CASE ANALYSIS STUDY OF POST - MYOCARDIAL INFARCTION CARDIAC FAILURE

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CERTIFICATE

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DECLARATION

I solemnly declare that the dissertation entitled **"CASE ANALYSIS STUDY OF POST - MYOCARDIAL INFARCTION CARDIAC FAILURE"** is done by me at Madras Medical College and Hospital, during 2003 - 2006 under the guidance and supervision of **Prof.K.CHANDRA, M.D.** This dissertation is submitted to The Tamil Nadu Dr.M.G.R. Medical University towards the partial fulfilment of requirements for the award of **M.D. DEGREE IN GENERAL MEDICINE (BRANCH I)**.

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INTRODUCTION

POST MYOCARDIAL INFARCTION - CARDIAC FAILURE

Post infarction cardiac failure is one among the common complication of acute myocardial infarction. It is clinically observed when the contractile dysfunction of the heart, resulting from Acute myocardial infarction exceeds more than 25 to 40% of left ventricular diameter, barring the limitation observed as compensation mechanism by means of increased adrenergic activity and increased Left ventricular End-diastolic volume; when these compensatory mechanisms are overwhelmed, the increased LVEDV will result in increased myocardial wall tension and augment the myocardial oxygen demand, predisposing further augmentation of increased left ventricular end diastolic pressure and pulmonary venous congestion. For individuals free of Heart failure at age to 40 years, the remaining life time risk for developing heart failure is 21% for men and 20.3% for women[1].

If mechanical complications are associated in Acute myocardial infarction such as Papillary muscle dysfunction, Mitral regurgitation, Ventricular septal rupture, further increase in left ventricular volume will result in further increase in wall tension and oxygen demand and pulmonary venous congestion and consequent left heart failure. Percentage to changes in norepinephrine and BNP over 12 months are independently associated with corresponding changes in morbidity and mortality[2].

The symptoms of post - myocardial infarction cardaic failure are varied. It is recognized in the usual way by the advent of dysponea, fine basal rales, orthopnea, gallop rhythm, distended neck veins, hepatic engorgement, positive abdomino - jugular reflux and in more advanced cases peripheral edema. In severe cases they may present as expectoration of frothy sputum and central cyanosis. Rarely it is diagnosed with the help of X-ray chest with slight enlargement of cardiac stilhoutte and alveolar infiltrate of pulmonary edema, in the absence of clinical features.

In general, male gender, the presence of coronary artery disease as the etiology of heart failure[3], the presence of an audible S_3 or elevated JVP[4], low pulse and systolic arterial pressures, a high NYHA classes and reduced exercise capacity[5], have been shown to be associated with increased mortality.

CLINICAL SUBSETS IN POST - MI CARDIAC FAILURE - NYHA CLASSIFICATION

Class	Features	Mortality
Class I	No Signs of CCF	-
Class II	Basal Rales	40 - 50%
Class III	Acute Pulm. Edema	30 - 40%
Class IV	Pul.edema, cardiogenic Shock	5 - 10%

Anterior wall infarction, especially Q wave infarction, as first episode to coronary heart disease are propensed to go for congestive cardiac failure. Infarct expansion, larger areas infarction, non collateral coronary arteries, are respective reasons attributed.

Inferior wall infarction sometimes present with congestive heart failure in which case, the following may contribute to it.

1. Associated posterior wall / Anterior wall infarction.
2. Persistent and prolonged peri-infarction zone ischemic ventricular dysfunction (Stunning).
3. Uncontrolled systemic complication such as SHT, NIDDM, COPD, Anemia etc.
4. Mechanical complications such as Papillary muscle dysfunction, Mitral regurgitation, true and false LV aneurysms, Ventricular septal rupture and etc.

COMMONEST CAUSES OF POST - MI CARDIAC FAILURE

1. LV Contractile dysfunction
2. LV Diastolic dysfunction
3. Right ventricular dysfunction
4. Acute mitral regurgitation
5. Ventricular septal Rupture

6. Cardiac free - wall rupture

7. Myocardial stunning

8. Hibernating myocardium

9. Stiffheart syndrome

10. Post-infarction ischemic cardiomyopathy

11. Ventricular aneurysms

12. Co-existing illness

13. Iatrogenesis

14. Pseudo heart failure.

AIM OF THE STUDY

It is a prospective study of 100 cases of post - myocardial infarction - cardiac failure analyzing various factors like

1. Incidence
    a. Age
    b. Sex
2. Influences
    a. No. of infarction
    b. Associated systemic illness
    c. Dietary Habits
    d. Occupational Stress
    e. Body mass index
    f. Substance abuse like smoking, alochol and drugs on clinical presentations, with reference to history, symptomatology, electrocardiogram and 2-Dimentional Echocardiogram and X-ray chest in selective cases.

REVIEW OF LITERATURE

ANATOMY OF CORONARY CIRCULATION

The coronary arterial system consists of 2 coronary arteries, viz. right coronary artery and left coronary artery.

RIGHT CORONARY ARTERY

Arising from (R) coronary sinus it courses through (R) side of the AV groove where it gives rise to branches to Right atrium and right ventricle and continues as posterior descending artery in the posterior inter ventricular groove and supply to the posterior - inter ventricular septum and posterior left ventricular wall. Besides, whole of right Atrium, posterior inter ventricular septum, posterior left ventricular wall, it supplies the whole of conduction system (60% of cases to SA node). Except (L) Bundle of His, the whole of right ventricle except a part adjoining anterior inter ventricular groove.

LEFT CORONARY ARTERY

Larger than RCA - originates from the left posterior sinus, runs left, and anterior to emerge infront of left auricle where it gives anterior intra ventricular branch (Which runs its groove) and it continues as left circumflex artery winds the left border of the Heart, runs close to the posterior I.V. groove, terminates with branches of right coronary artery. It supplies the whole of left atrium, left ventricle (Except the part adjoining the posterior IV groove), a smaller part of

the right ventricle adjoining the anterior I.V. groove, anterior part of the I.V. septum apart from the Left branch of the AV bundle.

Adding to this, the heart is also nurtured by the intercoronary anastomosis, extracardiac anastomosis, retrograde flow of veins (venae cordis minimae).

MYOCARDIUM AND TERRITORY OF CORONARY ARTERIES

Right Cor Artery	Left Circumflux	Left anterior Descending
Inferior Wall	High Lateral Wall	Antero Septal Wall
Sinus Node 60%	True Posterior Wall	Antero Lateral Wall
Conduction System (Except LBB)	(L) Atrium	Apex
Right Atrium		
Right Ventricle		

PHYSIOLOGY OF CONTRACTILITY OF CARDIC MUSCLE

Myofibril is the functional unit of the cardiac contractile apparatus. When it receives the action potential, Ca^{++} ions enters the cells through slow channels inactivate another protein - Treponin `C' which inturn activate actin / myosin filaments in cross bridges effect ATP release and accomplish the

contraction, quantum of myofibril contraction - reflects force of contraction (Inotrophic State of the Myocardium).

This inotrophic state depends on the load or sketch which the muscle has (preload) and the load against which it contracts (After load). In non failing heart, prestretch improves the force - velocity relations that is end diastolic volume and stroke volume relations whereas failing heart could not maintain this relation. This is called Frank starling law (or) relationship of heart.

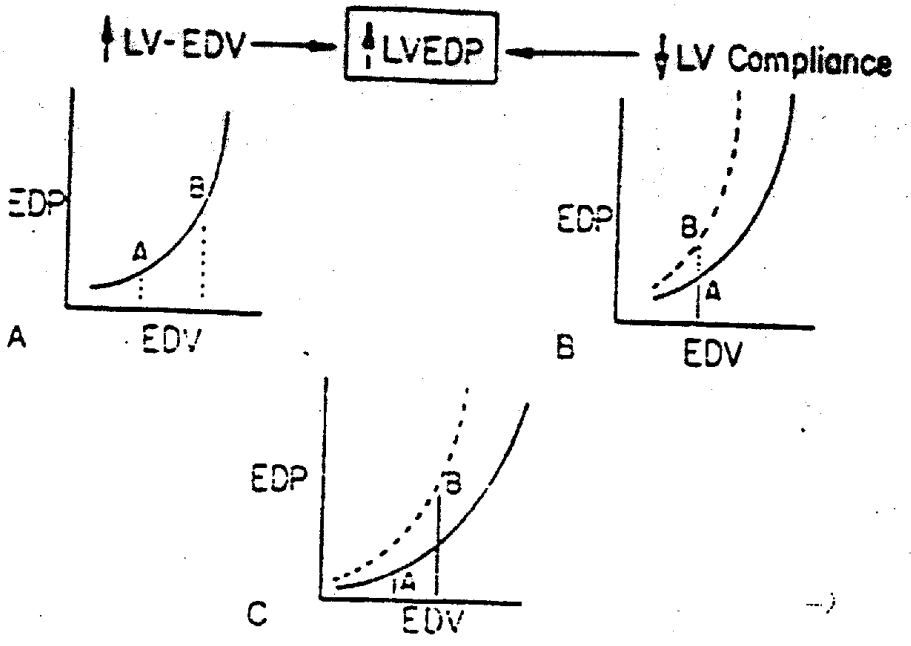
LAPLACE LAW

The pressure [P] within the sphere is proportional to wall stress (after load) [T] and inversely proportional to radius [r] of the chamber. When heart enlarges beyond the point at which starting law conferred an advantage, the increase in radius will decrease end diastolic pressure, ejection fraction, stroke volume and cardiac output ($P \alpha T/r$).

Both these laws are functional significance and operational in acute / chronic compensated coronary heart disease. As and when sudden reduction in cardiac output by infarction or ischemia reflexes sympatho - Adrenergic release and augment the preload (constriction of capacitance vessels) and after load (Constriction of resistance vessels).

MECHANISM OF INCREASED PCW

PCW ~ LVEDP



Mechnisms contributing to elevation of left ventricular EDP and consequently the pulmonary capillary wedge pressure in pressure - volume plots. Patterns of left ventricular regional contractile dysfunction and their influence on end diastolic volume, end systolic volume, stroke volume and ejection fraction relationship between LV ejection fraction and survival.

PATHOPHYSIOLOGY AND EVOLUTION OF PUMP FAILURE IN ACUTE MYOCARDIAL INFARCTION

The clinical syndrome, of pump failure is a complex, interplay of systolic and diastolic dysfunction and / or superimposed mechanical complications. The pump failure is precipitated worsened and perpetuated by additional factors such as sustained or recurrent supra ventricular or ventricular arrhythemias or persistent severe brady arrhythemias, potent negative ionotrophic agents and relative or absolute hypovolumeic states. Heart failure and left ventricular dysfunction are treatable but require a multi disciplinary inte-grated net work approach[6].

CONTRACTILE DYSFUNCTION

Severe reduction or total cessation of coronary blood flow results in loss of regional contractile function (Akinesia) or reduction (hypokinesia) or systolic expansion (dyskinesia) of the hypovascular segments. This altered contractility depends on the extend of the myocardium - damaged leads on to net changes in global ejection fraction, end systolic volume, end diastolic volume as depicted in the Figure No.1.

If a sufficient quantity of myocardium undergoes ischemic injury, left ventricular pump function becomes depressed; cardiac output, stroke volume, blood pressure and peak dp / dt are reduced and end systolic volume is increased[7].

FIG.1

PHYSIOLOGY OF DIASTOLIC DYSFUNCTION

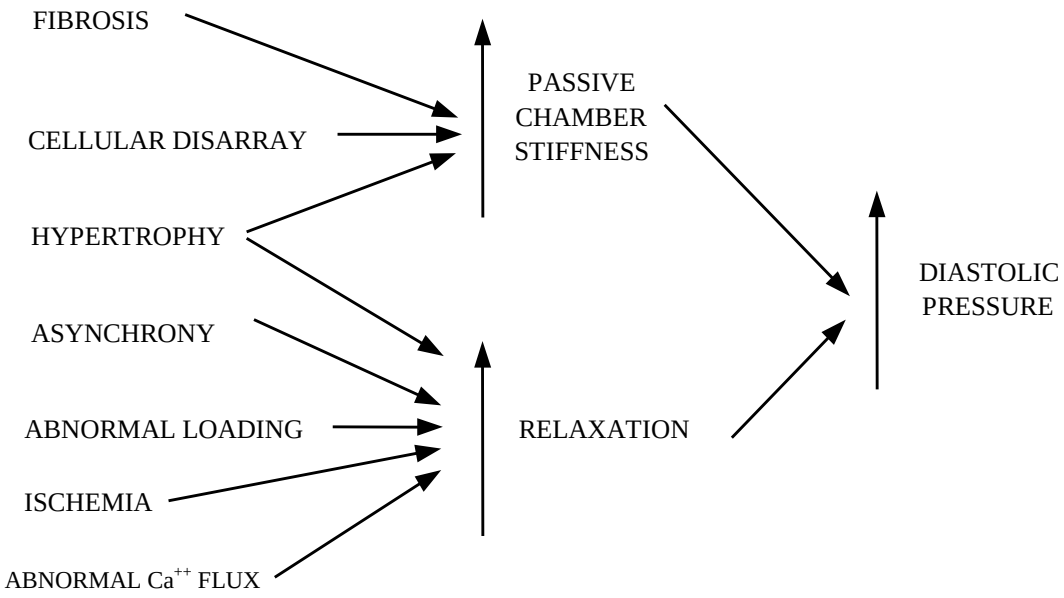


FIG.2

SHORT TERM RESPONSES TO IMPAIRED CARDIAC PERFORMANCE

Response	Short - Term Effects	Long - Term Effects
Salt and Water retention	Augments preload	Causes pulmonary congestion, anasarca
Vasoconstriction	Maintains blood pressure for perfusion of vital organs (brain, heart)	Excerbates pump dysfunction (after load mismatch); increase cardiac energy expenditure
Sympathetic stimulation	Increase Heart Rate and ejection	Increases energy expenditure
Sympathetic desensitization	-	Spares energy
Hypertrophy	Unloads individuals muscle fibers	Leads to deterioration and death of cardiac cells; cardiomyopathy of overload
Capillary deficit	-	Leads to energy starvation
Mitochondrial deficit	Increase in density helps meet energy demands	Decrease in density leads to energy starvation
Appearance of slow myosin	-	Increases force, decreases shortening, velocity and contractility; is energy sparing.
Prolonged action potential	-	Increases contractility and energy expanditure.
Decreased density of sarcoplasmic reticulum calcium-pump sites	-	Slows relaxation may be energy sparing.
Increased collagen	May reduce dilatation	Impairs relaxation

