REVIEW
OF
LITERATURE
Myocardial infarction is almost always due to the formation of occlusive thrombus at the site of rupture of an atheromatous plaque in a coronary artery. In the United States approximately 70,000 coronary artery related deaths and more than 1.1 million Acute Myocardial Infarction occur each year. The epidemics of cardiovascular disease especially coronary heart disease are emerging in India. In 1990 cardiovascular disease accounted for 2.4 million deaths in India with an annual loss of 28.5 million disability adjusted life years\textsuperscript{12}. The factors underlying the special predilection of South Asian for coronary heart disease are a high prevalence of glucose intolerance, central obesity, elevated plasma triglycerides, decreased plasma HDL cholesterol & hyper insulinemia. The confluence of genetically determined elevation of lipoprotein 'a' (LP(a)), possible genetic susceptibility to the metabolic syndrome and environmental influence which enhance the propensity for diabetes and elevated total cholesterol level appears to make the South Asian immigrants especially vulnerable to coronary artery disease.

The excess of coronary mortality and morbidity noted in persons of South Asian ethnicity in comparison to many other ethnic groups, implicate gene environment interactions which amplify the risk for Indians. Such susceptibility may be due to a "thrifty gene" selecting for survival over millennia or a thrifty phenotype emerging alive from the adverse intra-uterine environment of an impoverished pregnancy.
Studies of urban rural comparison within Indian and comparison of migrants with non migrants siblings, however, suggest that environment is a major determinant of the extent to which such susceptibility expresses itself as cardio vascular disease whether the programming is due to mother nature or mother’s nurture\textsuperscript{13,14,15}.

VENTRICULAR DYSFUNCTION

Even in the thrombolytic era, left ventricular dysfunction remains the single most important predictor of mortality following Acute Myocardial Infarction. In patients with Acute Myocardial Infarction heart failure is characterised either by systolic dysfunction alone or by both systolic and diastolic dysfunction. Left ventricular diastolic dysfunction leads to pulmonary venous hypertension and pulmonary congestion, whereas systolic dysfunction is principally responsible for a depression of cardiac output and of the ejection fraction. Clinical manifestation of left ventricular failure become more common as the extent of the injury to the left ventricle increase. The dysfunction however is due to 2 components:

(i) Irreversible i.e. due to necrosis of the myocardium.

(ii) Reversible this may be due to stunned or hibernating myocardium.

(a) Stunned Myocardium\textsuperscript{16} : Left Ventricle dysfunction due to the transient coronary occlusion followed by restoration of blood flow even though myocardial necrosis is minimal or absent. This may persist for hours to several weeks.
(b) *Hibernating Myocardium* 16: This is chronic depression of left ventricular function caused by prolonged periods of ischemia due to severe obstruction in coronary arteries. This coronary blood flow is inadequate to support myocardial contractile function but is adequate to maintain cellular structure and integrity. If myocardial perfusion is restored, systolic function improves. Duration may vary from months to years.

**Diastolic Dysfunction**: Implies elevation of left ventricular end diastolic pressure (LVEDP) with normal left ventricle end diastolic Volume (EDV) (55-85 ml/mt) and normal ejection fraction.

**Systolic Dysfunction**: Implies raised EDV with LVEDP and diminished EF. Systolic dysfunction is more common (70%) than diastolic dysfunction (30%).

* 2 D – Echo Doppler has emerged as the modality of choice for evaluating diastolic & systolic dysfunction.

S.H. Poulser et al17 had studied 65 consecutive patients admitted to the coronary care unit with first Acute Myocardial Infarction defined according to the WHO criteria with 2 out of the following three characteristics.

1. Typical chest pain.

2. Electrocardiographic evidence of myocardial infarction (ST elevation > 1 mm in contiguous leads or subendocardial injury pattern) and

3. Transient elevation of creatinine kinase MB ≥ 20 U/L (Normal < 6 U/L)
All patients were between 40-75 years of age. No patient had significant valvular heart disease and all were in sinus rhythm. Pulsed Doppler echocardiography of transmitral and pulmonary venous flow was assessed in 65 consecutive patients within 1 hr. of arrival in the coronary care unit. Isolated left ventricular systolic dysfunction (EF < 50% and normal filling pattern) was found in 18 patients (21%). Left ventricular diastolic dysfunction (defined as impaired relaxation or a pseudonormalized or restrictive filling pattern) and a normal systolic function was found in 15 patients (24%). Combined systolic and diastolic dysfunction was found in 24 patients (38%) and combined normal systolic and diastolic function in 11 patients (17%). Left ventricular diastolic dysfunction is present early after onset of symptoms of a first Acute Myocardial Infarction. Further more left ventricular diastolic function seems to play an important role in the development of clinical heart failure following Acute Myocardial Infarction. Impaired relaxation of the left ventricle was the predominant diastolic filling abnormality (37%) but pseudonormal or restrictive filling pattern were also frequently present (25%) in patients with a first Acute Myocardial Infarction. In addition 24% of the patients had isolated diastolic dysfunction. Development of congestive heart failure occurred in 31% of the patients during the first week after the acute events. Although most patients with heart failure had early systolic dysfunction with decreased EF, a sub group (23%) had an abnormal filling pattern with preserved ejection fraction. Patients at risk of developing congestive heart failure during the first week of a first Acute Myocardial Infarction are best identified by a short mitral E deceleration time.
CARDIOGENIC SHOCK\textsuperscript{18}:

Cardiogenic shock is defined as a critical reduction in tissue perfusion caused by a critical fall of cardiac output to levels that are inadequate to support end organ function. The two cardinal elements of cardiogenic shock are hypotension and hypoperfusion. The clinical presentation of cardiogenic shock includes:

1. Systolic pressure < 90mm Hg,
2. Peripheral vasoconstriction with cool extremities and acrocyanosis
3. Urine output – 20 ml/hr
4. Altered mental status with agitation or obtundation

Various pathologic condition in the setting of Acute Myocardial Infarction can induce cardiogenic shock e.g. extensive infarction involving a large area of left/right ventricular myocardium is the most frequent cause, other causes include mechanical complication such as severe mitral regurgitation, ventricular septal defect and cardiac tamponade with or without free wall rupture.

