardia - 4 patients.

b. Supraventricular Tachycardia - 3 patients.

c. Atrial Flutter - 1 patient.

d. Atrial Fibrillation - 1 patient.

e. Junctional Rhythm - 2 patients.

Of these arrhythmias sinus tachycardia was not considered as a major one. It does not need therapy unless it is causing circulatory embarrassment.

The patient who developed atrial fibrillation was already a known Ischemic heart disease patient and his presentation was atrial fibrillation. The other developed after being transferred to ward from CCU on day two.

VENTRICULAR ARRHYTHMIAS

Ventricular tachycardia is defined as 3 or more beats occurring in succession. The focus being in the ventricle.

Regarding the pathogenesis there is correlation between the plasma catecholamine and the incidence of ventricular arrhythmias has

been reported. Rajagopalan et al[28] have demonstrated an increased incidence of ventricular fibrillation in patients having low as well as high heart rate. In his opinion the optimum heart rate, following myocardial infarction would be 90 – 100.

The various ventricular arrhythmias encountered in our study were:

1. Mulitple ventricular premature beats - 11
2. Ventricular tachycardia - 5
3. Ventricular fibrillation - 3
4. Electro mechanical dissociation - 1

This incidence to the 70 patients that were studied. It is said that occasional ventricular premature beats occur in almost all myocardial infarction according to Green and Ruberman. The low incidence in our study only reflects the absence of continuous monitoring in our group of patients.

Of the 11 patients who had ventricular premature beats,

1. 6 did not develop any complications.
2. 3 developed ventricular tachycardia, 2 of them were reverted and 1 died due to asystole.
3. 1 had persistent ventricular tachycardia and later transferred to private hospital where he succumbed.

4. 1 had a heart rate of more than 240 on the monitor but his pulse rate was 84, who complained of severe chest pain and the diagnosis of electro mechanical dissociation was made and he later died ,this patient was the youngest in our study being only 24 years.

BRADY ARRHYTHMIAS

They from potentially lethal but treatable complication. They may be due to intensive vagal stimulation, ischemia of SA node according to Adgey[30], injury or necrosis of AV node, accumulation of metabolites or factors such as pain and fear producing reflex sinus slowing.

AV Blocks

Left anterior hemi block

This is the commonest isolated unifascicular block following acute myocardial infarction, with an incidence ranging from 4 – 15.2 % according to Coljj[40]. It is relatively benign. In our study 7 patients developed this block. They had anterior wall infarction in 2 patients, 2 with inferior wall infarction and 3 with extensive anterior wall infarction.

Left posterior hemi block

This a uncommon condition. Its presence usually indicates extensive myocardial damage. It occurs in about 0.22%[41] study as an isolated event. The mortality being around 50% and the main cause being pump failure according to Domenighetti.

Right bundle branch block

This occurs as an isolated event in about 5 – 60% of the cases according to Hindman[39] and Col J.J[40] .These patients usually have anterior wall infarction and usually carry a hospital mortality of 44%.
In our study 4 patients were seen with RBBB and who had anterior wall infarction. One developed failure symptoms and others did not have any complications during their hospital stay.

Left bundle branch block

The development of LBBB masks the infarction ion many cases. The incidence of isolated LBBB varies from 3 – 5 %. This occurs with identical frequencies with anterior wall infarction and lateral wall infarction according to Domenighetti[41]. Mortality rate is roughly equal to that of RBBB. No of cases of isolated LBBB seen in our group was one.

Combinations of fascicular blocks

In our group of patients three had combination of RBBB with LAHB. This carries a grave prognosis with higher incidence of sudden atria ventricular block and death occurring due to myocardial decompensation according to Domenighetti[41]. This the most common bifascicular block. The incidence ranging from 4 to 9 %(61). This is also the most common precursor of trifascicular block, with an incidence of complete heart block varying from 2 to 47 %.

One patient in our group developed cardiac failure and he was under Killips class III. He later died due to cardiac decompensation.

The other combination of RBBB with left posterior hemi block has a high mortality due to extensive myocardial damage which is needed to produce this block. Mortality ranges from 75 to 100% and incidence of complete heart block exceeds 50% according to Kostuk. This combination was not seen is our series.

There were six patients who had first degree AV block, where four were with inferior wall infarction and two with anterior wall infarction. They all reverted to normal. This block does not influence the final outcome.

Transient complete heart block with patient having Stokes Adams attack was seen in a patient. This patient later died due to cardiac systole. Patient had extensive anterior wall infarction. This patient was paced, the pacer was later removed.

In patients who develop complete heart block after anterior wall infarction, the prognosis seems to be influenced by the extent of infarct and so pacing might not improve long term prognosis. While in those patients developing complete heart block after inferior wall infarction improves well with pacing and their prognosis is better.

CONCLUSION

The early phase of myocardial infarction is the most critical period. The formation of Intensive Coronary Care Unit (ICCU) has drastically changed the management. The time delay in getting the appropriate management alters the disease status. The concept of Door to Needle time has led to the management of myocardial infarction to new pathways.

The Inhospital status of the patient and the development of complications during the initial phase like Tachy arrhythmias, Left Ventricular failure and conduction disturbances determines the prognosis in a short term basis as well as long time.

In this study 70 consecutive patients admitted and proved to be having major and minor infarcts in ICCU were chosen. The patients were grouped into four classes according to Killips classification.

The severity of myocardial infarction as detected by the degree of ST segment deviation was used for grouping them into major and minor infarcts. Bent Luyger Neelsons criteria were used for grouping the patients. The patients' course during the hospital was followed up.

An attempt was made to correlate the severity of myocardial infarction as per the ECG criteria with Killips classification in trying to assess the prognosis. The incidence of arrhythmias, cardiac failure and deaths was noted in each group. In our series 19 patients qualified to be classified as having major infarction.

There was an overall tallying as shown by a higher percentage of patients with severe infarction by ECG criteria, occupying class III or IV (Killips classification). They also had increased mortality mainly due to left ventricular failure present in 12 patients (60 percent), ventricular tachycardia 4 patients (20 percent), ventricular fibrillation 3 patients (16 percent) and one had electro mechanical dissociation (4 percent).