FIG.3

THE LOW OUTPUT STATE CAN ACCELERATE THE RATE OF CELL DEATH IN THE FAILING HEART BY STIMULATING THE RENIN-ANGIOTENSIN AND SYMPATHO - ADRENERGIC SYSTEMS WHICH ACT ON HEART AND CIRCULATION AND SEQUENTIAL CHANGES

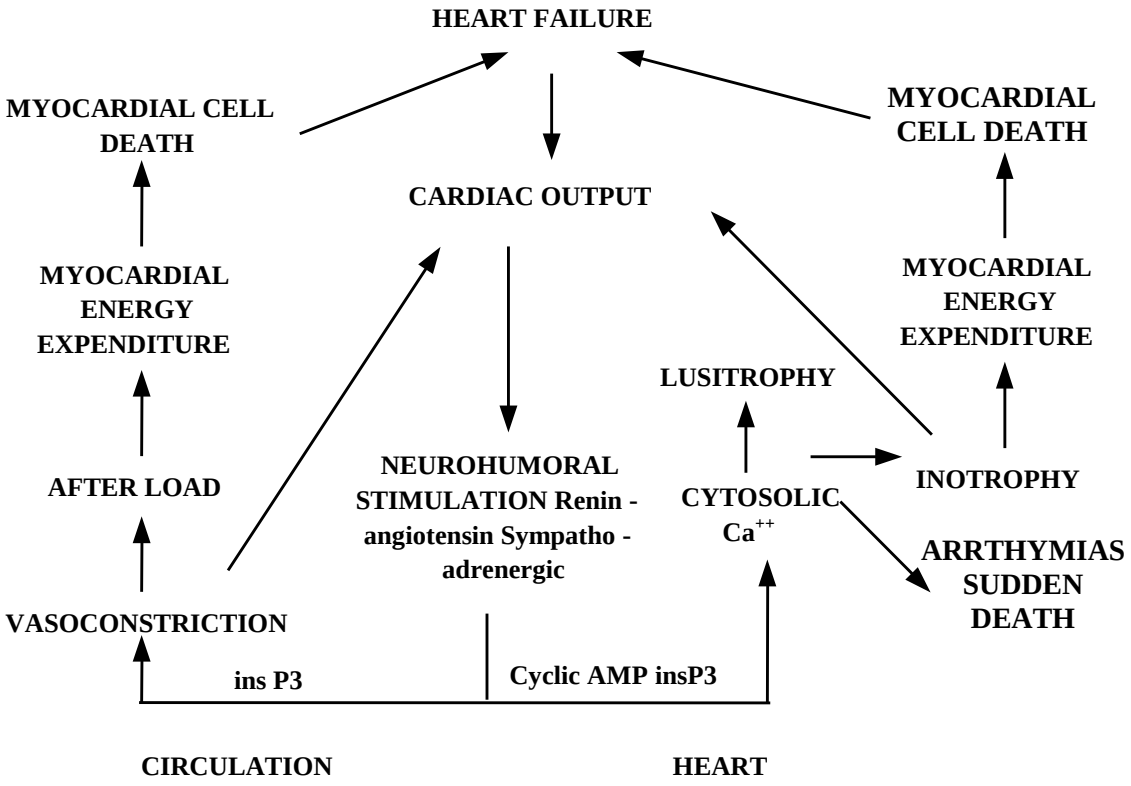
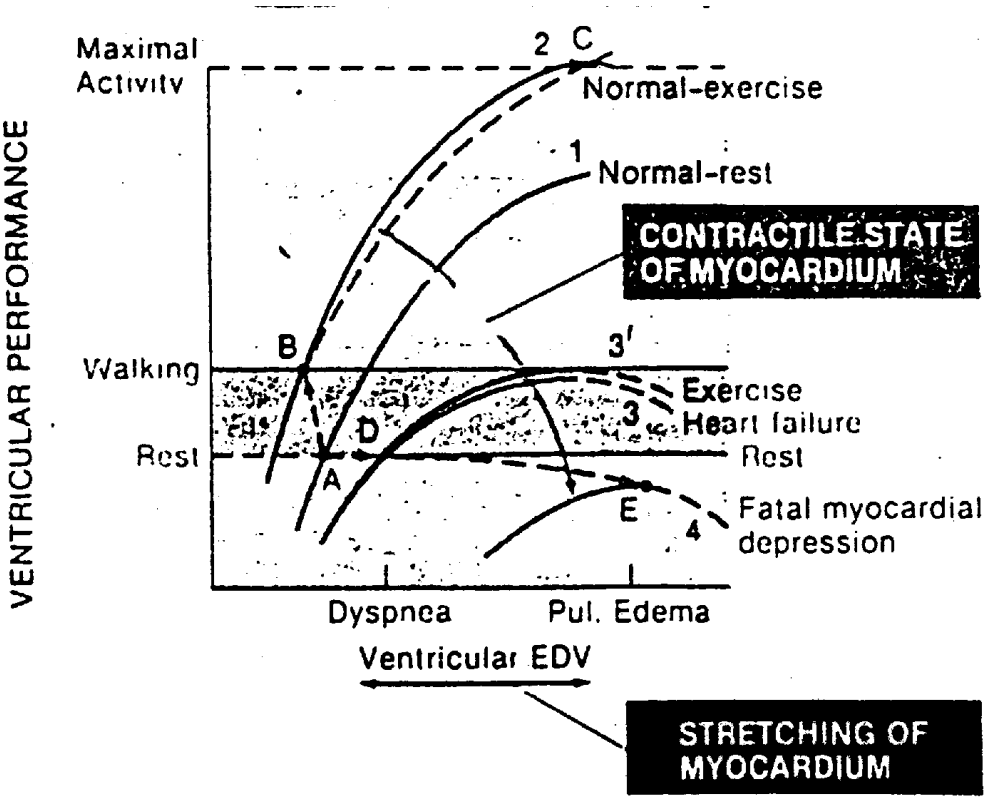


DIAGRAM SHOWING THE INTER RELATIONSHIP OF INFLUENCES ON VENTRICULAR EDV THROUGH STRETCH OF MYOCARDIUM AND THE CONTRACTILE STATE OF MYOCARDIUM AT REST - WALKING MAXIMAL ACTIVITY AND HEART FAILURE, FATAL MYOCARDIAL DEPRESSION.



When the myocardial infarction involves less than 10% of left ventricular mass the resultant reduction of stroke volume cardiac output, ejection fraction is negligible where as if it involved more than 40% gross reduction of the above indices results in fatal cardiogenic shock and acute left ventricular failure (Killip Class IV).

DIASTOLIC DYSFUNCTION

Following acute MI, abnormalities of diastolic function occurs as a result of increase in preload mediated through sympatho adenergic stimulation and renal retention of salt and water. This augmented preload, effect changes in myocardial fibre by the appearance of slow myosin, prolonged action potential, decreased density of sarcoplasmic retriculum calcium pump site, upward regulation of mitochondrial density and collagen in muscle tissues, facilitate the stretch and the force of contraction of the myofibril and in the absence of mitral valve disease, this result in elevated end diastolic volume, and pressure as in Fig.3. Pulmonary Capillary Wedge Pressure approxiamtes the left ventricular mean filling (Diastolic) pressure and elevated end diastolic pressure results in elevated PCWP. This permutations are adoptive and preventive of congestive cardic failure in short term but are maladoptive and deliterious that port ends in relentless progression to CCF as depicted in Figure.2. The degree of ventricular dilatation which depends closely on infarct size, patency of the infarct related artery[8].

The above changes at cell level along with disruption of connective tissue frame work and consequent slippage of myofibrils result in stretching, lengthening and thinning of the segment of the transmural necrosis result in increased LV - EDV.

These anatomical alterations bear parallel reductions in the visco - elastic properties of the myocardial avascular zones due to cellular infiltration and interstitial edema in Acute phase, and healing with fibrosis in sub acute and chronic phase. This all leads to reduction in LV compliance; Bronheim's effect sometimes add to the reduction in LV compliance by pushing the intraventricular septum towards the left ventricle, in case of RV Ischemia and infarction of patients with inferior - wall infarction. The degree to which end systolic volume increases is perhaps the most powerful predictor of mortality following STEMI[9].

Four abnormal contraction patterns develop in sequence:

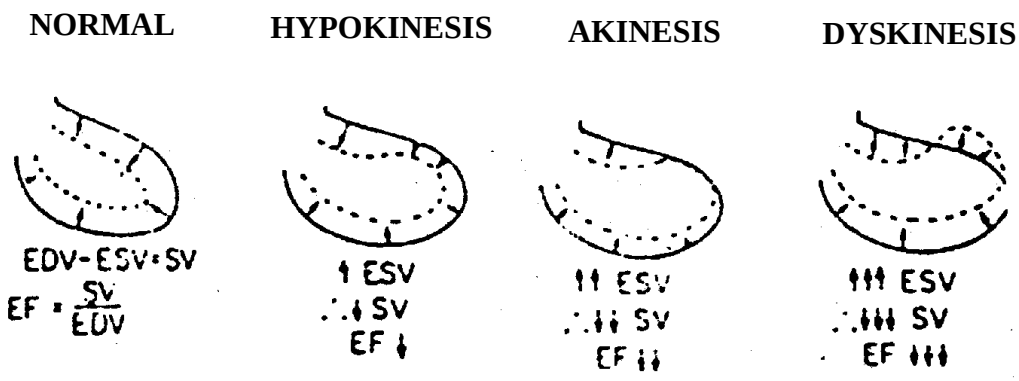
1. Dyssynchrony - dissociation in the time course of contraction of adjacent segments.

2. Hypokinesis - reduction in the extent of shortening.

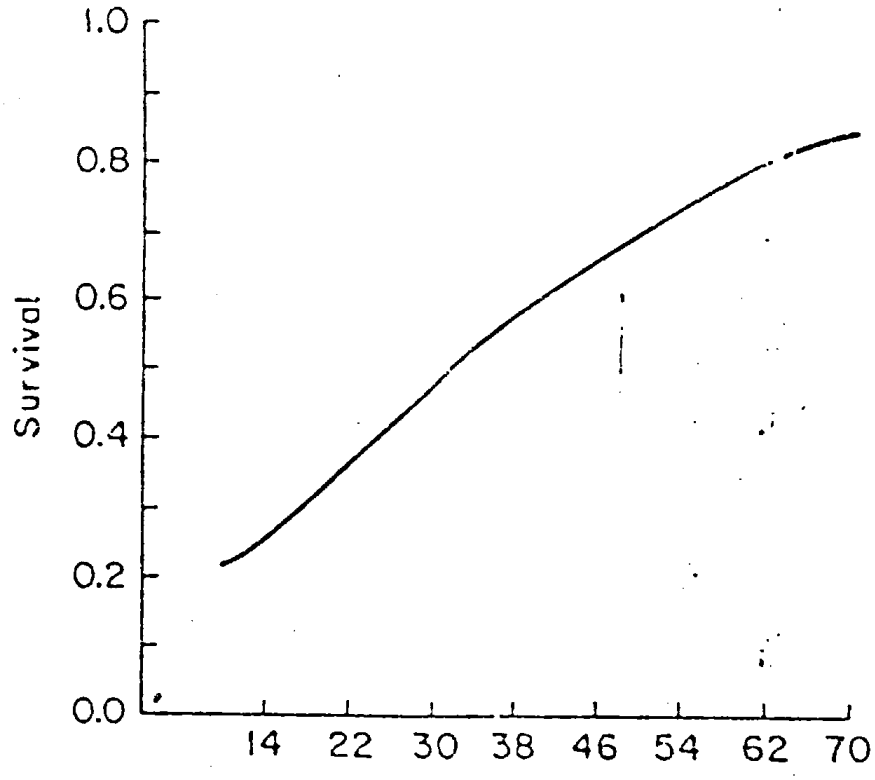
3. Akinesis - cessation of shortening.

4. Dyskinesis - paradoxial expansion and systolic bulging[10].

PATTERNS OF LEFT VENTRICULAR REGIONAL CONTRACTILE DYSFUNCTION AND THEIR INFLUENCE ON END DIASTOLIC VOLUME, END SYSTOLIC VOLUME, STROKE VOLUME AND EJECTION FRACTION.



RELATIONSHIP BETWEEN LV EJECTION FRACTION AND SURVIVAL



AETIOLOGY OF POST INFARCTION CARDIAC FAILURE

1. Contractile dysfunction
2. Diastolic dysfunction
3. Myocardial stunning
4. Hibernating myocardium
5. Stiff heart syndrome
6. Post infarction ischemic cardiomyopathy
7. Right ventricular infarction

MECHANICAL COMPLICATIONS

1. Acute mitral regurgitation
2. Ventricular septal rupture
3. True and false aneurysms
4. Cardiac free wall rupture

OTHER CAUSES

1. Co-existing illness like arrthymias
2. Iatrogenesis

I. LEFT VENTRICULAR DYSFUNCTION RESULTING FROM MYOCARDIAL INFARCTION

As earlier pointed out in the introduction, victims of infarction with more than 25 - 40% of the functioning left ventricular muscle mass, manage to reach the hospital and present with LVF with or without, power failure. This is either due to old infarction or due to extending old MI, reinfarction, fresh cases of infarct over old one.

The low output state in Acute infarction extend the infarct zone through:

a. Sympatho adrenergic release (after load).

b. Increase in Heart Rate (Increased Oxygen demand in compromised state).

c. Decrease in the stroke volume (Decreased Coronary Perfusion).