Cardiogenic shock occurs in 6\% to 20\% of patients with Acute Myocardial Infarction\textsuperscript{19,20,21}. In the Worcester heart attack study\textsuperscript{22} over a period of 13 years from 1975 to 1988, the incidence of cardiogenic shock ranged from 6.7 to 9.1\% in a sample of 4762 patients with Acute Myocardial Infarction admitted to 16 community hospitals. During that time interval, the rate of this complication showed an acute increase after adjustment for baseline variables with adjusted relative risk of 0.83 in 1978 & 1.65 in 1988. The in hospital survival of patients enrolled in this study actually worsened between 1975 &
1988 with a mortality rate of 74% in 1975 & of 82% in 1988. Aggressive strategies were applied only infrequently in this study < 2% underwent coronary angioplasty, further more, thrombolytic therapy was infrequently used.

In the Global utilization of streptokinase and Tissue plasminogen activator for occluded coronary arteries (Gusto-1) trial of 41,021 patients, cardiogenic shock occurred in 7.2% of patients. The 30 days mortality rate for the patients who had shock was 55% and accounted for 58% of the total mortality of the trial. The comparison between resource consumption in the United States and that of other countries for the patients with shock enrolled in the trial showed that every intervention was performed significantly more frequently in the US patients including cardiac catheterization (58% vs 23%), intra-aortic balloon pump (35% vs 7%), coronary by pass surgery (16% vs 9%) or coronary angioplasty (26% vs 8%). Adjusted 30 days mortality rate was significantly lower among pt treated in United States than among those treated in other countries (50% vs 66%) with a 24% reduction. In the multivariate analysis, age and systolic blood pressure were the only factors more strongly associated with increased 30 days mortality rate than geographical location. In all countries mortality rate at a 1 year was lower for patients who underwent coronary angioplasty, whereas coronary surgery did not significantly affect 1 year mortality.

Bengtson et al\textsuperscript{23} showed that the most important independent predictor of in hospital and long term mortality rates was patency of the infarct related artery. The in hospital mortality rate in patients with patent infarct-related arteries was 33% vs 75% in those with closed arteries and 84% in those with unknown status of the arteries. Therefore the main goal of current treatment of
cardiogenic shock in Acute Myocardial Infarction is aggressive myocardial revascularization, aiming to maximize myocardial salvage.

Dzavik et al\textsuperscript{24} described significant increase from 1989 to 1995 in the use of cardiac catheterisation interventional procedures and intra-aortic balloon pump in all age groups, although an aggressive strategy was less frequently used in patients > 75 years old. In hospital survival of patients < 65 years old improved from 10% in 1989 to 1990 to 59% in 1993 to 1995, survival of patients > 75 years old was 20% in 1993 to 1995. Use of thrombolytic therapy showed possible survival benefit only in patients > 75 years old (Thrombolysis 33% vs no thrombolysis 5%).

The GISSI study\textsuperscript{25} (The Gruppo italiano per studio della sopravvivenza nell' infarto miocardico) was the only large randomized, placebo-controlled trial of thrombolysis that enrolled patients with shock. In the small cohort including 280 patients, the 30 days mortality rate was 69.9% for patients of the streptokinase group.

In the meta analysis of several large, placebo-controlled, randomized thrombolytic trials by the Fibrinolytic therapy Trialists (FTT) collaborative Group\textsuperscript{26}, the 35 days mortality rate of patients with hypotension (systolic blood pressure < 100 mg hg) treated with thrombolysis was significantly lower that of patients of the placebo group (29% vs 35%). When both hypotension and tachycardia (heart rate > 100 beats/m in) were present the correspondent 35 days mortality rates were 54% and 61%. In regards to the choice of the rest thrombolytic agent in the setting of cardiogenic shock the international study\textsuperscript{27} found a higher mortality rate in patients
treated with recombinant tissue plasminogen activator (r-TPA) compared with streptokinase (78% vs 68%). Similar data are reported by GUSTO-1 study (30 days mortality rate 57% for patients treated with r-TPA vs 51% for patients treated with streptokinase)\(^{28}\).

In the society for cardiac angiography registry mortality rate was 72% in reperfused patients and 84% in those without reperfusion. In the Duke series, in hospital mortality rate was 30% for patients treated with thrombolysis of infarct related artery at the time of catheterization.

To improve the results of thrombolysis, an aggressive use of vasopressors or intra-aortic balloon pump (IABP) has been advocated. The consequent increase in blood pressure would have the potential to restore the rate of thrombus dissolution to normal values\(^{29,30}\).