This relationship was convincing as the number of patients having major infarction was nineteen.

Increased incidence of arrhythmias in patients with major infarction was demonstrated in our series and the main form was Ventricular Premature Complexes they also had ventricular tachycardia, ventricular fibrillation and also Brady arrhythmias. The conduction disturbances occurred frequently the main form being LAHB.

SUMMARY

Among the acquired cardiovascular diseases acute myocardial infarction is the most critical one. The ICCU is the place where it is managed.

When the concept of ICCU was started around 1960's it has grown in leaps and bounds.

After that the concept of mobile Coronary Care Unit (MCCU), Chest Pain Evaluation Units (CPU) and Intermediate Coronary Care Unit was started. Prehospital thrombolysis is a new addition to the concept of critical care of myocardial infarction.

The time delay or the door to needle time determines the prognosis of the patients. The clinical course of the patient in ICCU like development of complications like arrhythmias, cardiac failure and sudden death is very important.

In the present study conducted at ICCU of CMC hospital from April 2004 – March 2005 it was found that the management of acute myocardial infarction in ICCU determines the mortality and prognosis

and reduction in mortality was significant. Deaths were mainly due to arrhythmias and left ventricular failure.

Hence the management of the patients found to have acute myocardial infarction in the ICCU during the early phase of myocardial infarction makes a great positive impact in the survival and the prognosis both in short term and long term way.

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Master Chart

S. No	Name	Age (Yrs)	IP No.	Sex	Killips class	Smoking	DM	HT	CAHD / Angina	Infarct size	CPK – MB	LVF
1.	Abdul eisa	49	51444	M	II	YES	NO	NO	NO	AW	↑	NO
2.	Abraham	53	54907	M	I	NO	NO	NO	NO	AS	↑	NO
3.	Alagappan	27	43676	M	I	YES	NO	NO	NO	EA	↑	NO
4.	Alagarsamy	56	48644	M	II	YES	NO	NO	YES	EA	↑	NO
5.	Alamelu	54	55724	F	II	NO	NO	NO	YES	AL	↑	NO
6.	Anandhan	71	47067	M	I	NO	NO	NO	NO	AS	↑	NO
7.	Antony	39	47644	M	I	NO	NO	NO	NO	AW	↑	YES
8.	Arukannan	44	45679	M	I	YES	NO	NO	NO	IW	↑	NO
9.	Arulselvan	35	53724	M	I	NO	NO	NO	NO	AL	↑	NO
10.	Arumugam	55	52787	M	I	YES	NO	NO	NO	IW	↑	NO
11.	Arusamy	57	44780	M	II	NO	NO	YES	NO	IW	↑	NO
12.	Bannariappan	57	47110	M	I	NO	YES	NO	NO	AW	↑	YES
13.	Chandrasekar	54	46944	M	I	YES	NO	YES	NO	AS	↑	NO
14.	Chellam	57	47446	F	I	NO	NO	YES	NO	AW	↑	NO
15.	Chellamuthu	43	34216	M	I	YES	NO	NO	NO	AW	↑	YES
16.	Chinnakannu	64	47679	F	II	NO	NO	YES	NO	HL	↑	NO
17.	Chinnappan	59	48199	M	III	YES	NO	NO	YES	AL	↑	NO
18.	Chinnasamy	62	50114	M	I	NO	NO	YES	NO	IW	↑	NO
19.	Deivasakayam	51	48109	M	I	YES	NO	NO	YES	IW	↑	NO
20.	Devan	25	51274	M	II	NO	NO	NO	NO	AW	↑	YES
21.	Devikumari	57	40090	F	I	YES	NO	NO	YES	IW	↑	NO
22.	Dhanasekar	55	34569	M	I	NO	NO	NO	NO	EA	↑	YES
23.	Dharmalingam	52	46700	M	I	NO	NO	YES	NO	AL	↑	NO
24.	Dharman	47	48796	M	I	NO	YES	NO	NO	AW	↑	NO
25.	Duraisamy	57	48909	M	I	NO	NO	NO	NO	AW	↑	NO
26.	Enjappan	54	46700	M	I	YES	NO	NO	NO	EA	↑	YES
27.	Farooq	37	52274	M	I	NO	NO	YES	NO	AW	↑	YES
28.	Fathima	46	50101	F	I	NO	YES	NO	NO	AW	↑	YES
29.	Feroze ahamed	26	45734	M	I	YES	NO	NO	NO	AW	↑	YES
S. No	**Name**	**Age (Yrs)**	**IP No.**	**Sex**	**Killips class**	**Smoking**	**DM**	**HT**	**CAHD / Angina**	**Infarct size**	**CPK – MB**	**LVF**
30.	Ganesan	53	46443	M	I	YES	NO	NO	NO	AS	↑	NO
31.	Govindasamy	73	50203	M	I	YES	NO	NO	NO	AW	↑	NO
32.	Ilavarasan	54	46364	M	I	NO	YES	NO	YES	IW	↑	NO
33.	Illaippan	61	53477	M	I	YES	NO	NO	NO	AL	↑	NO
34.	Iqbal khan	23	48700	M	I	NO	NO	NO	NO	AW	↑	YES
35.	John Eapan	31	32167	M	I	YES	NO	YES	NO	IW	↑	NO
36.	Kalaivani	57	51104	F	II	NO	NO	NO	NO	IW	↑	NO
37.	Kamalammal	43	49709	F	II	NO	YES	YES	NO	HL	↑	NO
38.	Kandappan	62	47384	M	I	YES	NO	NO	YES	HL	↑	NO
39.	Kandasamy	31	44113	M	I	YES	NO	NO	NO	AS	↑	NO
40.	Karuppan	54	45555	M	I	NO	NO	YES	NO	EA	↑	NO
41.	Karuppayi	63	44676	F	III	NO	NO	YES	NO	AL	↑	NO
42.	Karuppusamy	56	43772	M	I	YES	NO	NO	NO	IW	↑	YES
43.	Kasthuriraja	53	47819	M	I	YES	NO	YES	NO	IW	↑	YES
44.	Konthaiappan	47	45103	M	II	YES	NO	YES	NO	AS	↑	NO
45.	Kumaralingam	35	52031	M	II	YES	NO	NO	YES	AW	↑	NO
46.	Kumaran	72	46716	M	IV	YES	NO	NO	YES	HL	↑	NO
47.	Kuppusamy	55	53766	M	I	NO	YES	NO	NO	AW	↑	NO