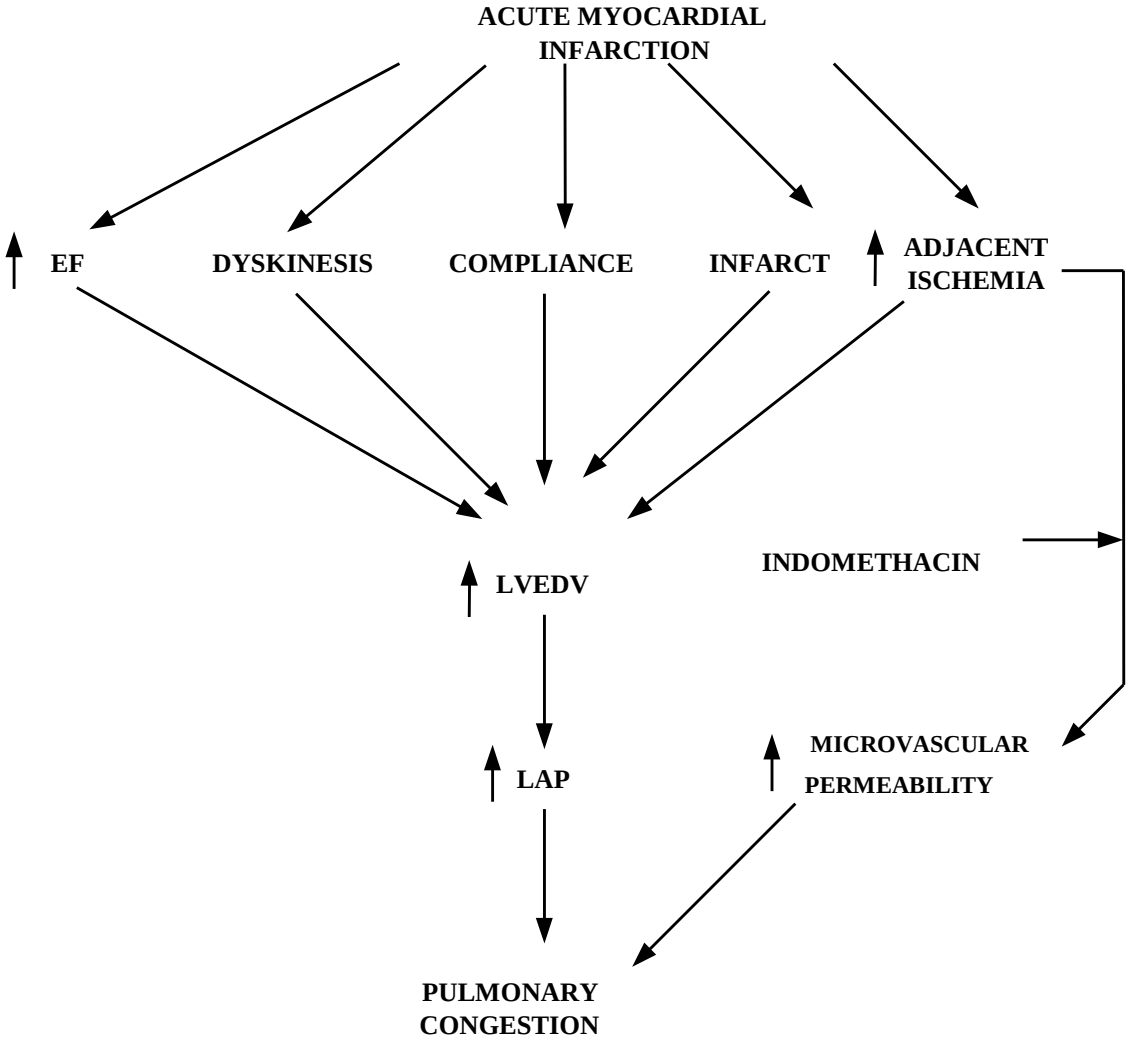
d. Reduction in Peripheral Circulation causes lactic acidemia (Depress myocardial function).

e. Vicious cycle of LVF → Pump Failure → Decreases Coronary Perfusion → Aggravation of LVF.

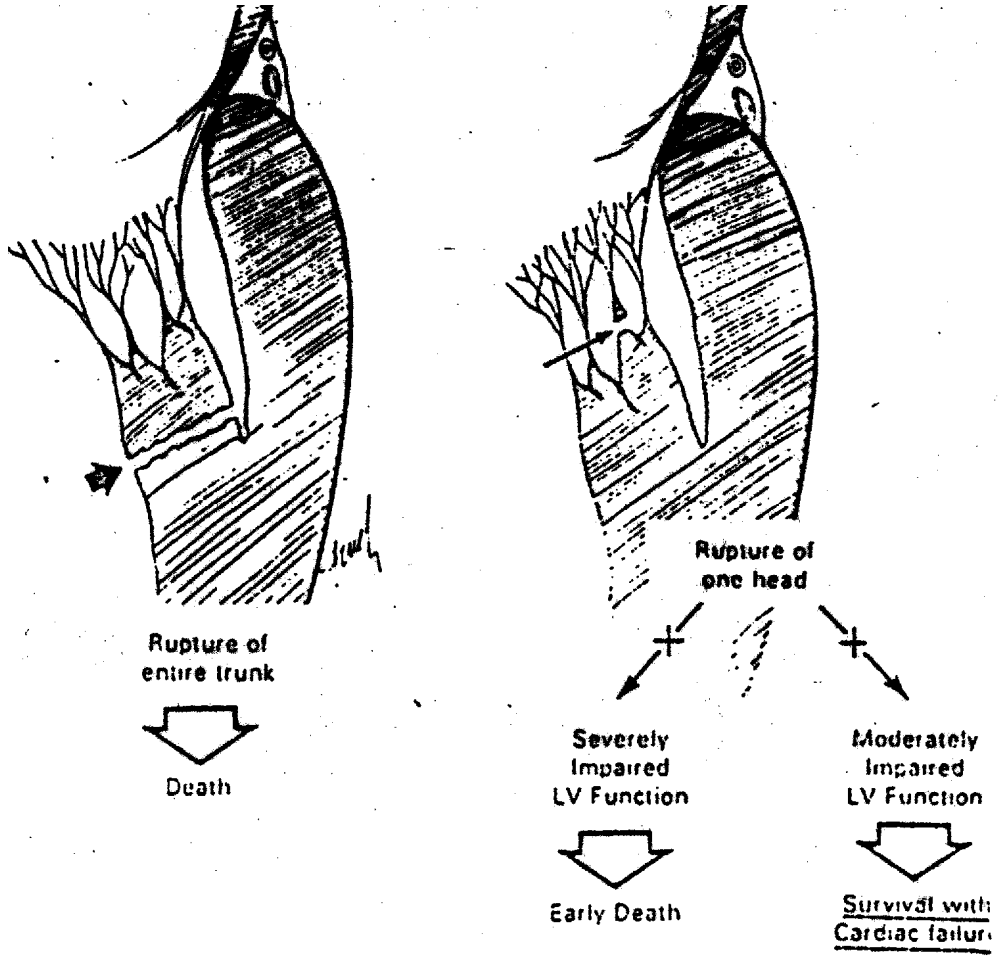
The features, mortality fall within the purview of Killip classification as indicated in the introduction.

FIG.4

MECHANISMS OF PULMONARY CONGESTION IN ACUTE MYOCARDIAL INFARCTION



COMPARISON OF EFFECTS OF RUPTURE OF TRUNK (OR) BELLY OF PAPILLARY MUSCLE OF LEFT VENTRICLE (LEFT) AND RUPTURE OF ONE HEAD OF PAPILLARY MUSCLE (RIGHT)



The diagnosis of left ventricular dysfunction in post MI patient rests on rest / exercise / pharmaco dynamic assessment of end systolic volume, ejection fraction (70% - mild, 40-70% moderate, < 40%- severe, LV failure), with the advent of stress echo, Doppler studies, radionucleotide, ventriculography, perfusion scan, positran emission tomography. It is now possible to have serial studies of the above and assess regional viability metabolic and functional ability of the myocardium. In patients with dilated cardiomyopathy, a significant reduction in myocardial blood flow as assessed by positran emission tomography was associated with an increased risk of death or progression of heart failure[11].

Treatment

Salt restricted diet, diuretics, ACE inhibitors, adequate oxygen through nasal cannula, vasodilator therapy are sheet anchor in the management of Acute post MI - LVF; patient with cardiac output <1.8 L / minute or pulmonary capillary wedge pressure > 20 mm Hg should be treated with positive ionotrophic agents. Since dopamine increases PCWP - it is reserved in case of severe low BP, decreased PCWP with failure. Digitalis is useful in atrial fibrillation complicating post infarction LVF. In all other cases dobtuamie 3.5 µg/min/ Kg shall be administered.

All patients are subjected to Echo to find out early mechanical complication. If no mechanical cause, LVF may be due to (1) Extensive reversible ischemic stunned myocardium, (2) irreversible myocardial necrosis; though they are difficult to diagnose in clinical grounds. Doppler study can

identify them and benefit them with mechanical or pharmacologic reperfusion in the former case.

Difficult differentiation between stunned and necrotic myocardium who present with Killip III or Killip IV who are resistant to medical treatment, initial stabilization can be obtained using Intra Aortic balloon pump pulsation for later contemplation of surgical revascularisation such as direct emergent coronary angioplasty (single vessel disease limited CAHD) or Coronary artery bypass Graft Surgery (Severe CAHD, two vessel or three vessel disease).

Long term mortality of left ventricular dysfunction

It depends on

1. Total extend of irreversible LV Damage.
2. The amount of myocardium at risk of damage.
3. The severity of ventricular arrthymitas 10-14 days following infarction.

Their effects are multiplicative and 2 year mortality if any of adverse feature above present is 50% and if none is present it is <3%.

Prognosis

1. The LV ejection fraction at rest is the valuable prognostic sign. Those with <40% of LV - Ejection fraction, Captoprile improves the long term survival and reduces the incidences of re-infarction and Heart - failure.

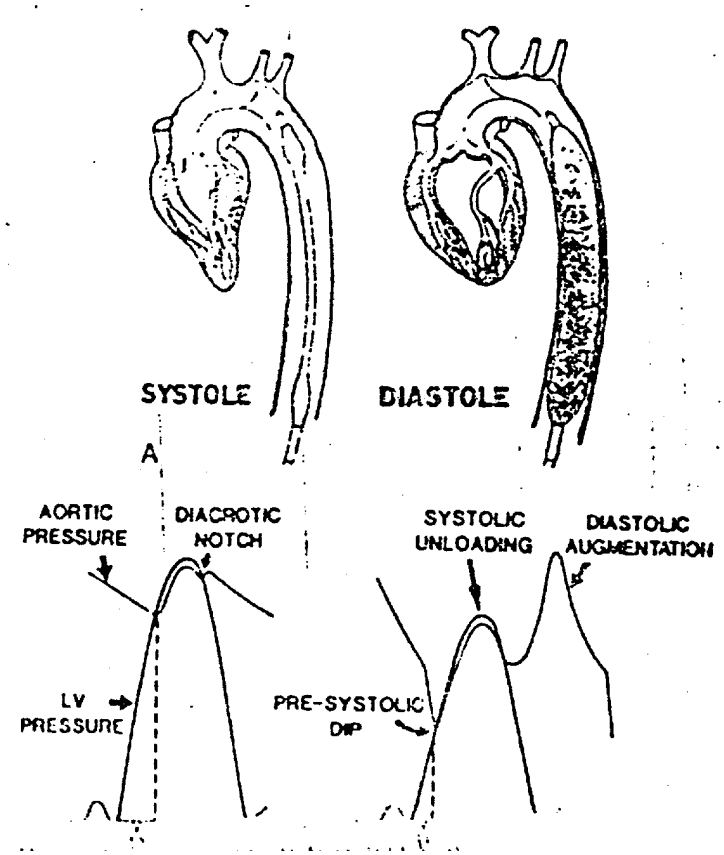
2. Patients with non Q infarction, frequently have marked reversible ischemic transient deterioration of LV function (as evidences by radionu - cleotide ventricular graphy) or large reversible Thallium defects with sub maximal exercise resting have a poor prognosis with medical Rx - ideal candidate for revascularisation (or) surgical procedures.

3. Patient with acute MI with well preserved LV function needs only empirical medical treatment.

II. ACUTE MITRAL REGURGITATION FOLLOWING MI

An acute myocardial infarction may involve the LV - papillary muscle by causing reversible ischemic dysfunction, irreversible infarction dysfunction, infarction with rupture of the papillary muscle head alone or whole papillary muscle with attached ventricular myocardium other than rupture of whole papillary muscle - cause only transient feature of LVF such as new appearance of systolic murmur. LVS_3, inspiratory crackles, tachycardia etc; symptoms of LVF such as orthopnoea / PND clinical evidence of LVF as mentioned above, with radiological features of Acute pulmonary edema are due to papillary

THE PRINCIPLE OF INTRA AORTIC BALOON PUMP PULSATION OF BALOON INFLATION IS TIMED TO TE ARTERIAL DICROTIC NOTCH PRODUCING DIASTOLIC AUGMENTATION IS ARTERY B.P. AND DEFLATION OF BALOON PRIOR TO NEXT VENTEICULAR SYSTOLE CONTRIBUTES TO SYSTOLIC UNLOADING.



muscle rupture or due to Acute Ventricular Septal Rupture. Clinically both papillary muscle dysfunction or rupture can be differentiated by localized ejection or mid systolic murmur - papillary muscle dysfunction; long drawn holo systolic murmur heard all over the precardium conducted to Axilla - papillary muscle rupture. Papillary muscle dysfunction can occur even in Ischamia / non Q infarction especially in inferior wall infarction (Postero medial papillary muscle) or in Anterior wall infarction (Antero lateral papillary muscle). The diagnosis rests on clinical suspicion, 2D echo, M mode echo studies and confirmed by Colour Doppler and right heart catheterization with a flow directed, baloon tipped catheter demonstration and the absence of oxygen step-up in the (a) ventricle (to exclude VSD as cause), large regurgitant V waves in the pulmonary capillary wedge pressure tracing. (Due to augmented antegrade filling of left atrium), right atrial, (R) Ventricular, main pulmonary artery oxymetry can some times needed to differentiate MR with VSR. Treatment principles are similar to LV dysfunction as mentioned above; In most individuals the mitral valve reconstruction / replacement surgery with-in 12 - 24 hrs of diagnosis reduce mortality to a significant extent in patients with severe LVF / Acute pulmonary edema after initial stabilization with medical (or) Intraaortic pulsed balloon counter pulsation.

III. VENTRICULAR SEPTAL RUPTURE AFTER M.I

Intraventricular septal rupture occurs in 1 to 2% of patients with acute M.I. and accounts about 5% death in Acute MI; Anterior wall transmural M.I.

is more common and easier to diagnosis and for surgical repair and prognostically better than inferior wall MI giving rise to VSR.

The patient with acquired VSD likely to have multi vessel disease and poor collateral flow to the ventricular septum. The magnitude of VSD depends on pumping ability of LV, size of defect, relative resistance to flow in pulmonary and systemic vascular bed.