In the GUSTO-1 study\(^{31}\), in which all patients underwent a thrombolytic regimen, early IABP institution was associated with a trend toward lower 30-days and 1-year mortality rates. In a retrospective evaluation of the National Registry of Myocardial Infarction the use of IABP in combination with thrombolytic therapy in patients with Acute Myocardial Infarction complicated by cardiogenic shock was associated with a marked reduction in mortality rate\(^{32}\), conversely, a benefit was not seen in patients undergoing coronary angioplasty. In the SHOCK trial registry 27, thrombolysis and IABP were associated with a 17% and 32% lower mortality rate, respectively, and a 39% lower mortality rate when combined in comparison with the mortality rate observed in patients not treated with either thrombolysis or IABP.
RIGHT VENTRICULAR INFARCTION

For a clinician myocardial infarction meant infarction of the "Left Ventricle" only till 1974, when Cohn et al for the first time described potentially serious and unique haemodynamic consequences of right ventricular infarction. About 2/3 of patients to the hospital with Acute Myocardial Infarction show the ECG evidence of infarction of the anterior wall of left ventricle and approximately one third show the evidence of inferior wall Myocardial Infarction. Right ventricular infarction has been reported to occur in about 1/3 of the cases of inferior wall Myocardial Infarction, Isolated Right Ventricular Infarction also does occur and has been reported to be in less than 2% of cases of Acute Myocardial Infarction patients seen on autopsy.

Various pathological studies reveal that Right Ventricle Infarction is present in 14 to 34% of patients with transmural left ventricular infarction. The evidence of Right Ventricle Infarction as compared to left ventricular infarction is low, the possible mechanisms are –

1. The smaller mass of right ventricle
2. The lower tension within the right ventricular wall
3. The richer collateral circulation of right ventricle
4. The thickness of the right ventricular wall perhaps enables the chamber to derive relatively more nutrition from blood within its cavity.
normal descent of right ventricle base virtually excludes haemodynamically significant RVMI.

**Mechanical Complications of Myocardial Infarction**

Includes Ventricular septal defect, free wall rupture, papillary muscle rupture as a group mortality due to these complications are about 15% from Acute Myocardial Infarction.

Rupture of the free wall of the infarcted ventricle occurs in upto 10% of patients dying in the hospital of Acute Myocardial Infarction.

Factor responsible for rupture are :-

1. Thinness of the apical wall
2. Marked intensity of necrosis
3. Poor collateral flow
4. The shearing effect of muscular contraction against an inert and stiffened necrotic area.
5. Aging of the myocardium with laceration of the myocardial microstructure.

Have all been responsible for rupture.

It occurs more, frequently in the elderly & women and appears to be more common in hypertensive than in normotensive patients. It occurs approximately 7 times more frequently in the left than the right ventricle and seldom occurs in the atria. Usually involve the anterior or lateral wall of the ventricle in the area of
In approximately 30% of the patients (20%-45%) with inferior left ventricular wall myocardial infarction there is some evidence of R.V. necrosis.

The classic diagnostic feature of RVMI in ECG is 1 mm or more of ST elevation in the right precordial leads from V₃R to V₆R, however it is more specific in V₄R. The ST segment, which is higher in lead V₄R than in leads V₁ and V₃R offers the highest specificity and sensitivity in diagnosis. The overall specificity and sensitivity is 80% to 90%. Echocardiography helps to assess the site extent and severity of right ventricle wall motion abnormality. The sensitivity and specificity for diagnosis are about 82% and 93% respectively.

Goldberger studied 24 cases of right ventricle Myocardial Infarction of which 9 were haemodynamically significant i.e. JVP > 17 cm H₂O or a RA pressure of >13 mmHg. The descent of right ventricle base (a measure of right ventricle ejection fraction) was 0.7 + 2 cm in systole as compared to 1.3 + 0.4 cm in patients with non haemodynamically significant RVMI and 2+0.2 cm in normals. The sensitivity of this finding was 100% and specificity was 80%. The respiratory caval index (% collapse of inferior vena cava with inspiration) an index of increased RA pressure, was 22% ± 11% in haemodynamically significant RVMI as compared to 45% ± 15% in patients with haemodynamical stability. Finally an increased ratio of right ventricle to left ventricle end diastolic dimension greater than 0.9 was 50% sensitive & 87% specific for the haemodynamically compromised. The clinical implication of this study indicated that haemodynamically most useful of these measurements is the descent of right ventricle base. A normal respiratory caval index or a
distribution of left anterior descending coronary artery. Commonly associated with large transmural infarction involving at least 20% of the left ventricle. It can occur between 1st day & 3 weeks but most commonly 1 to 4 days following infarction and usually occurs near the junction of the infarcted and normal muscle.

Rupture of the free wall of the left ventricle usually leads to hemopericardium and death from cardiac tamponade. Occasionally, rupture of the free wall of the ventricle occurs as the first clinical manifestation in patients with undetected or silent myocardial infarction and then it may be considered a form of "Sudden Cardiac death".

Incomplete rupture of the heart may occur when organizing thrombus and hematoma, together with pericardium, seal a rupture of the left ventricle and thus prevent the development of hemopericardium. With time this area of organised thrombus & pericardium become a pseudoaneurysm/false aneurysm that maintains communication with the cavity of the left ventricle in contrast to true aneurysm which always contain some myocardial elements in their walls. The walls of pseudoaneurysm are composed of organised hematoma and pericardium and lack any elements of the original myocardial wall. Pseudo aneurysm can become quite large even equaling the true ventricular cavity through a narrow neck. Frequently pseudoaneurysm contain significant quantities of old and recent thrombus which can cause arterial emboli.

_Cabral S et al_ (1998), reported that post Myocardial infarction left ventricular pseudoaneurysm resulting from free wall rupture is a very rare finding. Its recognition during life is even rarer.
Definitive preoperative diagnosis is difficult. A case of a left ventricular pseudoaneurysm as a mechanical complication of Acute Myocardial Infarction was diagnosed by transthoracic echocardiography.

*Mesa Garcia JM et al*36, 1998 reported that Mechanical complication after an Acute Myocardial Infarction, specially the subacute ventricular rupture was the most frequent complication. Mechanical complications constitute the second cause of death after Acute Myocardial Infarction following pump failure. The most frequent mechanical complication is ventricular rupture, which is the cause of death in 26% of the cases of Acute Myocardial Infarction. The incidence of septal & papillary muscle rupture is considerably less frequent.