48.	Mohammed	42	52307	M	I	YES	NO	YES	NO	AW	↑	YES	
49.	Mohanavelu	53	51977	M	I	NO	YES	NO	YES	AS	↑	NO	
50.	Mohankumar	58	50876	M	I	NO	NO	NO	NO	IW	↑	NO	
51.	Palaniandhavar	44	45704	M	II	YES	NO	YES	NO	IW	↑	NO	
52.	Palanisamy	53	40595	M	I	YES	NO	NO	YES	AW	↑	YES	
53.	Palanivelan	28	46378	M	I	NO	NO	NO	YES	EA	↑	NO	
54.	Pappan	51	49876	M	III	YES	NO	NO	NO	AS	↑	NO	
55.	Raman	64	37862	M	III	NO	NO	YES	YES	IW	↑	NO	
56.	Rasukannan	45	48707	M	I	YES	NO	NO	NO	AW	↑	YES	
57.	Rathnam	69	40596	M	III	YES	NO	YES	YES	HL	↑	NO	
58.	Rathnasamy	55	34569	M	I	YES	NO	NO	NO	EA	↑	YES	
59.	Raveendran	59	46967	M	I	YES	NO	YES	NO	AS	↑	YES	
60.	Selvamuthu	56	47034	M	I	NO	NO	NO	NO	IW	↑	NO	
61.	Sengaliappan	73	43780	M	IV	NO	YES	NO	NO	AW	↑	NO	
S. No	**Name**	**Age (Yrs)**	**IP No.**	**Sex**	**Killips class**	**Smoking**	**DM**	**HT**	**CAHD / Angina**	**Infarct size**	**CPK – MB**	**LVF**	
62.	Sengodan	37	52476	M	I	YES	NO	NO	NO	IW	↑	NO	
63.	Senthilkumar	43	54472	M	I	YES	NO	NO	YES	EA	↑	NO	
64.	Shanmugam	49	49109	M	I	NO	NO	NO	NO	HL	↑	NO	
65.	Silamban	56	51031	M	III	NO	NO	NO	YES	EA	↑	NO	
66.	Sundaram	51	50132	M	I	YES	NO	NO	NO	AW	↑	YES	
67.	Surendran	51	53499	M	I	YES	NO	NO	NO	AS	↑	NO	
68.	Thavamani	45	49116	F	I	NO	NO	NO	YES	EA	↑	NO	
69.	Vaitheswaran	42	50012	M	I	YES	NO	YES	YES	AW	↑	YES	
70.	Velusamy	68	35786	M	II	YES	NO	NO	YES	IW	↑	YES	

IP – Inpatient **M – Male** **F – Female**

DM – Diabetes Mellitus

HT – Hypertension **CAHD – Coronary Artery Heart Disease** **AW – Anterior Wall**