Clinically patients with acute MI, develop VSR after 1 to 7 days with signs and symptoms of acute LVF going to pulmonary Edema. A holo systolic murmur in the 4th LICS with probable with or without a mid diastolic rumble at cardiac apex almost diagnostic of VSR. LVS_3 / ECG / X-Ray evidence of bi-ventricular hypertrophy and both interstitial and alveolar edema seen in X-ray chest, should be sought for in all imminant and established case. Given its clinical utility, availability and non invasiveness 2D, Doppler, studies should be done in all suspected cases of VSR / MR. And if shunt quantification necessary, the right heart cathetarisation with a balloon tiped catheter shall be performed, which shows distinct rise in oxygen saturation >9% between SVC / pulm. artery in right ventricle Telltale and document the presence of intra - cardiac Left to Right shunt. The presence of Lt to Rt shunt be confirmed by dynamic myocardial scintography using Technetium per technetate labeled albumin or RBC's.

Principles of Management

- Diuretics, after load reducing agents, positive ionotrophic support.

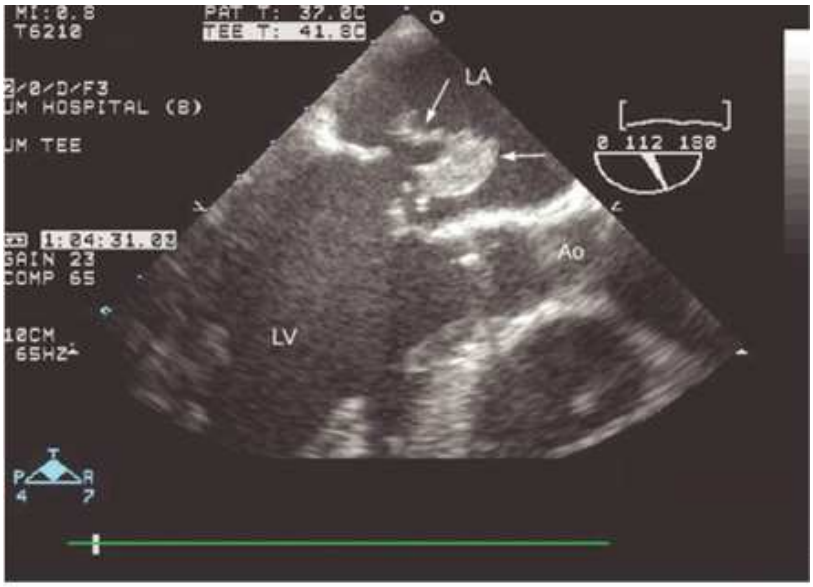
- After load reducing agents, favour increased forward flow of LV by reducing LV impedance, and thus promote lesser shunt.

Intra aortic balloon counter pulsation is extremely useful by decreasing systemic vascular resistance, decrease LVEDP and thus help to decrease intra cardiac shunt and promote increased diastolic filling of coronary arteries.

If pulmonary to systemic flow is >2:1, then surgical correction is mandatory; Anterior wall MI has apical through and through VSR - repair is easier but Inferior wall MI going to VSR is postero basal IV septal and serpigenous rupture associated with postero medial rupture of papillary muscle and hence both surgery and post operative mortality is both less predictable and less favourable.

IV. RUPTURE OF HEART AFTER MYOCARDIAL INFARCTION

Post MI - cardiac failure is common after rupture of the Heart. This occurs after 1 - 7 days of MI. Among the three causes of rupture, electro - mechanical dissociation, sub acute cardiac rupture, false and true aneurysms, the catastrophic rupture due to electromechanical dissociation is common. First infarction / Post MI persistent systemic hypertension / Old age / female gender



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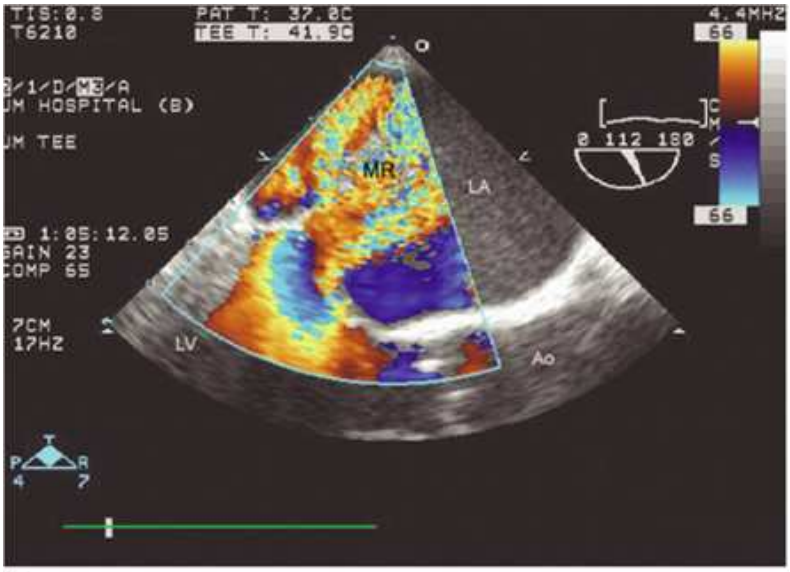
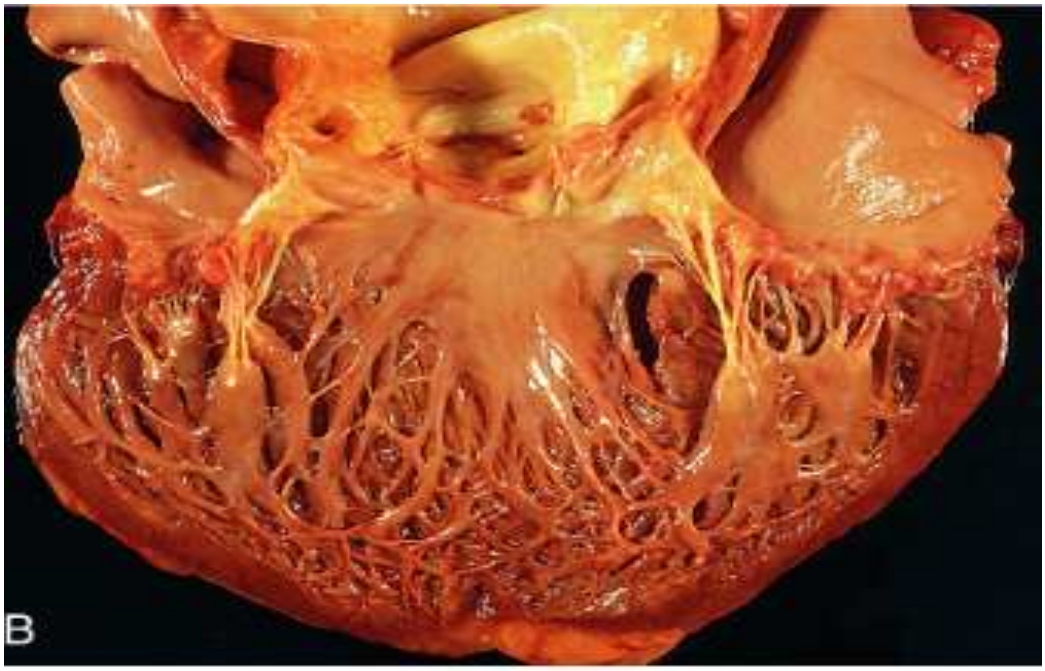


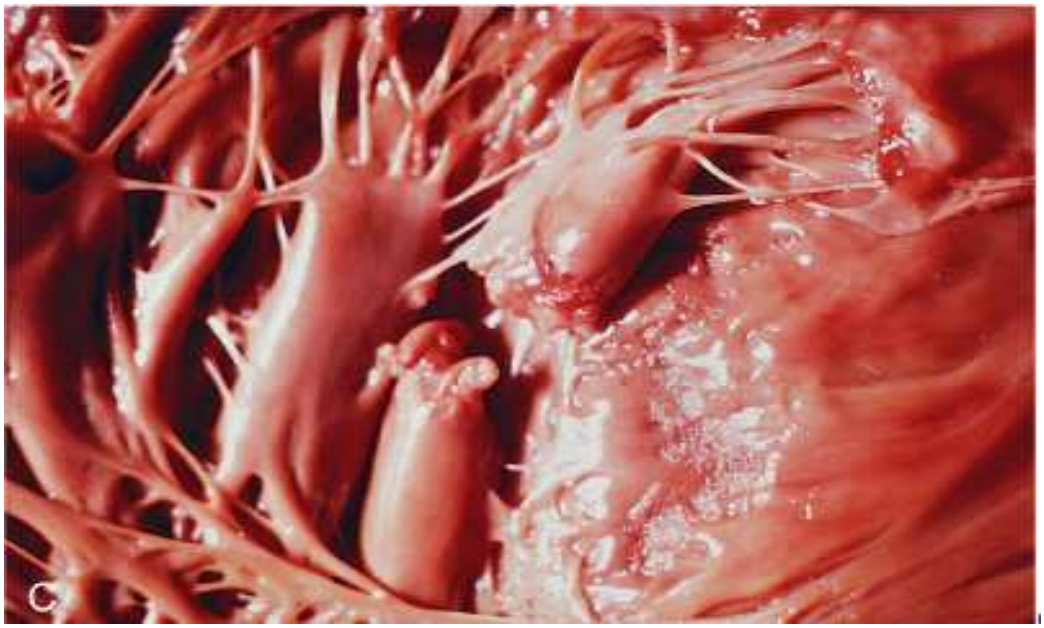
B

Trans-esophageal echo recorded in a patient with an acute MI and complete rupture of papillary muscle.

A. Recorded in a longitudinal view in systole - a large bulky muscular mass can be seen and prolapsing into the Lt. Atrium (arrow). This represents the body of a papillary muscle.

B. Recorded in the same view using colour flow imaging demonstrates the presence of severe MR. LA - Left Atrium, RA - Right Atrium, LV - Left Ventricle, RV - Right Ventricle, AO - Aorta.





Cardiac rupture syndromes complicating STEMI.

B. Rupture of the ventricular septum.

C. Complete rupture of a necrotic papillary muscle.

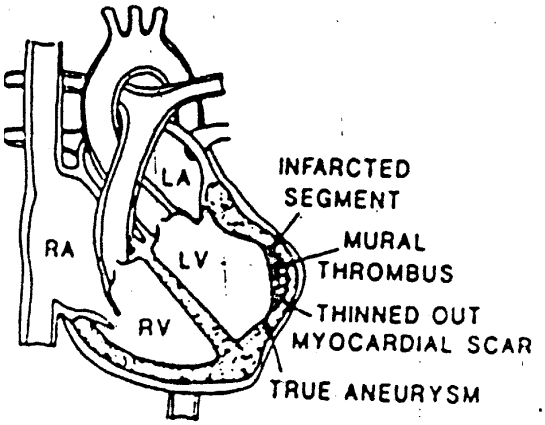
/ No LVH / Belated thrombolytic therapy / use of steriodal / NSAID's are commonest predisposing factors. Investigation and management is that of ventricular septal rupture. Cardiac rupture is the second cause of death during M.I. after heart failure, there is a higher incidence of cardiac rupture in infero - posterior - lateral M.I. after the first 24 hours particularly in the female gender; there is a low global incidence (1.4%)[12].

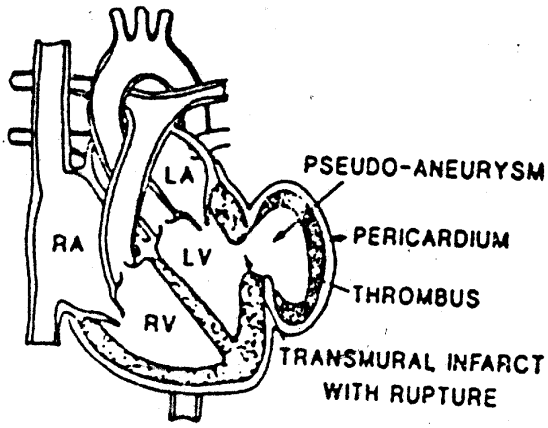
V. LEFT VENTRICULAR ANEURYSMS AND PSEUDO ANEURYSM

LV Aneurysm

The incidence of LV Aneurysms is 7 - 15%. Among them 90% are involving anterior wall and <10% are involving inferior wall. Post MI scar formation leads to larger areas of dyskinetic segments which moves paradoxically during systole in aneurysms. Clinically large anterior wall aneurysms can cause diffuse sustained apical impulse extending upward and medially. Sometimes an ectopic impulse away from apical impulse and double apical impulse may point to the presence of anterior wall aneurysms.

THE DIFFERENCES BETWEEN TRUE AND FALSE ANEURYSMS





TRUE ANEURYSM	**PSEUDO - ANEURYSM**
1. Wide base	1. Narrow base
2. Walls Composed of Myocardium	2. Wall composed of thrombus and pericardium
3. Low risk of free rupture	3. High - risk of free rupture

ECG

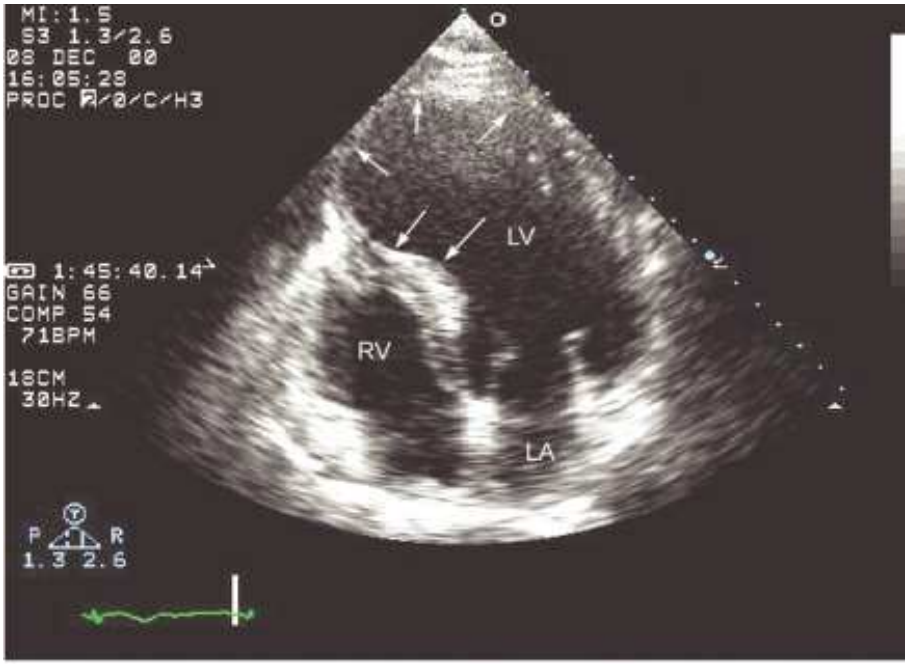
Persistent ST elevation after weeks / months of acute MI, similarly X-ray Chest shows an outward bulge from left ventricular contour; both are indicative of aneurysms, but the absence of aneurysms cannot be ruled out; This anterior wall aneurysms tend to have mural thrombus and subsequent thrombo - embolic phenomena even after / days / weeks / months of its formation and warrants the use of heparin in acute set - up and warfarin in chronic setup until 6 months, after which this embolic complication in Post MI aneurysms thrombus is remote.