*Tate Da et al*37, 1998 reported that ventricular free wall rupture is a well known catastrophe of Acute Myocardial Infarction. A significant number of patients present in a subacute fashion and can be successfully treated with surgery if diagnosed promptly. They presented a case of subacute free wall rupture that occurred after an undiagnosed myocardial infarction. The findings at pericardio centesis were unusual in that the fluid was sanguinous but not frank hemopericardium. This patient represents the first known reported case to present without frank hemopericardium who survived and was successfully treated surgically. The absence of frank hemopericardium showed not exclude the diagnosis of free wall rupture.

*Tikiz H et al*38, 2001 had done study on 350 consecutive patients suffering from first attack of Acute Myocardial Infarction.
The study aimed to determine the independent factors involved in the development of left ventricular aneurysm in Acute Myocardial Infarction. The overall incidence of left ventricular aneurysm was 11.7% (41/300) and no statistical difference was found between the incidence of left ventricular aneurysm between the 2 group i.e. thrombolytic group and control group. In univariate analysis, vessel patency, proximal left anterior descending artery (left anterior descending) stenosis, total left anterior descending occlusion, multivessel disease and hypertension were found to be important factors in left ventricular aneurysm formation after Acute Myocardial Infarction. Conclusion of the study is that not all patients who received thrombolytic therapy, but only those with PIRA (Patent infarct related artery) had evidently reduced incidence of left ventricular aneurysm. Patients with total occlusion with proximal left anterior descending stenosis and without PIRA (Patent infarct related artery) were found to have increased risk for formation of left ventricular aneurysm after Acute Myocardial Infarction. These findings indicate that the presence of vessel patency has a preventive effect on left ventricular aneurysm formation in Acute Myocardial Infarction.

The incidence of rupture of the interventricular septum is probably in the range of 2% of Acute Myocardial Infarction patients because death usually is not immediate and patients frequently can reach the hospital. Clinical features associated with an increased risk of rupture of the IVS (inter ventricular septum), include lack of development of a collateral network, advanced age, hypertension and possibly thrombolysis. The perforation may be a direct through and through opening, or it may be more irregular and serpiginous. The size of the defect determines the magnitude of the left to right
shunt and the extent of hemodynamic deterioration. Rupture of the septum with an anterior infarction tends to be apical in location, whereas inferior infarction are associated with perforation of the basal septum and with a worse than those in an interior location. Clinically it presents with a new harsh, loud holosystolic murmur that is best heard at the left lower sternal border and usually accompanied by thrill. Biventricular failure generally ensues within lows to days. The defect can also be recognized by 2-dimensional echocardiography with colour flow doppler imaging or insertion of a pulmonary artery balloon catheter to document the left to right shunt.

*Di Sunama et al* 1997 had reviewed 34 patients complicating Acute Myocardial Infarction. They reported ventricular septal defect represents a serious complication after Acute Myocardial Infarction with an incidence 1-2%.

*Cooley DA* 1998 reported the post infarction ventricular septal rupture is an uncommon but serious complication of Acute Myocardial Infarction.

*Mesa Garcia J.M. et al* 1998, reported that Mechanical complication after an acute Myocardial Infarction, specially the subacute ventricular rupture was the most frequent complication. Mechanical complication constitute the second cause of death after Acute Myocardial Infarction following pump failure. The most frequent mechanical complication is ventricular rupture which is the cause of death in 26% of the cases of Acute Myocardial Infarction. The incidence of septal & papillary muscle rupture is considerably less frequent.
Partial or total rupture of a papillary muscle is rare but often fatal complication of transmural Myocardial Infarction. Inferior wall infarction can lead to rupture of the posteromedial papillary muscle which occurs more commonly than rupture of the antero lateral muscle, a consequence of anterolateral Myocardial Infarction. Rupture of a right ventricular papillary muscle is rare but can cause massive tricuspid regurgitation and right ventricular failure. Complete transection of a left ventricular papillary muscle is incompatible with life because the sudden massive mitral regurgitation that develops can not be tolerated. Rupture of a portion of papillary muscle, usually the tip or head of muscle, resulting in severe mitral regurgitation, is much more frequent and is not immediately fatal. Unlike rupture of the ventricular septum, which occurs with large infarcts, papillary muscle rupture occurs with a relatively small infarction in approximately one of the case seen in the patients with papillary muscle rupture manifest a new holosystolic murmur and develop increasingly severe heart failure. In both conditions i.e. ventricular septum defect & mitral regurgitation the murmur may become softer or disappear as arterial pressure fall. Mitral regurgitation due to partial or complete rupture of a papillary muscle may be promptly recognized echocardiographically. Color flow Doppler imaging is particularly helpful in distinguishing acute mitral regurgitation from ventricular septal defect in the setting of Acute Myocardial Infarction.

Villavicencio R et al 1991, reported that mitral regurgitation was due to valve ring dilatation with an increase of left ventricle diameter and decrease on ventricular systolic function.
Curcio Ruigomez et al 1997 studied a case of double posterior Acute Myocardial Infarction complication and find ventricular septal defect, Ventricular aneurysm & acute and severe mitral regurgitation and the patients developed pulmonary hypertension.

Honma H et al 1997, Tokyo Japan Evaluated 223 patients of Acute Myocardial Infarction by colour doppler echo. Mitral regurgitation present in 21% of the patients at the onset & developed in 18% of the patients during follow up. The group with unsuccessful recanalization and un-improved mitral regurgitation showed a significantly greater left ventricular end diastolic volume as well as lower left ventricular fraction than the patients with successful recanalization and no Mitral regurgitation. The results suggest that successful recanalization after myocardial infarction may decrease the incidence of mitral regurgitation and may prevent left ventricle remodeling resulting in a secondary improvement of mitral regurgitation.