AS - Antero Septal

IW – Inferior Wall **AL – Antero Lateral** **IL – Infero Lateral**

HL – High Lateral

EA – Extensive Anterior Wall **CPK – Creatinine Phosphokinase**

LVF – Left Ventricular Failure

Tachy arrhyth – Tachy arrhythmias **Brady arrhyth – Brady arrhythmias**

Cond. Dist. – Conduction Disturbances

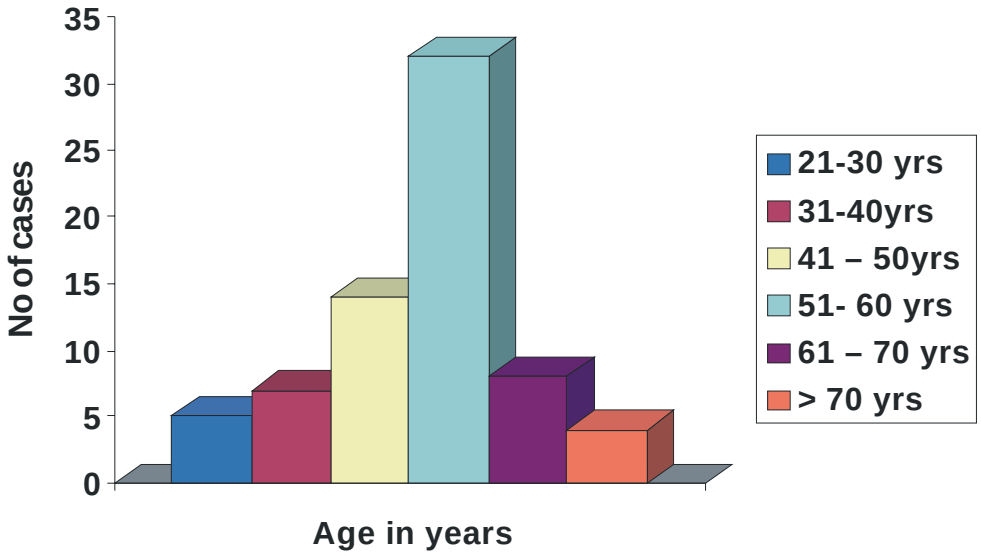


Fig1. Showing Age wise distribution of AMI

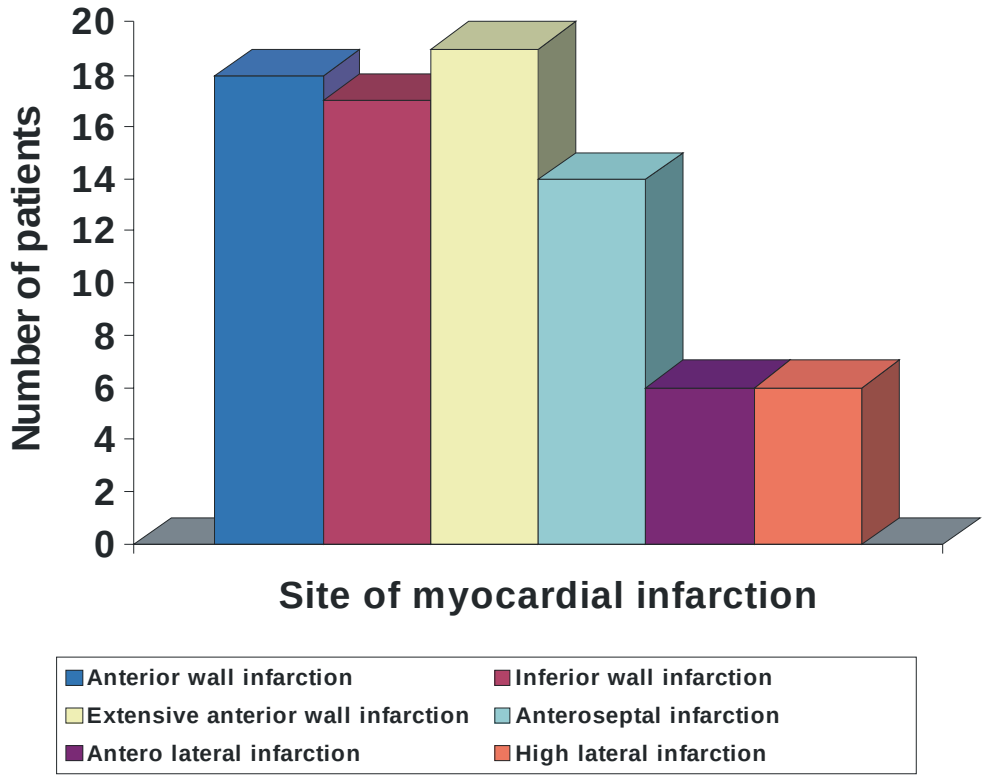


Fig2. Showing Site of myocardial infarction

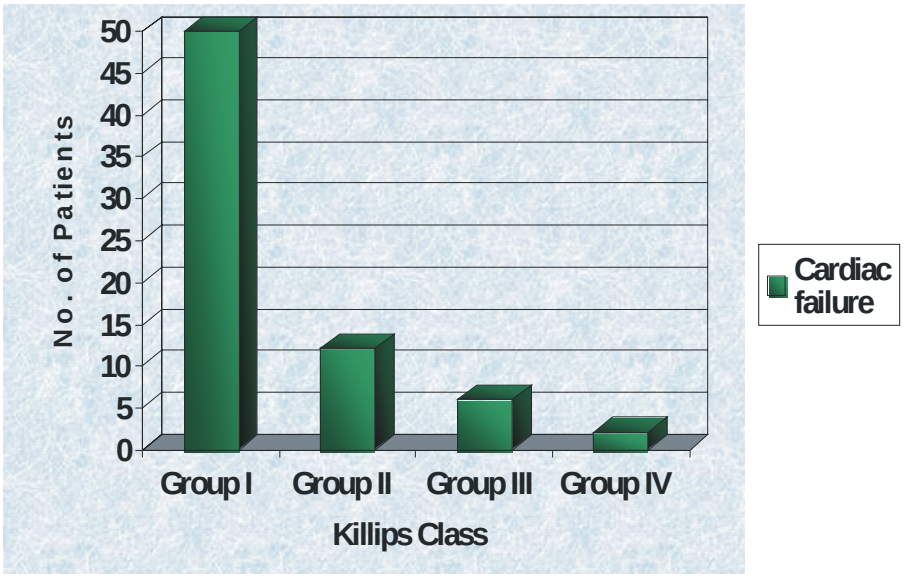