In inferior wall MI post scar aneurysm, thrombus formation and thrombo embolic phenomena are rare and hence no need of anticoagulants therapy.

Pseudo Aneurysm

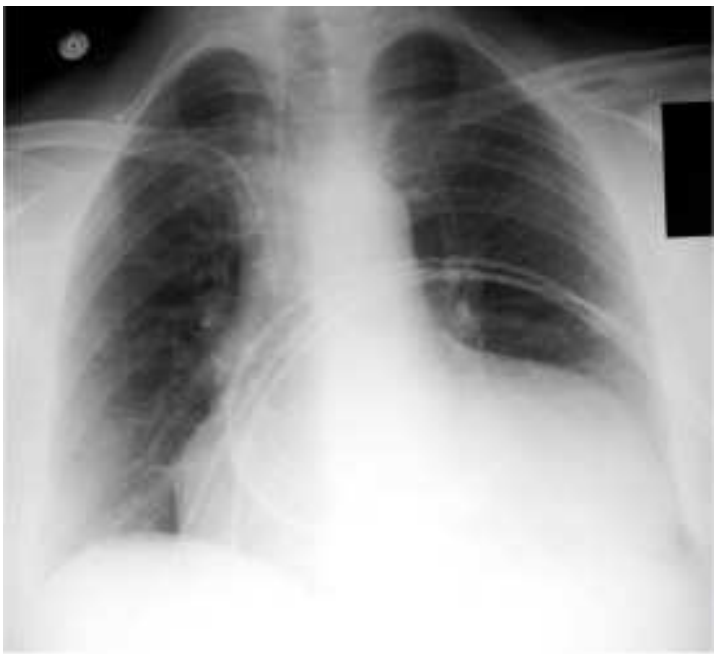
After chest wall trauma / mitral wall surgery / post MI VSR, result-ant hemopericardium in suspicion or complication heals - leaving a small area of adherent pericardial tissue with thin walled sac with myocardium with a small neck called pseudo aneurysm. In post MI VSR, this some times rupture to cause free wall rupture of the LV and post infarction LVF.

ECG, X-ray, 2D Echo, Transthoracic echo are all useful in identify of aneurysm but of its nature. Ultra fast tomography, gated equilibrium blood pool scintigraphy, Doppler studies and LV angiography demonstrates its nature



Apical 4 chamber view recorded in a patient with a large antero apical aneurysm.

Note the marked distortion in geometry seen in this diastolic frame with marked aneurysmal bulging of the distal septum and apex (arrow). LA - Left Atrium, RA - Right Atrium, LV - Left Ventricle, RV - Right Ventricle.



Chest X-ray of a patient with CAHD / HF / Large Anterolateral M.I.

A soft horizontal contour of the Lt ventricle is suggestive of Anterior wall aneurysm

whether pseudo or true aneurysm. LV aneurysms are treated conservatively. Refractory LVF, refractory ventricular arrhythmias and recurrent thrombo embolic phenomena are treated surgically with resection of aneurysms.

VI. RIGHT VENTRICULAR INFARCTION

Over the past 20 years cognisance of RV infarction become more with the advent of Radionucleotide Ventriculography, 2D Echo, electro - cardiography with right sided leads RV_3, RV_4, RV_5 infarction is demonstrable in 40% of Acute IWMI and they dominate the clinical picture in 10% of cases of IW-MI. Proximal right coronary artery occlusion in virtually the cause of RV-infarction in every case, clinically they present with retrosternal chest discomfort, nausea, vomiting and diaphoresis. On Examination, Elevated JVP, Kussamaul's sign, Low BP, RVS_3, holosystolic murmur of tricuspid origin, pulses alternans, rapidly developing peripheral edema with clear lung fields. Approximately 50% of patients with inferior infarction have some involvement of the right ventricle[13]. The Rt ventricle can sustain long periods of ischemia but still demonstrate excellent recovery to contractile function after reperfusion[14].

In ECG ST segment elevation in one or more of (R)side leads RV_3, RV_4 could identify RVMI. Especially RV_4 is specific for acute right ventricular infarction.

Scintigraphic Techniques

1. Technetium 99m stannous pyrophosphate.

2. Thallium 201 scintigraphy.

3. Gated equilibrium Blood pool scintigraphy.

Dominant and hemodynamically significant RV dysfunction and RV infarction can be assessed accurately with flow directed balloon tipped catheter. This shows

1. Disproportionately elevated filling pressures,

2. Normal or only minimally elevated left side filling pressure,

3. Right artrial pressure more than 10 mm of Hg,

4. Less than 5mm of Hg below the pulmonary capillary wedge pressure in a patient with acute inferior wall MI is diagnostic of RV dysfunction / infarction.

Treatment

They are usually silent.

If they produce haemodynamic compromise, the corner stones of therapy are :

1. Patency restoration of infarct - related artery.

2. Intravascular volume expansion.

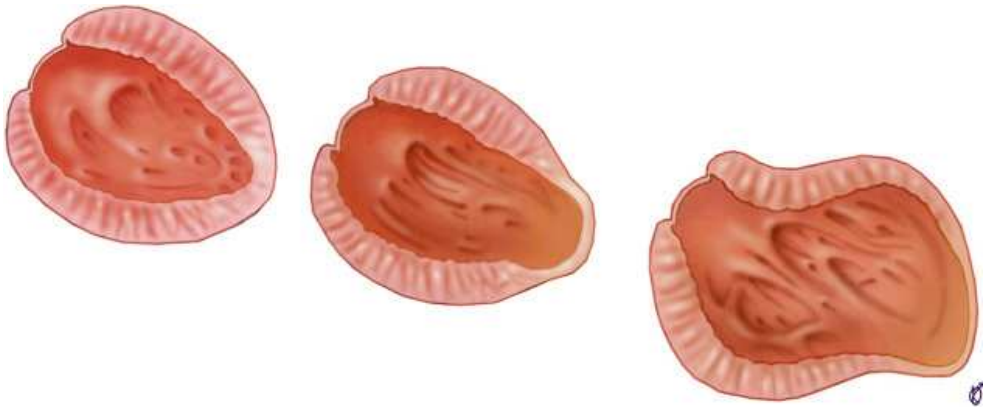
3. Inotrophic support.

   Since right ventricular infarction frequently manifests as atrial fibrillation, prompt restoration of sinus rhythm by transvenous pacing / sequential AV pacing.

4. Markedly elevated right atrial pressure may cause right to left shunt through patent foramen ovale resulting hypoxemia, render the patient unresponsive to supplementary oxygen. Other complication include RV papillary muscle dysfunction, rupture resulting in tricuspid regurgitation and RV thrombus formation and pulmonary embolism. They are managed in the empirical way.

VII. INFARCT EXPANSION AND VENTRICULAR REMODELING

Ventricular remodeling comprising changes in mass, volume, shape and composition, constitutes one of the principal mechanisms by which the heart compensates for an increased load[15]. Patients with severely unfavourable cardiac remodeling such as biventricular enlargement have extremely high myocardiocyte apoptosis at necropsy, even late after LV myocardial infarction,



Remodeling of LV after STEMI.

On the left is shown an apical STEMI (White zone of LV). Over time, the infarct zone elongates and thins. Progressive remodeling of the LV occurs (Center and Rt images) ultimately converting the LV from an oval shape to a spherical shape.

supporting the role of myocardiocyte loss in determining post infarction adverse remodeling[16].

Within hours of transmural necrosis, the necrotic myofibrils slips and cause permanent fixed regional thinning and dilatation of infarct zone over a course of one week. 35 to 42% of anterior wall infarction has this and pose additional wall stress to the systolic contraction and incraesed ESV causes and decreased ejection fraction and subsequent LVF.

Slipping of Myocardial Fibrils

Thinning

|

Dilatation→ Increased ESV→Decreased EF→Ventricular remodeling

|

Pose additional wall stress - Increased oxygen demand

|

Leads to hypokinesia, akinesia, dyskinesia →aneurysm

|

Free wall rupture

As anterior wall MI frequently goes to this complication, early nitroglycerine, ACE inhibitors will prevent LV size enlargements; Early reperfusion with salvaged epicardial rim of myocardium also prevents infarct expansion. Interestingly the recanalisation of infarct related artery, when

significant salvage of myocardium is unlikely - may also prevent LV remodeling.

Variables such as cardiac index, Stroke volume index and both left and right ventricular ejection fractions[17] have been shown to correlate directly with survival in patients with heart failure where as Heart rate, systemic and pulmonary vascular resistances, PAP and PCWP correlate inversely[18].

VIII. MYOCARDIAL STUNNING

Even after reperfusion - (Spontaneous or Therapeutic) periods of persistent and prolonged ischemia for hours, days, weeks, present with Biochemical, ultra - structural and contractile dysfunction which may be accompanied by transient `Q' wave and reversible cardiac failure.

IX. HIBERNATING MYOCARDIUM

Persistent left ventricular dysfunction may be the principle clinical manifestation of chronic myocardial ischemia. The myocardium down regulates or depress its function to match its oxygen supply. Positron emission tomography can identify such hibernating myocardium by both quantification of regional blood flow and metabolic activity.

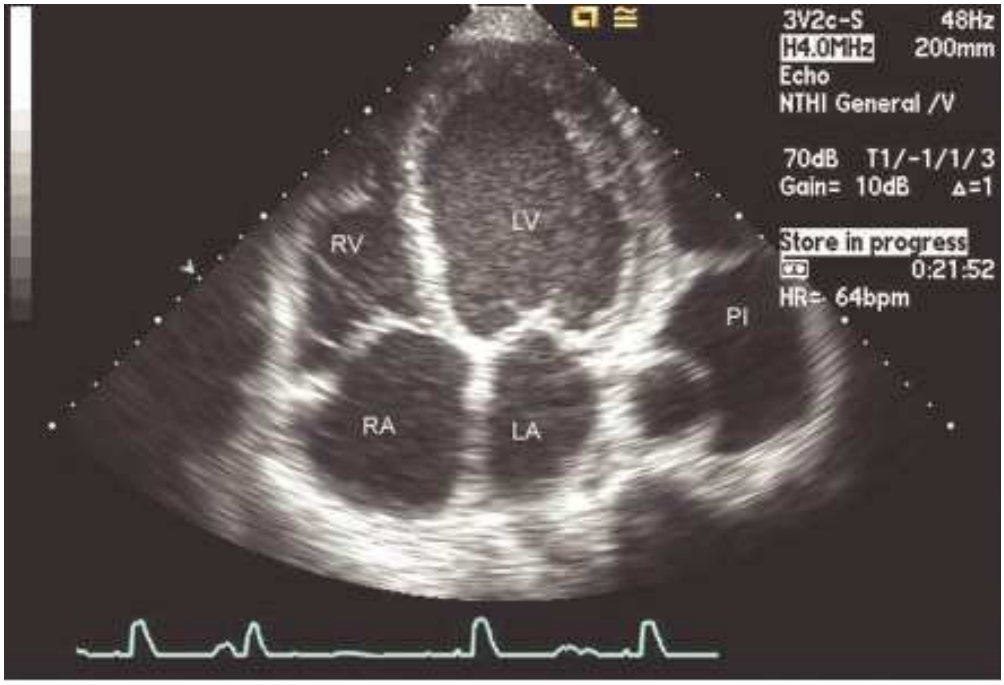
X. STIFF HEART SYNDROME

Acute ischemia of the left ventricle some time caused diastolic dysfunction and acute elevation of left atrial pressure and even cause acute

pulmonary adema. But x-ray chest and ejection fraction are normal during the episode and this condition is called stiff heart syndrome.

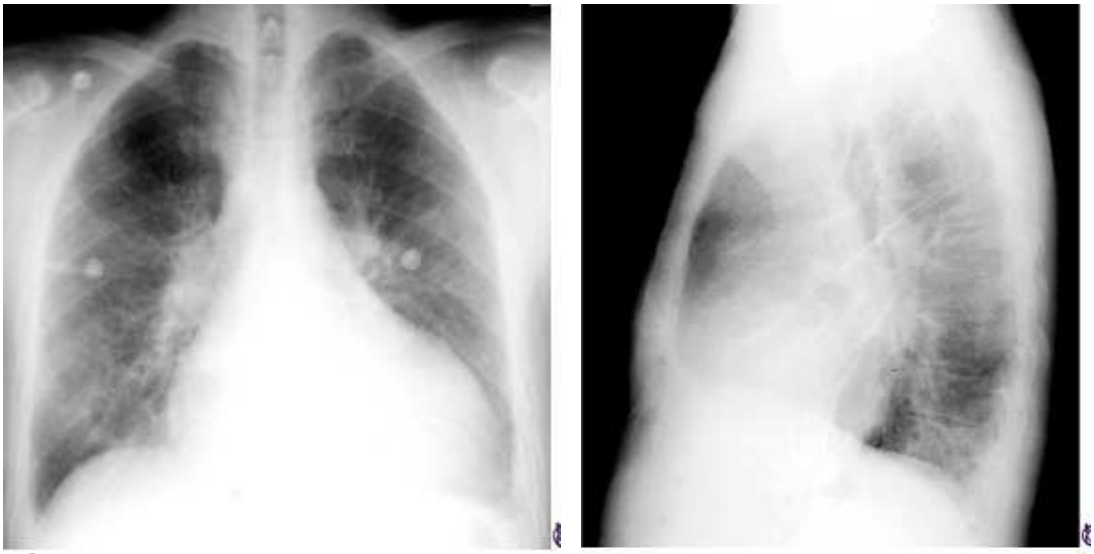
XI. POST INFARCTION ISCHEMIC CARDIOMYOPATHY

This term describes the condition, in which a patient with coronary heart disease has progressive congestive heart failure with prior history of either silent / manifest angina or myocardial infarction. BNP levels corelate with the severity of heart failure and predict survival. A serum BNP of less than 100 pg/ml excludes heart failure as primary diagnosis in dyspnoeic patients[19]. Other wise called end stage IHD is due to multiple or contagious single large infarction in more than one CAD territory. Since their prognosis is poor, even with guarded or no scope with CABG operative mortality is acceptably in patient who present with recurrent angina or recurrent LVF; hence after trial medical treatment, CABG can be suggested in selective cases. Systemic inflammation leads to the release of cytokines that contribute to vasodilation and fall in systemic vascular resistance[20].