Benico Barzilia et al in their study documented that only 43% of the total patient with mitral regurgitation detectable by doppler had murmur suggestive of mitral regurgitation when auscultated by an experienced observer, 17% of patients without mitral regurgitation had similar murmur. It may not be surprising that mitral regurgitation increase in incidence between 30 days and 3 months post infarction where remodeling is though to be more extensive. The lack of a relation to the extension of infarction makes ischemia/infarction of papillary muscle, which is thought to be more frequent in patients with inferior wall infarction.
Chuttane S.K. et al\textsuperscript{44} studied 46 patients of Acute Myocardial Infarction with hemodynamic impairment 46% of the total group had echo proved mitral regurgitation while only 24% of them had regurgitation clinically. According to Killip classification 1\textsuperscript{st} class had 25%, 2\textsuperscript{nd} class 44.44%, 3\textsuperscript{rd} and 4\textsuperscript{th} class showed 100% of the clinical mitral regurgitation/echo mitral regurgitation. And as the time passes the tendency of mitral regurgitation increase as during 2\textsuperscript{nd} visit (8\textsuperscript{th} weeks after discharge) 43% of the patients of Killip class 1\textsuperscript{st} and 2\textsuperscript{nd}, and the patients of Killip class 3\textsuperscript{rd} and 4\textsuperscript{th} either died at admission or during follow up.

Yanagi H, et al\textsuperscript{45}, 1998 reported a case of acute mitral regurgitation caused by complete posterior papillary muscle rupture as a complication of acute inferior myocardial infarction. A 64 years old women developed sudden, cardiogenic shock shortly after the onset of acute inferior Myocardial infarction, with posterior papillary muscle totally ruptured.

Moustapha A, et al\textsuperscript{46} 2001 present a case of an Acute Myocardial Infarction presenting solely as rupture of the head of antero-lateral papillary muscle of the mitral valve with an echocardiographic appearance of a mitral valve vegetation. A 61 Year old male Patients presented to the hospital with cardiogenic shock. Transesophageal echocardiography revealed function with the echo-cardiographic appearance of a large vegetation attached to the anterior mitral valve leaflet & severe mitral regurgitation.

ARRHYTHMIAS:

The incidence of Arrhythmias is higher in those patients seen earlier after the onset of symptom. Many serous Arrhythmias
develop before hospitalization even before the patient is monitored\textsuperscript{47}. They include ventricular arrhythmias, Supraventricular arrhythmias, bradyarrhythmias.

*Electrical instability* leads to ventricular premature beats ventricular tachycardia ventricular fibrillation, accelerated idioventricular rhythm & non paroxysmal A-V-junctional tachycardia.

*Pump failure / Excessive sympathetic stimulation* is responsible for sinus tachycardia atrial fibrillation, atrial flutter or paroxysmal supraventricular tachycardia.

*Bradyarrhythmias and conduction disturbances* include sinus bradycardia, junctional escape rhythm, atrio-ventricular block and intra ventricular block.

The conventional definition of ventricular ectopic impulses at rate of 120 or greater. They are of 2 type; *sustained* and *non-sustained* ventricular tachycardia. The sustained ventricular tachycardia lasting for 30 seconds of more or associated with hemodynamic compromise, while non sustained tachycardia lasts for less than 30 seconds.

Prevalence of NSVT varies in relation to timing of Myocardial Infarction, within first 24 hours in pre thrombolytic era, the prevalence of NSVT was 45% which is 75% now in thrombolysed patients\textsuperscript{48}. The prevalence of NSVT during late hospital phase (10-30 days) in prethrombolytic-era was 7%-16% compared to 7-9% in thrombolysed patients.
Their incidence remain fairly constant over the first year of infarction. The most important factor associated with increased occurrence of NSVT is low LVEF and approximately 3/4 of patients with NSVT EF < 40%. Other associated factors are multivessel disease, increased regional wall motion abnormality, inducible ischemia, ventricular aneurysm and abnormal hemodynamic. NSVT detected 10-30 days after acute infarction more than double the risk of subsequent sudden death compared to patients without NSVT. NSVT detected 3 month to 1 years post Myocardial Infarction is also associated with a significant higher mortality rate.

**MUSTT** (Multicentre Unsustained Tachycardia Trial) has shown that the risk of sudden death was reduced by 27% in patients randomized to electrophysiologically guided therapy. Patients randomized to electrophysiologically guided therapy who failed to responded to pharmacologic antiarrhythmic therapy, judged by serial electrophysiologic tests and thereby with implantable cardioverter defibrillators (ICD) experienced a 76% reduction in the risk of sudden death as compared to patients with no antiarrhythmic therapy. A recently published study **MADIT** (Automatic Defibrillator Implantation Trials) has shown that post Myocardial Infarction patients with reduced left ventricular ejection fraction < 0.35 and non suppressible NSVT with procainamide treated with ICD have 54% reduction in mortality compared to amiodarone therapy. A 33% reduction in mortality was also obtained in patients who were on antiarrhythmic therapy and whose VT suppressed by these drugs. Thus does considering results of these trials patients with coronary artery disease, abnormal ejection and symptomatic NSVT are at high risk of sudden death which can be
reduced with EPS guided anti arrhythmic drug therapy and more effectively by ICD\textsuperscript{48}.