Fig3. Showing Class wise distribution of cardiac failure

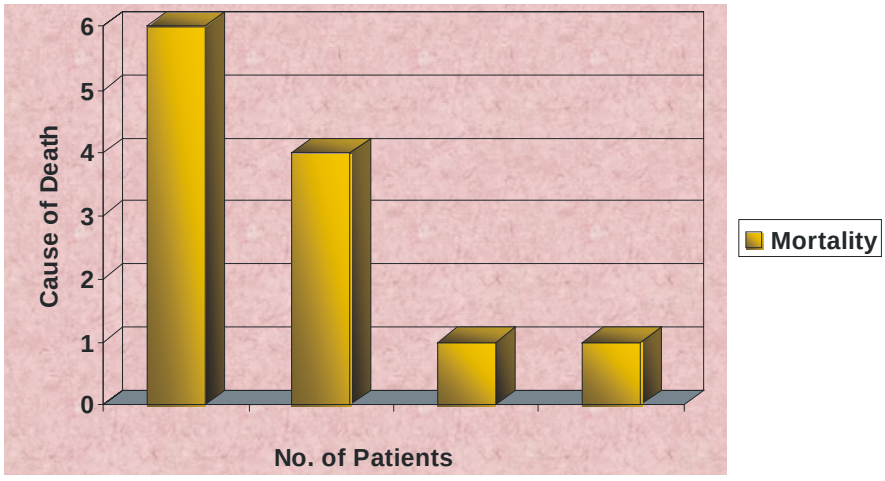


Fig4. Showing distribution of deaths in AMI

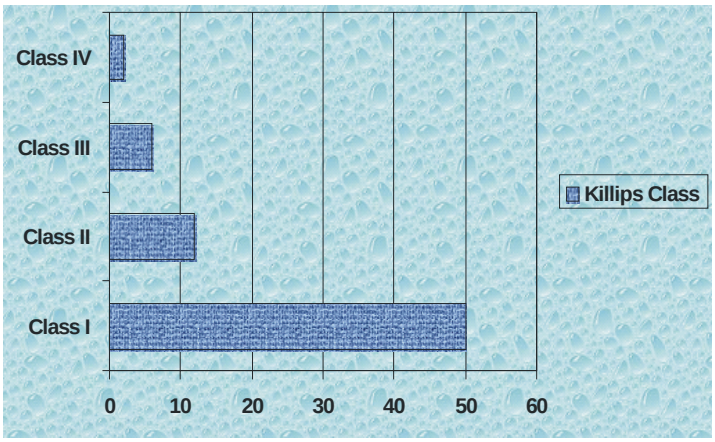


Fig5. Showing No. of patients in killips class

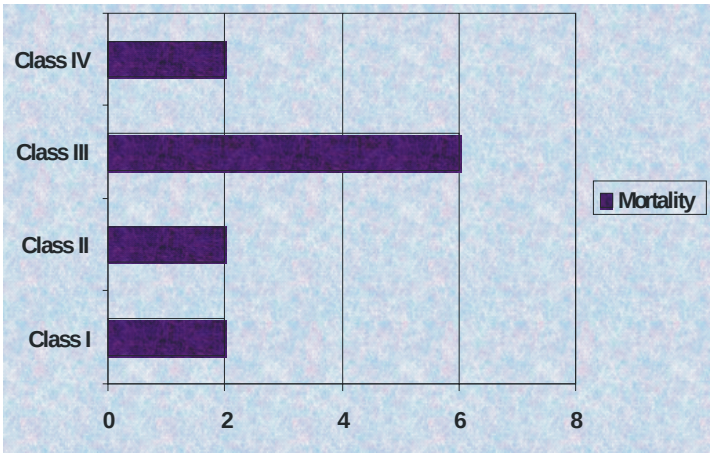


Fig6. Showing No. of deaths in killips class

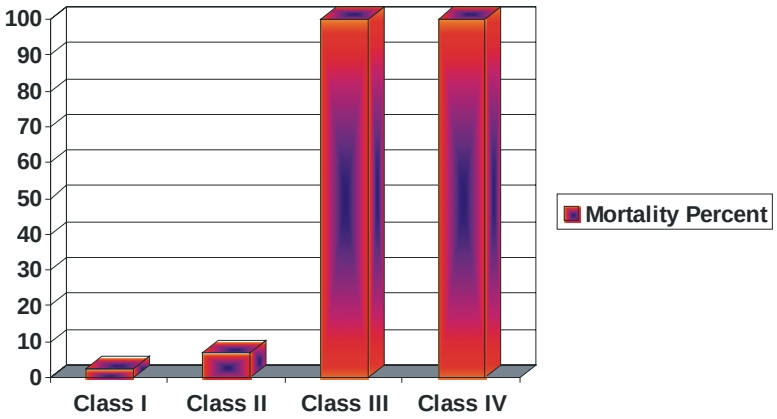
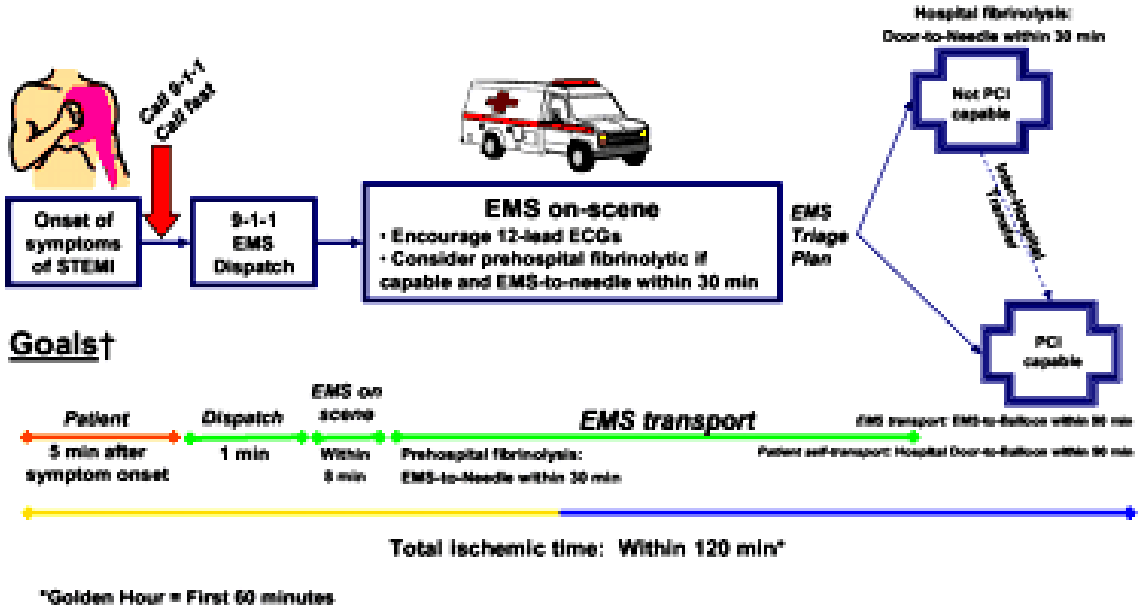


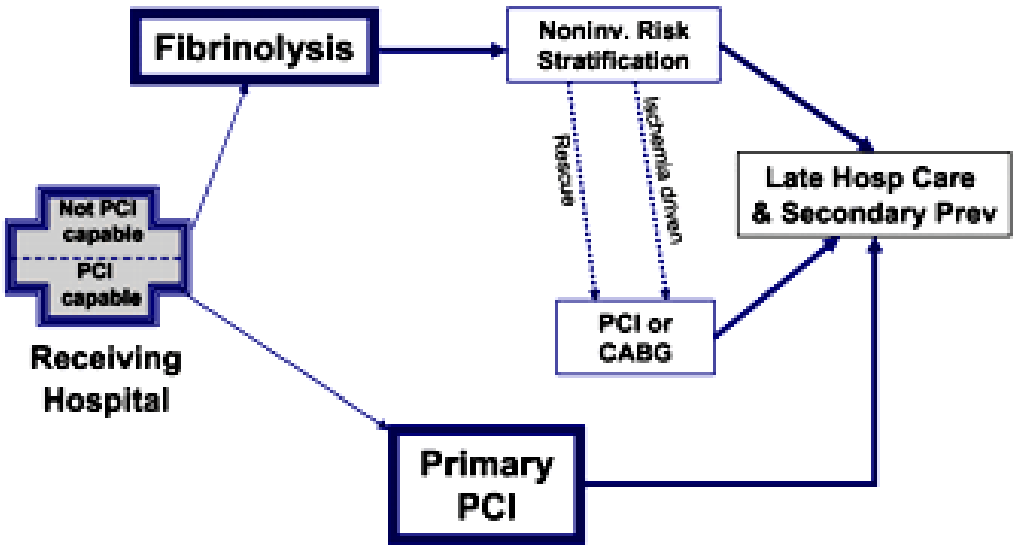
Fig7. Showing percentage of deaths in killips class