Apical 4 chamber view of a patient with DCMP

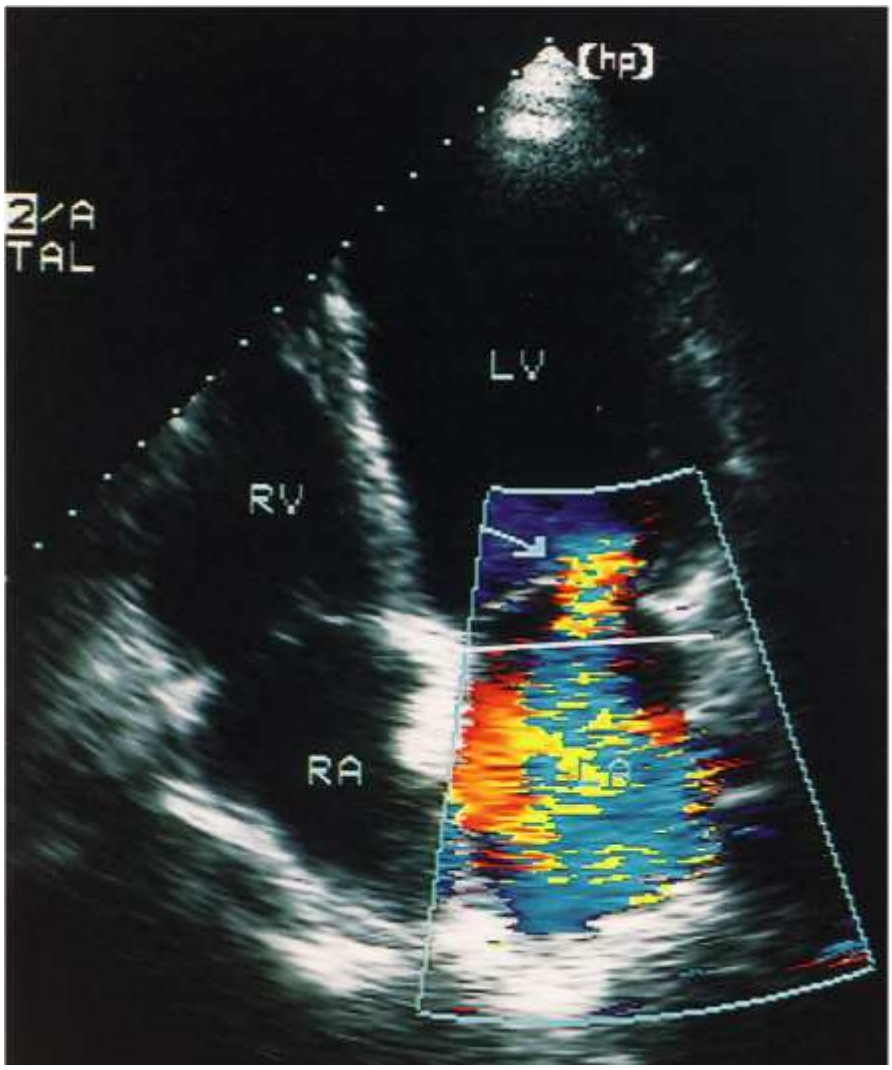
Note the dilation of all 4 chambers and the relatively spherical geometry of the Lt. ventricular cavity. Incidental note is made of a pleural effusion (Pl). LA - Lt. Atrium, RA - Rt. Atrium, RV - Rt. Ventricle, LV - Lt. Ventricle.



A **B**

Chest X-ray of a patient with severe ischemic DCMP with (A) Frontal (PA view) (B) Lateral view

Shows enlarged cardiac silhoutte. Clear enlargement of individual chambers and marked pulmonary vascular redistribution.



Apical 4 chamber view in a patient with DCMP and Severe MR due to dilated annulus abnormal mitral valve coaptation.

The solid horizontal white line represents the plane of mitral annulus. Note that the mitral valve closes well within the cavity of LV. The actual origin of MR jet can be seen as it accelerates toward the regurgitation orifice (arrow) and is likewise displaced into the cavity of the Lt. Ventricle. LA - Left Atrium, RA - Right Atrium, RV - Right Ventricle, LV - Left Ventricle.

INVESTIGATIONS

1. Echo cardiogram

Early and ultimate development of congestive heart failure following first MI were associated with an moderately reduced EF less than 40%, pseudonormal diastolic filling indices and an increased index of myocardial performance (IMP)[21].

a. Mode Echocardiogram

b. Two - Dimensional Echo cardiogram

c. Trans esophageal Echo cardiogram

d. Pulsed Doppler Echo cardiogram

e. Color Doppler Echo cardiogram

f. Contrast Echo cardiogram

g. Three dimensional echo cardiogram

h. Stress - echocardiogram

2. Intra - vascular ultrasound

They are painless and harmless and less costly procedures in the diagnosis of post infarction failure especially in the diagnosis of contractile, diastolic dysfunction, RV infarction, mitral regurgitation, ventricular septal

rupture, cardiac free wall rupture, stunning, hibernation, aneurysms, post infarction ischemic cardiomyopathy.

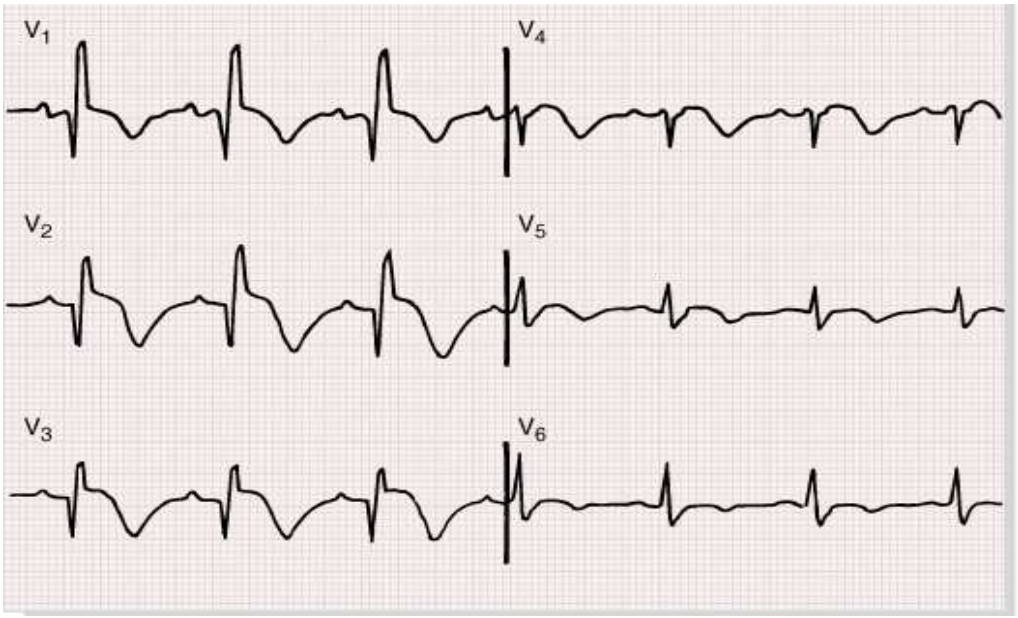
They have limitation that it requires (a) expertise, (b) interpretation competence (c) poor transmission of ultrasound waves through bony structure, air containing lungs (emphysematous patient), difficult to perform the echo examination. Transesophageal echo, circumvent this problem. Pulsed Doppler, colour Doppler assess the pressure gradient between chambers and great vessels and in between chambers and thus diagnose even trivial leaks. Stress echo diagnose exercise induced and drug induced regional wall motion abnormalities among global LV ischemic changes.

3. Electrocardiogram

Heart rate variability as the most relevant derived ECG parameter of sympathetic tone fluctuations may be of important prognostic significance in CCF patients[22]. The sum of ST segment elevations measured from multiple precordial leads correlates with the extent of myocardial injury in patients with the extent of myocardial injury in patients with anterior M.I[23].

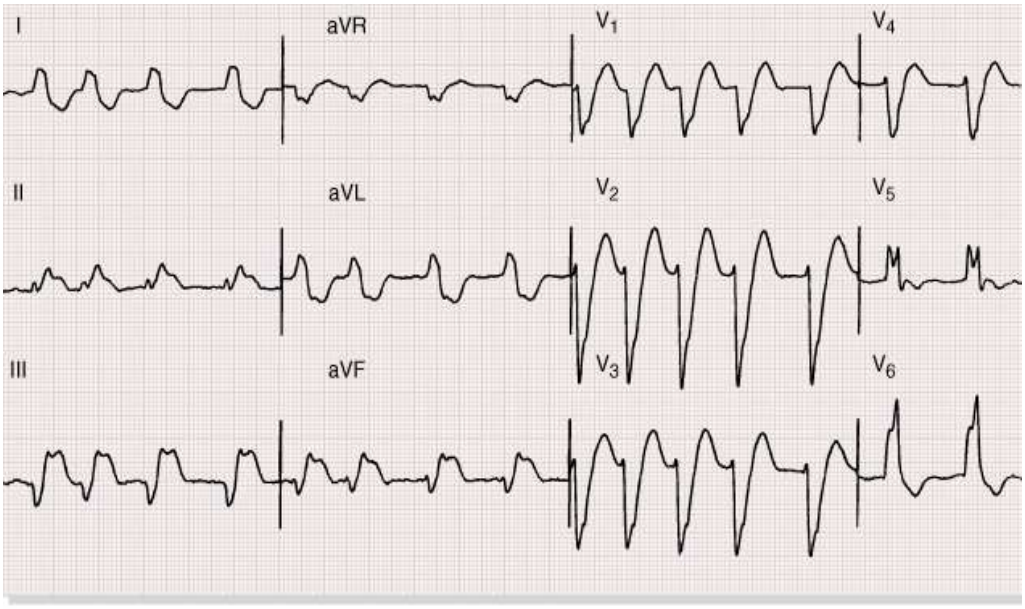
It studies

1. Whether the patient has Q wave / non Q wave infarction.
2. Anterior / inferior wall infarction / RV infarction.
3. Whether the patient has persistent ST-T wave changes (Suggesting LV aneurysm).



RBBB with Acute Anterior wall MI.

Loss of anterior depolarization forces results in QR type complexes in the right precordial to mid precordial leads, with ST elevations and evolving T wave inversions (V_1 through V_6)



Complete LBBB with acute inferior wall MI

Prominent ST segment elevation in Leads II, III, aVF, with reciprocal ST segment depression in Lead I and aVL super imposed on secondary ST - T changes. The underlying rhythm is Atrial fibrillation.

4. To find out both right and left atrial abnormalities indicative of their cavity pressure.

5. Bi ventricular hypertrophy (Acquired VSR).

6. To findout dysrythmias, various cardiac conduction blocks, etc.

4. Radiology of the Heart

Two principal features of the chest radiograph are useful in evaluation of patients with heart failure - size and shape of cardiac silhouette[24].

1. X-ray chest routine PA view: left ventricle, Lt Atrium, pulmonary arteries, LV aneurysm, right atrium. Enlargements can be visualized. Caclified valves and coronary arteries are some times visible.

2. Lateral view : Especially useful in the diagnosis of right ventricle enlargement by obliteration of retrosternal space.

3. Right anterior oblique view : To visualize the left atrium and its enlargement in Post Infarction failure.

4. Lt Anterior oblique : It is superior to other projections for detecting right ventricular enlargement, enlargement of right atrium, septal defects etc.

5. Cardiac fluorscopy

With the advent of Echo and other hitech echo procedures - Fluroscopic analysis become obsolete.

6. Coronary Angiography

This is indicated in suspected VSR or Acute MR with serve congestive cardiac failure and in suspected pseudo aneurysm and any causes of congestive cardiac failure following infarction when present with hypertension and resistant to medical treatment.

Convalescent non Q-MI patients for feasibility of future surgical reperfusion technique.

Techniques

i. Right heart catheterization. This is useful for measurement and analysis of right heart pulmonary artery, PCW pressure etc.

ii. Left heart catheterization. (Hudkin's techniques) useful to find the left ventricular end systolic and diastolic volume / pressure etc.

iii. Trans-septal catheterization.

iv. Angiograms directed insertion of Intra aortic balloon counter pulsation. Devices - useful in the treatment of Post Infarction failure.

7. Nuclear Cardiology

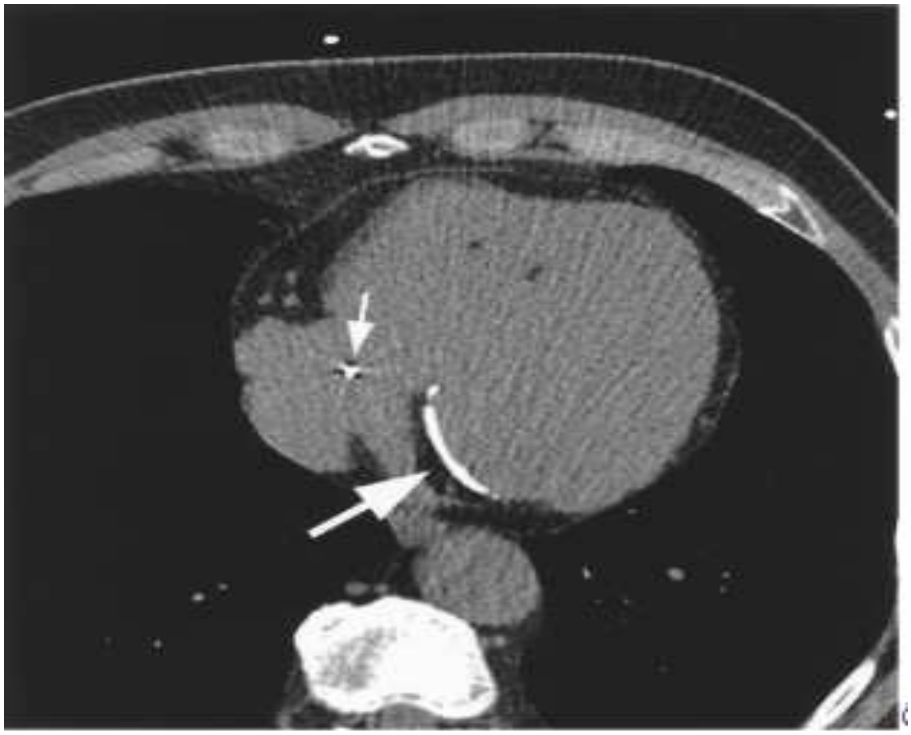
This provides early insight to the presence of systolic and diastolic dysfunction and valvular problem following infarction, right ventricular dysfunction can be assessed by studying first pass and equilibrium techniques (f-PERNA) and ERNA (Equilibrium Nucleotide angiography technique). Radionuclide angiography, perfusion imaging, infaract-avid scintigraphy and positron emission tomography have been used to evaluate patients with STEMI [25].