The sustained ventricular tachycardia may develop in specialized conduction system distal to bundle of His, in ventricular myocardium, or by an interaction between two. It occur at heart rate of > than 100 beats / min and last for 30 seconds or more associated with hemodynamic compromise\textsuperscript{53,54,55}. Most common underlying heart disease is coronary artery disease accounting for more than 50% of cases followed by cardiomyopathy. Less, common causes include primary electrical disease, MVP, Valvular heart disease, congenital heart disease and miscellaneous causes\textsuperscript{55,56}. Left ventricular hypertrophy and transient artery spasm may also lead to VT. Complex can occur after CABG sustained ventricular tachycardia are more likely to have reduced ejection fraction, slow conduction and ECG-abnormality left ventricle aneurysm, the abnormal signal average ECG, and previous Myocardial Infarction that patients with ventricular fibrillation\textsuperscript{55,55,56}.

Patients with sustained ventricular tachycardia or Ventricular fibrillation in the absence of reversible or transient cause has sudden death 30% and 2 years of sudden death of 50\%\textsuperscript{55,57}.

\textbf{Bobrov VA et al\textsuperscript{58}} studied 85 patients with acute large size myocardial infarction admitted within 12 hours of the condition development. 24 hours, monitoring was instituted. The result obtained showed only 27\% of patients at low risk development of life threatening arrhythmias in all others high grades ventricular extrasystole are recorded including ventricular tachycardia in 50\% of the patients.
Atrial fibrillation is the most common supraventricular arrhythmia in patients with Acute Myocardial Infarction.

Denes P et al⁵⁹ studied 1,211 patients with Acute Myocardial Infarction. Patients with bundle branch block were excluded from the analysis and remaining 1,158 were followed for up to 1 year after infarction out of them 45 patients had a serious arrhythmic event.

Eldar M et al⁶⁰, 1998, had observed that paroxysmal atrial fibrillation is considered a frequent complication of Acute Myocardial Infarction associated with increased in hospital stay and long term mortality rates. The incidence of paroxysmal atrial fibrillation was (8.9% - 9.9%) and after 30 days (25.1%-27.6%) and at 1 year (38.4%-42.5%).

Pizzetti F et al⁶¹, 2001, data derived from GISSI – 3 trial, wherein included 17944 patients within the first 24 hrs. after Acute Myocardial Infarction. Atrial fibrillation was recorded during the hospital stay, and followup visits were planned at 6 weeks & 6 months. Survival of the patients at 4 years was assessed through census offices has done. The incidence of in hospital atrial fibrillation or flutter was 7.8%. Atrial fibrillation was associated with indicators of a worse prognosis (age > 70 years, female sex higher Killip class, previous myocardial infarction treated hypertension, high systolic BP at admission, IDDM, signs or symptoms of heart failure) and with some adverse clinical events (reinfarction, sustained ventricular tachycardia, ventricular fibrillation).
After adjustment for other prognostic factors, atrial fibrillation remained an independent predictor of A increased in hospital mortality 12.6% vs 5%. Long term mortality i.e. 4 years after Acute Myocardial Infarction confirm the persistent negative influence of AF. So conclusion is that atrial fibrillation is an indicator of worse prognosis after Acute Myocardial Infarction in the long term even in un selected population.

Ischemic injury can produce conduction block at any level of the AV or intraventricular node and the bundle of His, producing various grades of atrio-ventricular block.

*Altun A, et al*⁶² 1998 reported after the study of 51 patients with inferior wall Acute Myocardial Infarction that advanced atrioventricular block is a frequent complication in patients in hospital, it occurs concurrently, with other complications and is associated with high mortality.


*Ozdemir K, et al*⁶⁴ 2001, studied 172 patients (141 men & 31 women) between 28 & 84 years of age with acute inferior wall infarction out of 172,25 patients developed left ant hemiblock (LAHB). LAHB development during acute inferior-myocardial infarction can be indicator of left anterior descending lesion.
RECURRENT CHEST DISCOMFORT:

Post infarction recurrent chest discomfort may be due to recurrent chest discomfort, recurrent angina or infarction from non ischemic causes of discomfort that might be caused by infarct expansion, pericarditis, pulmonary embolism and non cardiac conditions. Important diagnostic maneuvers include a repeat physical examination, repeat ECG and assessment of the response to sublingual nitroglycerin, 0.4 mg.

The incidence of post infarction angina without reinfarction is between 20% and 30%. It does not appear to be reduced by thrombolytic therapy but has been reported to be lower in patients who undergo primary PTCA for Acute Myocardial Infarction. Usually extension and reinfarction refer collectively under the more general term, recurrent infarction. Extension of the original zone of necrosis or reinfarction in a separate myocardial zone is difficult to differentiate within the first 24 hours after index event. Beyond the first 24 hours, when serum cardiac marker such as CK-MB have usually returned to the normal range, recurrent infarction may be diagnosed either by re-elevation of the CK-MB above the upper limit of normal and increased by at least 50% of the previous value or the appearance of new Q waves on the ECG. The incidence of this complication of Acute Myocardial Infarction range from about 5% to as a higher as 20% within the first 6 weeks and may be some what higher in patients who have received thrombolytic therapy. Marmor reported that recurrent infarction occurred frequently in obese females and was most common in patients with diabetes mellitus, those with a previous Myocardial Infarction and those with an early peaking CK-MB curve (< 15 hours) but it is not predictable from the
angiographic appearance of the coronary artery early after infarction.

Regardless of whether post infarction angina is persistent or limited, its presence is important because short-term morbidity is higher among such patients, mortality may be increased if the recurrent ischemia is accompanied by ECG changes and hemodynamic compromise recurrent infarction (due in many cases to reocclusion of the infarct related coronary artery) carries serious adverse prognostic information because it is associated with a two to four fold higher rate of in hospital complication (CHF, heart block) and mortality. The mortality rate at 1 to 3 years following the initial infarction is higher in those patients who suffered from recurrent infarction during their index hospitalization. Presumably the higher mortality is related to the larger mass of myocardium whose function become compromised.