Panel A

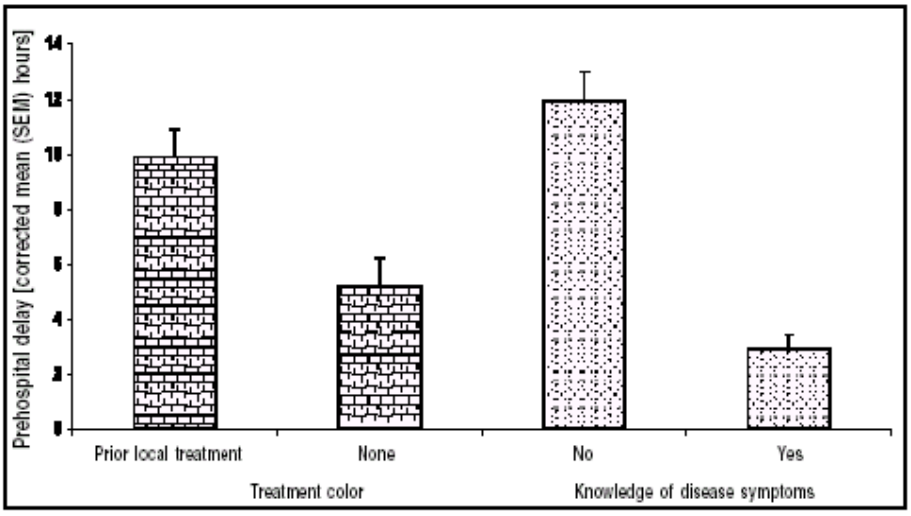


*Golden Hour = First 60 minutes

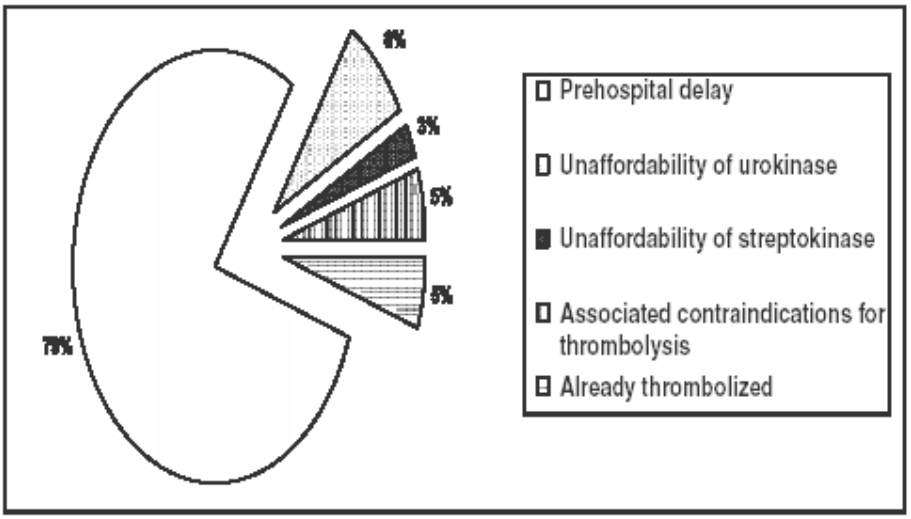
Panel B



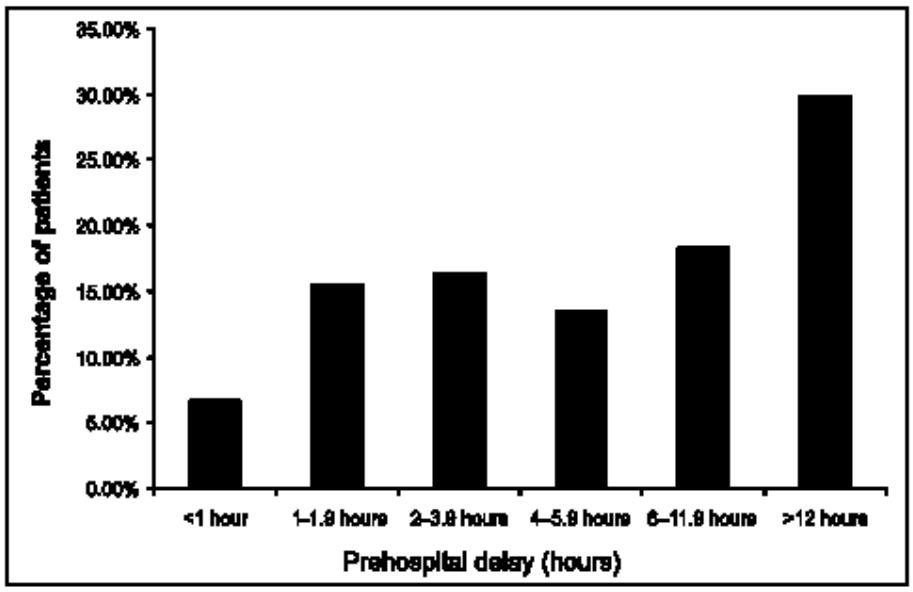
PREHOSPITAL CORONARY CARE



EFFECT OF TREATMENT ORDER AND PATIENTS KNOWLEDGE OF DISEASE SYMPTOMS ON PRE-HOSPITAL DELAY

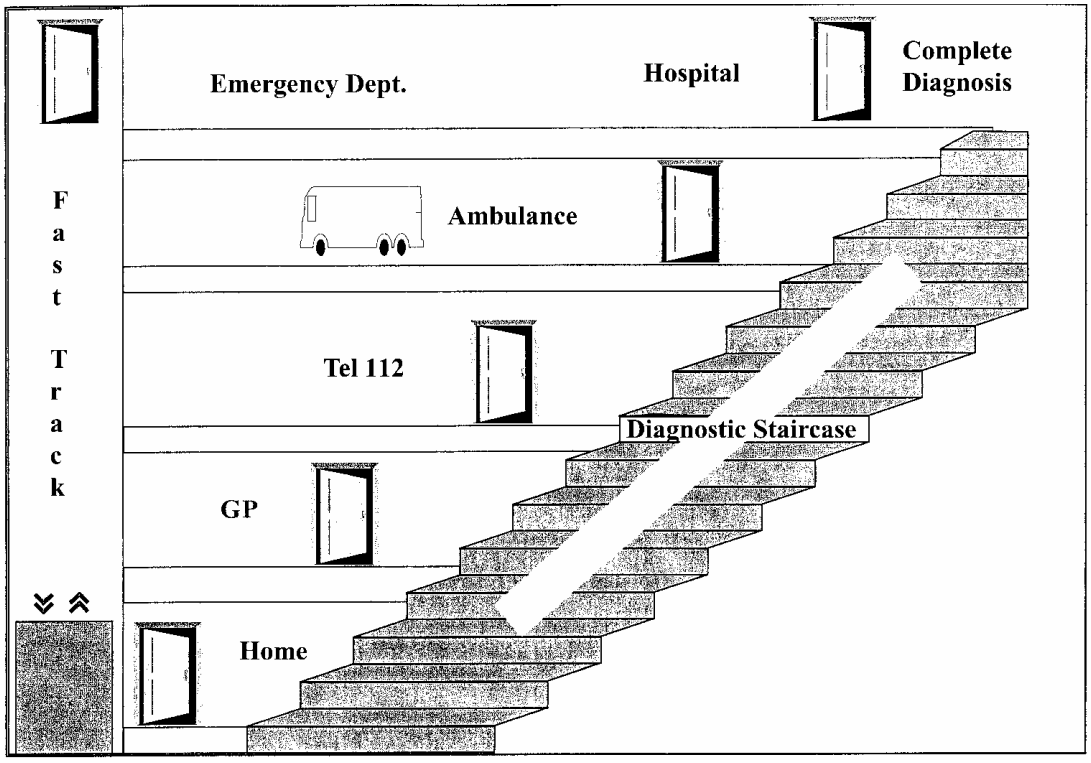


REASONS FOR NOT RECEIVING THROMBOLYTIC THERAPY

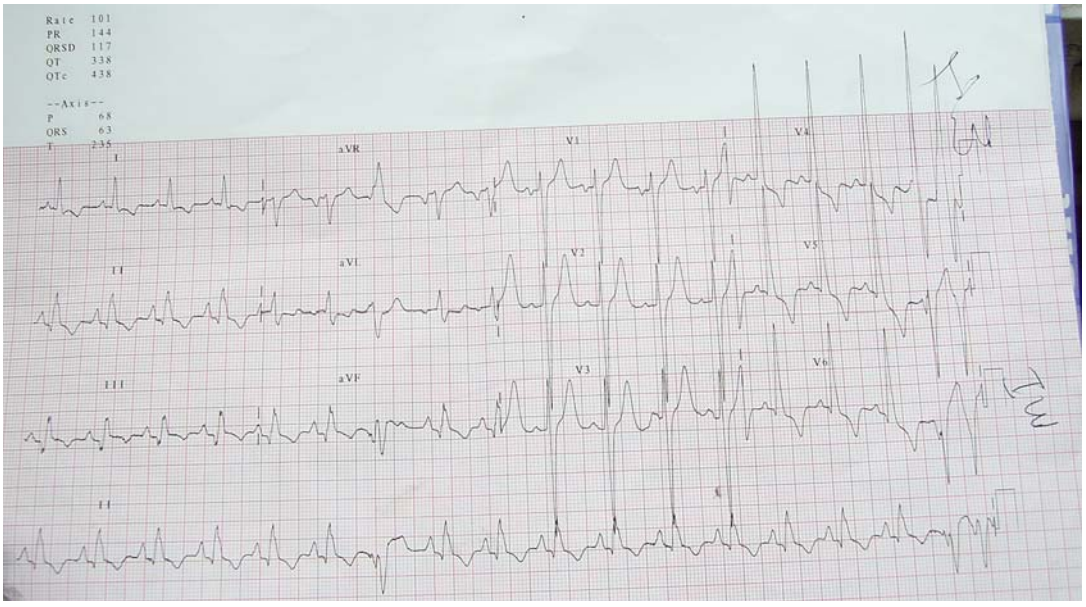


PREVALENCE OF PRE-HOSPITAL DELAY

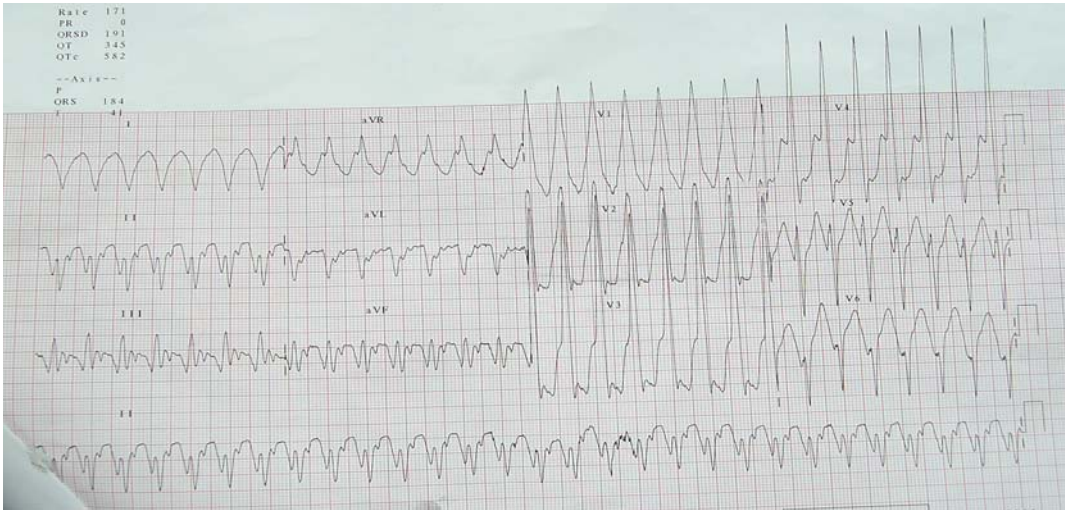
MANAGEMENT OF CHEST PAIN



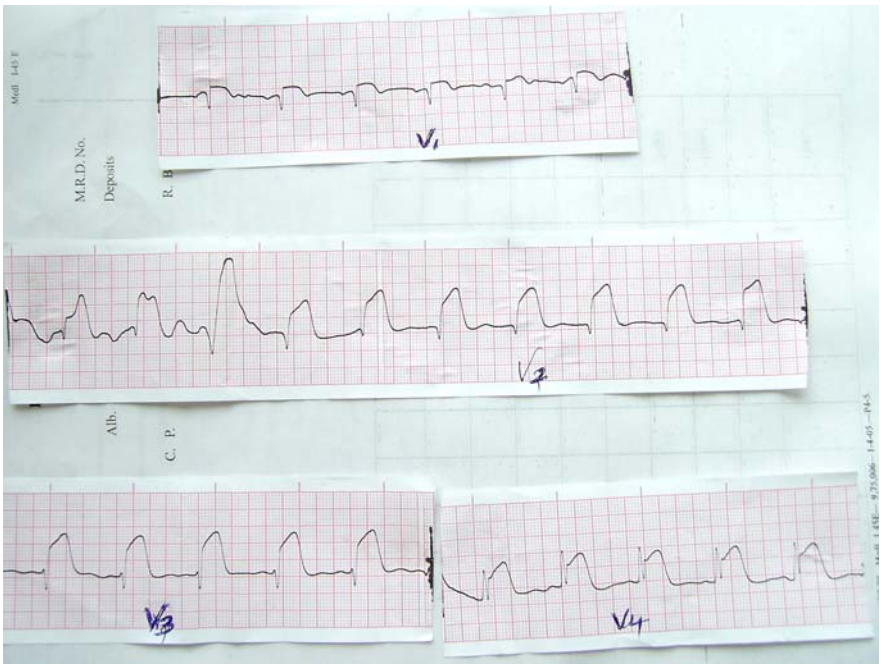
The Five Doors Representing Five Diff Levels of Decision Making



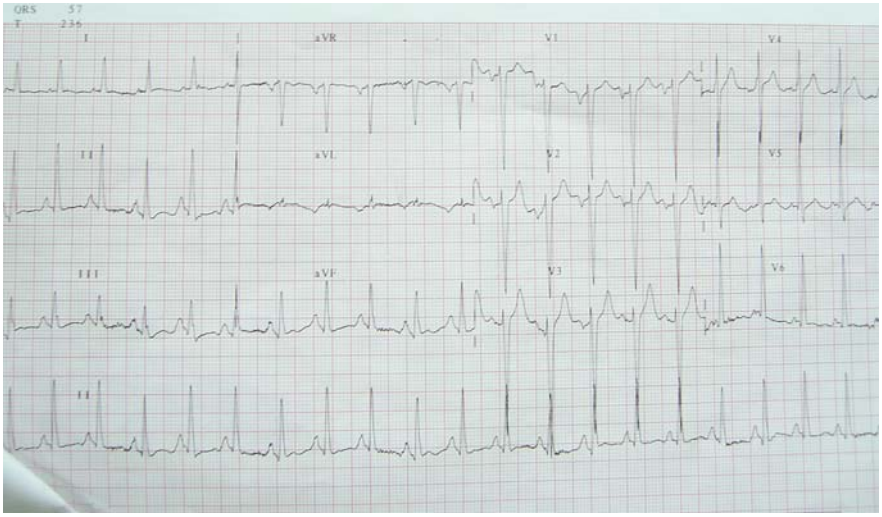
ECG SHOWING LEFT BUNDLE BRANCH BLOCK



ECG SHOWING BROAD COMPLEX TACHYCARDIA