8. Positron Emission Tomography

Useful for the diagnosis of myocardial viability and regional or global left ventricular dysfunction.

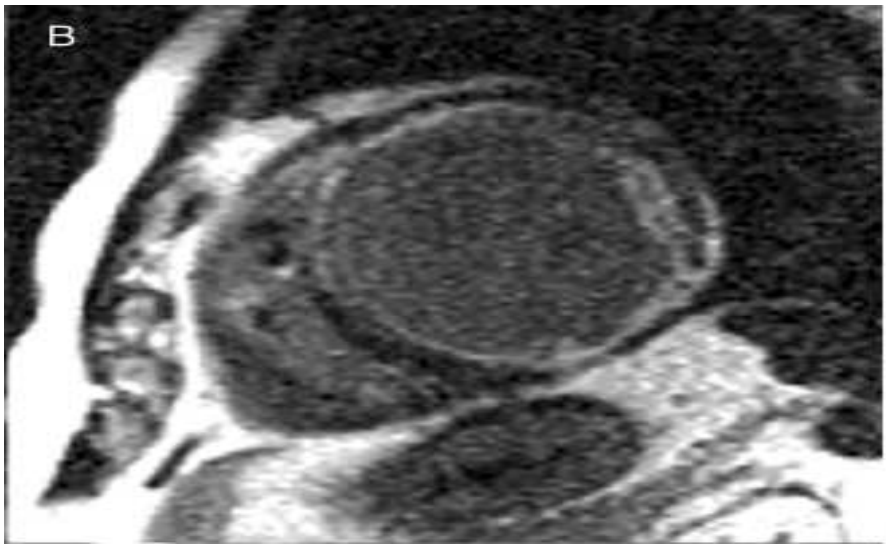
9. Magnetic Resonance Imaging (MRI)

It provides direct visualization of the myocardium and its segmented wall thinning, thickening and presence of thrombus. True and false aneurysms; mitral valvular regurgitation in phase images. To identify myocardial edema, fibrosis, wall thinning and hypertrophy; to assess ventricular chamber size and segmental wall motion and to identify the temporal transition between ischemia and infarction[26].



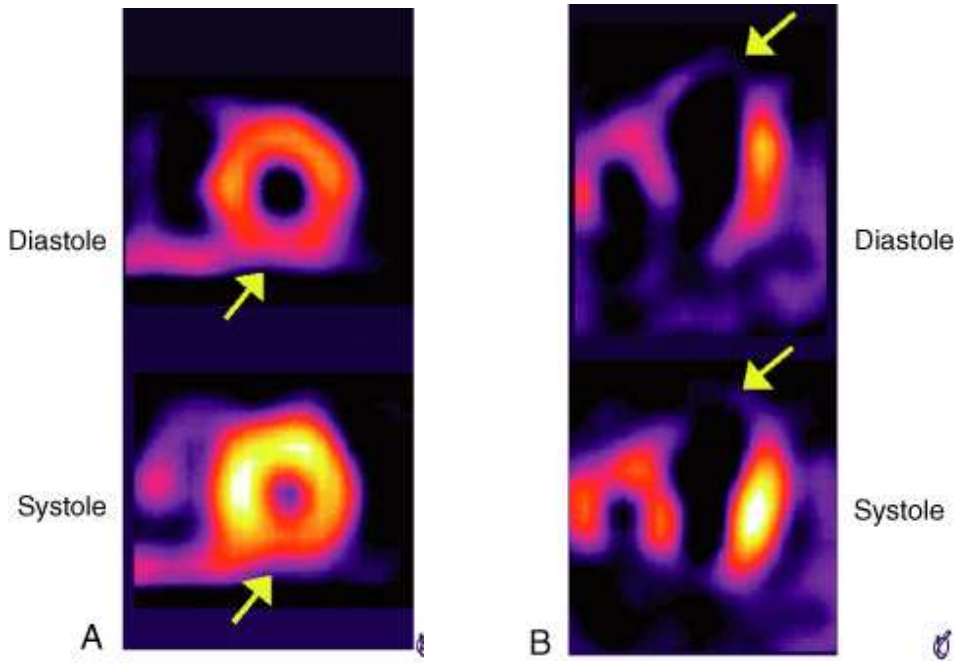
In Post MI patient with non enhanced CT scan showing

Calcified LV aneurysm (large arrow) and Pacemaker lead (small arrow).



Patient with known previous MI resulting in HF with Cardiovascular Magnetic Resonance (CMR) imaging

Note late gadolinium enhancement seen through the subendocardium causing thinning particularly in the septum and inferolateral wall.



Examples of regional dysfunction detected by ECG gated Single Photo Emission CT (SPECT) perfusion imaging.

A. Hypokinetic inferior region appears to brighten less (arrow) than the other regions from diastole to systole.

B. The akinetic apex in the horizontal long axis (arrow) appears to have no change from diastole to systole in contrast to the normally thickening (brightening) lateral walls.

TREATMENT

This is either medical or surgical; medical treatment of post MI - cardiac failure is use of diuretics to relieve congestion, vasodilators to unload the heart, ionotrops to strengthen pumping ability of the heart. They all in single or combination relieve symptoms, improve hemodynamic indices, improve survival, forstall LV remodeling and prevent progression of cardiac failure. In combination with nitrates, hydralazine improves survival in patients with heart failure[27]. Along with this, ACE inhibitor has further advantage of prevention of further intimal proliferation of atherosclerotic coronary and other vessels and prevent restenosis.

ACE inhibitors attenuate ventricular enlargement[28].

Angiotensin II inhibition may contribute to myocardial protection include attenuation of endothelial dysfunction and direct anti - atherogenic effects[29].

ARBs reduce mortality and morbidity associated with heart failure in patients who are receiving an ACE inhibitor[30].

However, the effects of long term exercise training on survival are not defined[31].

One group of patients whose ejection fraction is normal but present with gross diastolic dysfunction and CCF - Beta blockers are ideal (like atenolol, sotolal, metaprolal etc) in whom needless to say ionotrophs and after load

reductors are contraindicated. Eplerenone, a selective aldosterone receptor antagonist without the hormonal side effects of sprinolactone reduces mortality in patients with heart failure associated with Acute M.I.[32]. In patients with advanced heart failure, the cardio - renal syndrome may be aggravated by several commonly used classes of drugs including ACEI, ARB, diuretics, NSAID and Cox 2 inhibitors[33]. Treatment with nocturnal Continuous Positive Airway Pressure improves symptoms and LV ejection Fraction[34].

If mechanical complications are present assess whether angina or CCF is predominant symptoms respectively, CABG or intraaortic baloon counter pulsation, left ventricular assisted device and dynamic cardiomyoplasty. Any one shall be addressed which may bridge to eventual cardiac transplantation if donor became available.

MATERIALS AND METHODS

This study was conducted at Madras Medical College, Chennai - 3, during the period of May 2004 to January 2006. Hundred cases of post myocardial infarction cardiac failure were included in this study. The patients were selected from those who were admitted in the medical wards and those who attend cardiology clinics. All the patients were evaluated in detail by clinical, ECG, X-ray, Echo - methods.

In the analysis of clinical findings elaborate history regarding the modes of presentation and symptoms were sought for and recorded. Symptoms of cardiac failure such as NYHA, Grade dyspnoea, orthopnoea, PND, NYHA classifiaction of angina, palpitation, fatiguability, giddiness, syncopy, coughs, hemoptysis, dysphagia and hoarseness of voice are recorded. In history taking, number of previous infarction, time window between onset of chest pain and admission in hospital, angina and dyspnoea free interval between attacks were recorded, systemic diseases, treatment regularity, relevant family history, substance abuse, basic investigations and their findings were also documented. All volunteers of this study were subjected to ECG, echo study and X-ray (in selective cases). In echo studies, the territory of wall motion abnormalities, systolic, diastolic dysfunction, presence and absence of pericardial effusion, clot, aneurysm and calcification were noted. Left atrial and right ventricular dilatation were looked into for the presence of residual post MI failure especially when they are planned for discharge. A copy of the echo cardiogram was issued to the patient for the future reference and follow up. In relevant patients blood investigation were done to study the biochemical parameters.

OBSERVATION, ANALYSIS AND DISCUSSION

TABLE-1

AGE AND SEX INCIDENCE

Age group (in year)	Male	Percentage	Female	Percentage
20-30	2	2%	-	-
31-40	8	8%	-	-
41-50	20	20%	2	2%
51-60	30	30%	6	6%
61-70	20	20%	2	2%
71-80	10	10%	-	-
Total	90	90%	10	10%

Analysis And Discussion

The incidence of post MI cardiac failure is seen as early as in the younger age group such as 20-30. In our study they were males from rural, non sedentary worker aged about 28 yrs and 30 yrs.

This incidence goes up as age advances, and peaks at 51-60 yrs group paralleling the incidences of coronary artery disease. In the female population, the incidence starts from the age group 41-50, that is from perimenopausal age, where oestrogen deficient endocrinopathy are prevalent and progression of this rate from 2 to 6% to 51-60 age group could be attributed to this.

TABLE-2

DWELLING AND INCIDENCE OF POST MI-CARDIAC FAILURE

Age group (in year)	Urban	Percent-age	Semi urban	Percent-age	Rural	Percent-age
20-30	-	-	-	-	2	2%
31-40	6	6%	-	-	2	2%
41-50	14	14%	4	4%	4	4%
51-60	20	20%	4	4%	12	12%
61-70	10	10%	4	4%	8	8%
71-80	4	4%	4	4%	2	2%
Total	54	54%	16	16%	30	30%

Analysis And Discussion

Observed data showed the incidence of post MI cardiac failure are more in urban followed by rural and semi urban population. The reasonable explanation of this finding could be ascertained. Type A individuals (aggressive, impatient, competitive) are known to have CAHD; and substantial number of type A individuals prevalence in the urban shall be the explanation or the incidence could be co-incidental rather casual.

TABLE - 3

NATURE OF PHYSICAL ACTIVITY AND INCIDENCE OF POST MI FAILURE

Age group (in year)	Sedentary	%	Mixed nature	%	Non sedentary	%
20-30	-	-	-	-	2	2%
31-40	6	6%	-	-	2	2%
41-50	14	14%	4	4%	4	4%
51-60	20	20%	4	4%	12	12%
61-70	10	10%	4	4%	8	8%
71-80	4	4%	4	4%	2	2%
Total	54	54%	16	16%	30	30%

Analysis And Discussion

Sedentary life style have more incidences than either non sedentary or mixed pattern of physical activity in almost all age group as in coronary heart disease.

Garrow states that in the U.K. an increasing proportion of the food eaten is processed to make it more palatable and easy to prepare with long shelf life. These prototypic convenient foods are preparations of fat and sugar such as biscuits.

Urban population constantly consumes these foods. In addition urban civilisation also reduces the need for strenuous physical exercise. These trends increase energy intake and decrease energy output leading to obesity, dyslipidemias, CAHD and consequent post infarction failure.

TABLE-4

INFLUENCE OF SUBSTANCE ABUSE, DIET PATTERN, BODY MASS INDEX AND FAMILY HISTORY IN 100 CASES OF POST MI CARDIAC FAILURE

Factors	Age group (in years)							
	20-30	**31-40**	**41-50**	**51-60**	**61-70**	**71-80**	**Total**	**%**
Family History Positive History	-	6	10	10	10	6	42	42
Negative History	2	2	12	26	12	4	58	58
Diet Vegetarian diet consumers	-	2	6	6	6	2	22	22
Mixed diet	2	6	16	30	16	8	78	78

Non Vegetarian Diet	**Frequency**							
	Daily	**Bi-weekly**	**Weekly**	**Monthly**	**Occasionally**	**-**	**Total**	**%**
Beef	2	16	12	2	4	-	36	36
Mutton	2	16	18	6	6	-	48	48
Fish	2	4	2	-	2	-	10	10
Eggs/Chicken	2	6	12	2	10	-	32	32
Substance abuse Alcohol	8	22	8	-	16	-	54	54
Tobacco chewing	4	-	-	-	30	-	34	34
Smoking	**Few**	**<5/day**	**6-10 /day**	**11-20/day**	**>20/day**	**-**	**-**	**-**
	-	6	22	18	30	-	76	76
Body mass index	**<20**	**<22**	**<24**	**<26**	**< 28**	**> 30**	**-**	**-**
	30	10	10	14	16	20	100	100

Analysis And Discussion

Contrary to the observation made in various text books about the familial inheritance of CAHD, in our study negative family history out number of +ve family history. Especially poor people with mixed or non sedentary (Daily waged) manual labourers are noticed developing acquired hypercholesterolemia with beef consumption the observation needs large scale, multicentric randomized and blind trials.

Smoking appears to be a major risk factor for CAHD even with-out significant coronary narrowing (SUGISHI M, TAKATSU F-Cigarette smoking is a major risk factor for acute MI and subsequent cardiac failure, circulation 1993, 87: 76-9).

Consumption of alcohol regularly in cardiac disease may predispose them to transient and chronic form of left ventricular dysfunction observed as decrease EF, increase EDV and cause left ventricular failure (Spodick DH, Pigott VM, CHIRIFER-Preclinical malformation in chronic alcoholism-New England Journal of medicine 1972, 287; 677-680).

Cigarette smoking suppresses body weight, discourging many smokers from trying to quiet. It has been demonstrated that exposure to nicotine increases metabolic rates. Of more than 80 studies conducted, a majority indicate that smoking suppresses body weight. Smokers weight less than non smokers. (The cardiac thoracic journal volume.2, No.11, January 1997, page 14-16).

This study could be referred and addressed for reason how poor body mass index could lead on to increasing incidences of infarction and post MI failure in our study.