PERICARDIAL EFFUSION AND PERICARDITIS:

Pericardial effusion occur in approximately 25% of patients after Myocardial Infarction effusion are more common in patients with anterior wall Myocardial Infarction and with large infarcts and when congestive failure is present. The majority of pericardial effusion that are seen following Acute Myocardial Infarction do not cause hemodynamic compromise. Cardiac tamponade occur usually due to ventricular rupture or hemorrhagic pericarditis.

The reabsorption rate of a post infarction pericardial effusion is slow, with resolution often taking several months. The presence of an effusion does not indicate that pericarditis is present, although
they may occur together, the majority of effusion occur without other evidence of pericarditis.

Pericarditis, when secondary to transmural Acute Myocardial Infarction, may produce pain as early as the first day and as late as 6 weeks after Myocardial Infarction. The pain of pericarditis may be confused with post infarction angina, recurrent infarction or both. An important distinguishing feature is the radiation of the pain to either `trapezius ridge, a finding that is nearly pathognomonic of pericarditis and rarely seen with ischemic discomfort. Transmural Myocardial Infarction by definition, extends to the epicardial surface and is responsible for local pericardial inflammation. An acute fibrinous pericarditis (*Pericarditis episteno cardica*) occurs most commonly after transmural infarction. Although transient pericardial friction rubs are relatively common among patients with transmural infarction within the first 48 hrs, pericardial friction rub appears to be correlated with a larger infarct and greater hemodynamic compromise. The discomfort of pericarditis usually becomes worse during a deep inspiration, but it may be relieved or diminished when the patients sits up and leans forward. Although anticoagulation clearly increases the risk for hemorrhagic pericarditis early after Myocardial Infarction, this complication has not been reported with sufficient frequency, but the detection of a pericardial effusion on echo is usually an indication for discontinuation of anti-co-agulation.

Dressler Syndrome also known as the post myocardial infarction syndrome, usually occurs 1 to 8 weeks after infarction. Dressler cited an incidence of 3 to 4% of all Acute Myocardial Infarction patients in 1957 but the incidence has decreased dramatically since that time. Clinical features of syndrome are
malaise, fever, pericardial discomfort, leukocytosis, an elevated sedimentation rate and a pericardial localized fibrinous pericarditis containing polymorphonuclear leukocytes. The detection of antibodies to cardiac tissue has raised the notion of an immunopathological process.

**Cerebrovascular accidents**: clinically apparent thromboembolism complicates Acute Myocardial Infarction in 10% of cases, but in 20% of patients it is clinically silent. Thromboembolism is considered to be least an important contributing cause of deaths in 25% of patients with Acute Myocardial Infarction who die after admission to the hospital. Arterial emboli originate from left ventricle mural thrombi, while most pulmonary emboli arise in the leg veins.

Thromboembolism typically occurs in association with large infarct (especially anterior) CHF and a left ventricle thrombus detected by echocardiography 2-D echocardiography reveals left ventricle thrombi in about 1/3 of patients with anterior wall infarction but in few patients with inferior wall infarction. Arterial embolism often presents as a major complication, such as hemiparesis when the cerebral circulation is involved in hypertension if the renal circulation is compromised. The incidence of embolic complication appears to be markedly lowered by such therapy.

Intracranial hemorrhage is an uncommon but very dangerous complication in patients receiving thrombolytic therapy for Acute Myocardial Infarction. Neuro surgical evacuation is often an available treatment option. GUSTO-1 trial randomly assigned 41,021 patients with Acute Myocardial Infarction to 1 to 4 thrombolytic strategic in
1081 hospitals in 15 countries. A total of 268 patients (0.65%) had an intra cranial hemorrhage. **Mahaffey KW et al** assessed difference in clinical characteristic, neuro imaging features, glasgow coma scale scores, functional status (disable : moderate or severe deficit, not disable; no or minor deficit) a 30 days mortality rate between the 46 patients who underwent neuro surgical evacuation and the 222 patients who did not. Mortality rate at 30 days for all patients with intracranial hemorrhage was 60%, an additional 27% were disabled. Evacuation was associated with significantly higher 30-day survival (65% versus 35%) and a improved functional status (non disabling stroke : 20% versus 12%) conclusion is that although intracranial hemorrhage is uncommon after thrombolysis for Acute Myocardial Infarction, 87% of patients die or have disabling stroke.
**Electrocardiogram**

It is electrical device which records the changing potentials of the electrical field imparted by heart. It is extremely useful clinical laboratory tool and it the only practical means of recording the electrical behaviour of the heart so ECG serves as a gold standard for the diagnosis of arrhythmias.

The electrocardiographic recording paper is divided into small and large squares. The small squares are 1mm square. The large squares are 5mm squares. The square facilitate the measurement of (i) time parameters (horizontal measurement) and (ii) Deflexion amplitude (vertical measurement). Conventionally electrocardiogram is nearly always recorded at a paper speed of 25mm per second. At this paper speed 5 large squares represent one second and one small square represents 0.04 second. Most graph papers used for the recording of ECG have every 15th large square[ 3 sec. Period ] marked by a vertical line on the upper border.

**The conventional electrocardiographic leads:**

In clinical practice, however there are 12 conventional leads, divided into 2 groups.

1. **The Frontal plane leads** – These are oriented in frontal or coronal plane of the body I, II, III, aVR, aVL and aVF

2. **The horizontal plane leads** – These are oriented in the transverse or horizontal plane of the body and are formed by the precordial leads V1 to V6.
The frontal plane leads:

Standard lead I – This lead is derived from the placement of negative electrode on the right arm and the positive electrode on the left arm.

Standard lead II – This lead is derived from the placement of the negative electrode on the right arm and the positive electrode on the left foot.