ECG SHOWING ACUTE ANTEROSEPTAL MI



ECG SHOWING NARROW COMPLEX TACHYCARDIA

PROFORMA

Name : **Age :** **Sex :**

Address : **Ward :** **IP No :**

Date of Admission :

Date of Discharge :

History of present illness :

1. Dyspnoea
2. Dyspnoea grade
3. Orthopnoea
4. PND
5. Chest pain
6. Easy fatiguability
7. Giddiness
8. Syncope
9. Hemoptysis
10. Palpitation

Past History :

I. History of myocardial infarction

Number of infarction/hospital admission for heart ailment

History of first infarction

1. Time of onset of chest pain
2. Time window for hospitalization
3. Thrombolytic therapy
4. Duration of hospital stay

II. Systemic hypertension

III. Diabetes

IV. Rheumatic heart disease

V. Peripheral vascular disease

VI. Bronchial asthma

VII. Congenital heart disease

VIII. Any other chronic/acute relevant medical illness

TREATMENT HISTORY

FAMILY HISTORY

PERSONAL HISTORY

Nature of Job :

Type of Food :

Smoking :

Alcohol :

Oral Contraception :

Tobacco Chewing :

CLINICAL EXAMINATION

Temperature :

Pulse :

Respiration :

BP :

JVP :

Edema / Ascites :

Height :

Weight :

Obesity :

Arcus senilis :

Xanthoma :

Xanthelasma :

Carotids :

Locomotor Brachii :

Anaemia :

Clubbing :

Cyanosis :

Jaundice :

Polycythemic features :

SYSTEMIC EXAMINATION

EXAMINATION OF CARDIOVASCULAR SYSTEM :

SOUNDS :

MURMURS :

RUB :

KNOCK SOUND :

ADDITIONAL EVENTS :

EXAMINATION OF RESPIRATORY SYSTEM

BREATH SOUNDS, RALES, WHEEZE

EXAMINATION OF ABDOMEN

EXAMINATION OF CNS

INVESTIGATIONS

ECG

SERUM-CPK-MB LEVELS

FINAL DIAGNOSIS

COMMENT