Standard lead III – This lead is derived from the placement of the negative electrode on the left arm and the positive electrode on the left foot.

The lead axes of these 3 leads form a triangle and as the electrodes of these leads are regarded as equidistant from the heart, the lead axes too may be considered to be equidistant from the heart. These lead axes thus form an equilateral triangle with the heart at the centre – Einthoven's triangle.

The horizontal plane leads:

Lead $V_1$ - Placed over the 4th intercostal space immediately to the right of sternum.

Lead $V_2$ - Placed over the 4th intercostal space immediately to the left of sternum.

Lead $V_4$ - is placed over the 5th intercostal space in the mid clavicular line.

Lead $V_3$ - is placed on the chest exactly midway between the lead $V_2$ and lead $V_4$ electrode position.
Lead $V^6$ - is placed at the same horizontal level as lead $V_4$ but on the anterior axillary line.

Lead $V^6$ - is placed at the same horizontal level as lead $V_4$ and $V_5$ but on the mid axillary line.

Orientation of the conventional electrocardiographic leads:

- Standard lead II, III and AVF – oriented to the inferior surface of the heart.

- Standard lead I, AVL tend to be oriented to the high or superior left lateral wall.

- Lead aVR is oriented to the cavity of the heart.

- Lead $V_2$ also tends to be oriented towards the cavity of the heart.

- Leads $V_1$ to $V_6$ are oriented towards the anterior wall of the heart. These may be arbitrarily subdivided into:

  (i) Antero – septal leads – $V_1$ to $V_4$

  (ii) Apical or lateral leads – $V_5$ to $V_6$

  (iii) Lateral leads – I & AVL

- Leads $V_1$ & $V_2$ tend to be oriented to the right ventricle

- Leads $V_4$ & $V_5$ tends to be oriented to the left ventricle

- There is no lead which is oriented directly to the posterior wall of the heart.
Genesis of the normal ECG:

P wave = due to atrial depolarization

QRS complex = due to ventricular depolarization

T wave and 'U' wave = due to ventricular depolarization 'U' wave
best seen in leads V2 to V4

Echocardiography

The term echocardiography refer to a group of tests that utilize ultrasound to examine the heart and record information in the form of echoes i.e. reflected sonic waves. The sonic frequency used for echocardiography ranges from 1 to 10 million cycles/second or 1 to 10 mega Hertz (MHz) while in children they are usually higher ranging from 3 to 10 MHz. Whereas a barrel chested, emphysematus patients needs a 1.6 MHz transducer. The resolution of the recording which is the ability to distinguish 2 objects that are spatially close together, varies directly with the frequency and inversely with the wavelength. Waves passes radially through liquid, such as blood or pericardial fluid and these are displayed as black on 2 dimensional image. When ultrasound is reflected off more solid structures, such as the myocardium and valves, these is gray scale display. Structure such as calcium produce intense acoustic reflection and are displayed as bright white on 2D-image.

M-mode: Previously M-mode (time motion-mode) echo cardiography was used. M-mode echocardiography provides information about –

- fractional shortening
- ejection fraction
- septal and wall thickness and
- left ventricle mass

The M-mode technique is limited that it provides only a one dimensional view of the heart.

2D Echocardiography: It has allowed cardiac structure to be visualised in a real time fashion with the help of this we can now assess intra cardiac lessons observe (i) contractility & (ii) valvular function.

Doppler echocardiography: It utilizes ultrasound to record blood flow within the cardiovascular system.

Echocardiography can be performed in 2 ways e.g.

(i) Transthoracic  
(ii) Transesophageal

Transthoracic: Echocardiogram, the imaging is performed with a hand held transducer placed directly on the chest wall.

Transesophageal: Echocardiogram may be performed in which an ultrasound transducer is mounted on the tip of an endoscope placed in the esophagus and directed towards the cardiac structure, so that high resolution images of the posterior structures are obtained.

Method

The imaging is performed from multiple acoustic windows with different transducer location so that the entire heart and great
vessels can be displayed in real time and in various 2 dimensional planes. The patients are generally examined in the supine or left lateral semidecubitus position. Cardiac window is usually found between 3rd to 5th intercostal space slightly to the left of the sternal border.

**Views**: Standard cross sectional imaging planes are as following

1. **Parasternal**
   
   (a) *Long axis-*
   
   - Left heart : aortic valve
   - Mitral valve & left ventricle.
   - Right ventricular inflow tract
   - Right ventricular out flow tract
   - Main pulmonary actery
   - Cardiac apex

   (b) *Short axis-*
   
   - Aortic valve & left atrium
   - Left ventricle (Mitral valve level)
   - Left ventricle (papillary muscle level)
   - Left ventricle apex

2. **Apical**
   
   - 4 chamber
   - 5 chamber
   - 2 chamber
   - Long axis (left ventricle)

3. **Sub costal**
   
   - Long axis of the heart
   - Long axis of the right ventricular out flow tract.

4. **Suprasternal**
   
   - Long axis of the aortic arch
   - Short axis of the aortic arch
**Indication of Echo**: In coronary artery diseases are

(1) Diagnosis of coronary artery disease  
(2) Estimation of infarct size  
(3) In reperfusion therapy  
(4) In detection of complications  
(5) Management of hemodynamically compromise patients  
(6) Determination of prognosis and post myocardial risk stratification  
(7) Determination of myocardial viability.

**Advantages of echo**:  
(1) It is non invasive & cost effective  
(2) No radiation involved  
(3) Easy management  
(4) Early diagnosis of myocardial infarction can be made even before enzyme level rises  
(5) Associated diseases can be detected  
(6) Detection of complications  
(7) Prognosis & treatment can be selected.