TABLE-5

SYSTEMIC ILLNESS AND NATURE AND NUMBER OF INFARCTIONS IN POST MI CARDIAC FAILURE

Symptoms	Age group (in year)							
	20-30	31-40	41-50	51-60	61-70	71-80	Total	%
Systemic high blood pressure	-	6	8	21	5	8	48	48
Type II diabetes	2	2	12	5	3	-	24	24
Non diabetic and non hypertensive	-	-	2	10	14	2	28	28
1st Episode MI & PIF	2	2	6	10	10	2	32	32
Known IHD	2	6	16	26	12	8	70	70
Silent MI-Present as PIF	-	-	-	-	2	-	2	2
Non Q MI	-	-	2	4	2	-	8	8
H/o One MI Episode	-	2	6	14	10	6	38	38
H/o Multiple MI Episodes (2 time MI)	-	2	4	6	4	2	18	18
H/o 3 Time MI	-	-	-	2	2	-	4	4
Bronchial asthma/COPD	-	-	4	8	2	2	16	16
Peripheral occlusive arterial disease	-	-	2	2	2	-	6	6

Analysis And Discussion

From among the data available, though non diabetic and high blood pressure recorded individuals are also prone to develop POST MI failure, recorded high blood pressure accounts for in substantial No. of cases (48%) and NIDDM the third in the list (24%).

Post infarction cardiac failure in 30% cases present on 2nd to 7th day of acute infarction with no H/o. of previous CAHD. They are due to transient, reversible LV dysfunction following MI and in most of them the failure symptoms disappeared when they were discharged from hospital.

In substantial number of cases failure is due to either with H/o. one episode of MI (38%) or 2 episodes of MI (18%) or multiple episodes of MI (4%) or with H/o. non QMI (with available previous ECG and Enzyme studies) (8%) or even with silent infarction (No. H/o. of recent or old infarct related chest pain) but present with Q wave in ECG left ventricularly symptoms (2%) in descending orders of frequency.

Peripheral occlusive arterial disease was noticed in 6 patients with absent pulses alone in two case and in four cases with established and recovered neurological dysfunction, all were in the later age group and blood VDRL (-ve) in them and hence luteal or other angitis related coronary artery disease were excluded as the cause of post MI failure.

Obstructive airway disease was found in 16% cases which are parallel comorbidity factor in the mortality assessment in post MI failure. The percentage of COPD in our study would have been still higher, but 2 of them died while on study and hence could not be incorporated in the study.

TABLE-6

SYMPTOMATOLOGY IN POST MI CARDIAC FAILURE

Symptoms		Age (in year)							
		20-30	**31-40**	**41-50**	**51-60**	**61-70**	**71-80**	**Total**	**%**
Breathlessness	NYHA I	-	-	2	2	-	-	4	4
	NYHA II	-	4	4	8	6	-	22	22
	NYHA III	-	4	8	6	12	2	32	32
	NYHA IV	-	-	4	10	12	2	28	28
	PND	2	-	6	20	20	4	52	52
	ORTHOPNOEA	2	-	8	22	22	6	60	60
Angina	REST	-	6	12	16	4	6	44	44
	EFFORT	-	6	8	8	6	4	32	32
	NOCTURNAL	2	-	4	12	10	2	30	30

Analysis And Discussion

In the younger age group 31-40, 41-50 the post MI cardiac failure presents as rest/effort angina after an episode of infarction. This post infarction angina, in the context of non-collaterality of coronary circulation and poor LV long term adoptive hypertrophy leads to post MI failure; adding to it, a better lung reserve in young age preclude them to have dyspnoeic manifestation over the old age post MI patients in whom the reverse incidence (i.e.) more dyspnoeic manifestation and less angina manifestation may be attributed to poor lung reserve due to associated smoke related and age related lung changes and collaterality of coronary circulation respectively. As age of infarction advances the cardiac myofibrils shrink left ventricle dilates (rather hypertrophic) and left ventricle became atrophic, probably the compromised to blood supply could adequate the need and hence the lesser anginal manifestation, as age of CAHD advance.

TABLE-7

OTHER SYMPTAMATOLOGY IN POST MI FAILURE

Symptoms	Age group (in year)							
	20-30	**31-40**	**41-50**	**51-60**	**61-70**	**71-80**	**Total**	**%**
Palpitation	-	2	-	6	6	-	14	14
Easy Fatiguability	-	-	2	4	4	-	10	10
Giddiness	-	-	2	2	4	-	8	8
Syncope	-	-	2	4	2	-	8	8
Postural cough	2	-	2	14	8	-	26	26
Hemoptysis	-	-	-	6	2	-	8	8
Dysphagia	-	-	-	10	4	-	14	14
Hoarseness of voice	2	-	-	14	4	-	20	20
Decreased weight and appetite	-	-	-	8	-	-	8	8

Analysis And Discussion

Symptoms of poor cardiac output such as tissue fatiguability, giddiness, syncopy are lesser common than symptoms of progressive left atrial enlargement and lung congestion such as cough, hemoptysis, dysphagia and hoarseness of voice in Post MI failure. Because of acute and chronic adoptive mechanisms following myocardial infarction to maintain the stroke volume, cardiac output at the expense of increased LV EDV and left atrial pressure, the virtual absence of these symptoms in 71-80 age groups has no reference in our study. Though symptom adaptability, possessiveness and poor neurogenic reflex could be speculated.

TABLE-8

ANALYSIS OF SIGNS IN POST MI CARDIAC FAILURE

Signs	Age group (in year)							
	20-30	**31-40**	**41-50**	**51-60**	**61-70**	**71-80**	**Total**	**%**
Tachycardia	2	8	22	36	22	10	100	100
LVS_3	2	2	4	12	16	8	44	44
Cardiomegaly (shift of Apical impulse)	2	-	12	18	20	8	60	60
JVP / Edema / Ascitis	2	2	8	14	10	4	40	40
Apical Pansystolic Murmur	2	4	4	14	14	6	44	44
Other Non - pansytolic murmur	-	2	6	6	6	2	22	22

Analysis And Discussion

Virtually the classical signs of post Infarction Cardiac Failure is negligible in younger age group and at extremes of age in our study. They present with only tachycardia whereas 41 - 70 age group does present with such signs. Where the history of one or multiple infarction with chronic slowly progressive LVF could be related to this signs.

In 40% of cases increase JVP / Edema / Ascitis are due to chronic congestive heart failure and in 4% of cases due to right ventricular infarction. In 3% of our cases, the 4 LICS pansystolic murmur were heard (VSR), but could not be incorporated in this study as they were died during the course of the study.

TABLE-9

ANALYSIS OF ELECTRO CARDIOGRAM IN POST M1 CARDIAC FAILURE

Myocardial wall frequency	**Antero septal**	**Extensive anterior**	**Inferior wall**	**Anterior & Inferior**	**Non Q MI**	**RV infarct**	**True posterior wall**
No of patients	24	24	22	20	10	4	2
Percentage	24	24	22	20	10	4	2
Chamber Enlargement	**LAE**	**LVH**	**LAE + LVH**	**RAE**	**RVH**	**RAE+ RVH**	**RVH+ LVH**
No. of patients	20	12	4	6	2	-	-
Percentage	20	12	4	6	2	-	-
Conduction Blocks	**Peri infarction Block**	**LBBB**	**RBBB**	**LAFB**	**LPFB**	**1^{o} Heart Block**	**$2^{o}/3^{o}$ Heart Block**
No. of patients	18	12	4	2	-	2	-
Percentage	18	12	4	2	-	2	-
Dysrythmias	**Atrial premature contraction**	**Atrial Fibrillation**	**Junctional Rhythm**	**SVT**	**Vent. bigemini**	**VPC's**	**-**
No. of patients	4	6	2	2	6	10	-
Percentage	4	6	2	2	6	10	-

Analysis and Discussion

Combined anterior wall and inferior wall MI is cause of 20% of post MI CCF because of global systolic dysfunction. The incidence of Anteroseptal and Anterior wall MI in PIF in our study was in consistent with the report published in American Heart journal.

Incidence of peri-infarction block are commoner in our study is due to focal degeneration of conduction pathways in the area of infarction and subsequent scar formation.

Dysrythmias : Among the available data, atrial fibrillation and ventricular premature contractions are common dysrythmias encountered.

Bigeminy : Bigeminy and ventricular premature contractions are individual predictors of survival of post MI failure patients. Together both constitutes 16 percent in our study (Mukerji, Rude JE, Poole WK, Gustaton N, Thomas, Braunwald E, Risk factors for sudden death in post MI, American Journal of Cardiology 1997, 54 : 316).

TABLE - 10

ECHO FINDINGS IN POST MI CARDIAC FAILURE

Regional wall motion abnormality	**Akinesia**	**Hypokinesia**	**Dyskinesia**	**Hyperkinesia**	**Combined Lesions**	**No Lesions**
No. of Patients	12	72	16	-	-	-
Percentage	12	72	16	-	-	-
Functional Abnormalities	**Systolic dysfunction**	**Diastolic dysfunction**	**Combined dysfunction**	**RV dysfunction**	**Global dysfunction (ischaemic CMP)**	**No dysfunctin**
No. of patients	72	8	10	6	4	-
Percentage	72	8	10	6	4	-
Possible mechanical Defect in 2D ECHO	**PMD / MR**	**VSR**	**Anuresym**	**Pericardial effusion (small)**	**Calcification**	**Thrombus / clot**
No. of patients	22	-	8	32	20	8
Percentage	22	-	8	32	20	8
Chamber Enlargement	**LAE**	**LVH**	**RAE**	**RVH**	**LAE + LVH**	**No enlargement**
No. of patients	42	28	4	4	22	30
Percentage	42	28	4	4	22	30

Analysis and Discussion

Large number of patients had uniform pattern of hypokinesia, mild to moderate and severe systolic dysfunction with or without left atrial enlargement and LVH. In subtle number of cases the diastolic dysfunction is due to symmetrical or asymmetrical hypertrophy of left ventricle and global

dysfunction are due to post infarction ischemic cardiomyopathy (Who had QS complexes in ECG but with or without history of myocardial infarction).

Striking to observe is the presence of mild pericardial effusion in 32% of cases.

1. They are due to sympathetic effusion.
2. Sterile effusion (As a part of congestive heart failure)
3. Dressler's syndrome
4. Focal pericarditis
5. Co-incidental rather causal

Dyskinetic LV segments (Apical) and clot formation in that segment are all witnessed in 2D echo in 8 patients and the benefit of 2D echo study was transmitted to the patients by instituting long term anticoagulant therapy at least for six months beyond which likelihood of systemtic embolisation are negligible.

The calcifications are due to mitral valve / Aortic valve calcification. Coronary artery calcification and calcification of aneurysm wall could not be made out in our study.

CONCLUSIONS

1. Post MI cardiac failure is commoner in urban dwelling (54%) than rural (30%) than semi urban (16%).

2. Post infarction cardiac failure is common in male (90%). After 40 years of age raising incidence are observed in female.

3. Smoking (More than 20 cigarette's per day) and consumption of alcohol has linear relationship with incidence of post MI failure.

4. Sedentary Life Styles (58%), mixed diet pattern (78%), consumption of sheep or goat flesh rather chicken, egg, fish have positive and profound influence over the occurrence of post MI failure.

5. Most common clinical presentation is Tachycardia 100%, in a patient with chest pain or history of infarction. This is followed by cardiamegaly in 60% of case and LVS_3 in 44% of patients and pansystolic murmur in 44%.

6. Most common symptomatology are NYHA IV 28%, orthopnoea 60% and Paroxysmal Nocturnal Dysponea 52%, NYHA III 32%, Rest angina 44%, effort angina 32%, nocturnal 30% and postural cough 26%.

7. Systemic high blood pressure with infarction (48%) was the single most aetiological factor for Post MI cardiac failure. Multitude of other factors,

NIDDM 24%, history of one or two more infarction, chronic obstructive airway disease (16%) are other factors for post MI cardiac failure.

8. Anteroseptal wall and extensive anterior wall infarctions are the leading causes of post MI cardiac failure (24%) each. Perinfarction blocks (18%), ventricular premature contractions (10%) are the ECG manifestations concluded.

9. In 2D echo analysis hypokinesia (72%), Systolic Dysfunction (72%), small pericardial effusion (32%), Papillary muscle - Dysfunction / Mitral Regurgitation (PMD / MR) (22%), valves calcification (20%), left atrial enlargement (42%) are the observations made.

SUMMARY

It is a prospective study of 100 cases of post myocardial infarction cardiac failure analyzing various factors like incidence, influences on clinical presentations with reference to history, clinical examination and investigations. PIF is one among the common complication of acute MI and it is caused by contractive and diastolic dysfunction, ischemic cardiomyopathy, RV MI, myocardial stunning, stiff heart syndrome and acute mitral regurgitation. From our study, PIF is common in male and after 40 years of age raising incidences are observed in females also. PIF is common in urban dwelling and patients with sedentary life styles and in chronic smokers. SHT with MI was the single most aetiological factor for post MI cardiac failure. Anteroseptal wall and extensive anterior wall infarction are the leading causes of post MI cardiac failure.

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PROFORMA

1. General Information

1. Serial Number
2. Patient Name and Age Sex / Address
3. Occupation and Income
4. Social Group
5. Dwelling
6. IP / OP

II. Chief Complaints

1. Dyspnoea
2. Dysponea Grade
3. Orthopnoea
4. PND
5. Angina (Rest)
6. Angina (Effort)
7. Palpitation
8. Easy Fatiguability
9. Giddiness
10. Syncopy
11. Cough
12. Hemoptysis
13. Dysphagia / Hoarseness of Voice
14. Loss of Weight / Appetite

III. Past History :

1. History of myocardial infarction.
2. Number of Infarction / Hospital Admission for Ailment
3. History of First Infarction
    1. Time of Onset of Chest Pain
    2. Time Window for Hospitalisation
    3. Thrombolytic Therapy
    4. Duration of Hospital Stay
    5. Angina Free Interval After Hospital Discharge
4. Systemic Hypertension
5. Diabetes
6. IHD
7. Syphilis / AIDS
8. Rheumatic Heart Disease
9 Congenital Heart Disease
10. Peripheral Vascular Disease
11. Tuberculosis
12. Bronchial Asthma
13. Corpulmonale
14. Any other Chronic / Acute Relevant Medical Illness

IV. Treatment History

Coronary Vasodilators / Beta
Blockers / Calcium Channel Blockers / Anti
Arrhythmics / Antiplatelets / Frequency of Sublingual Nitrates
ACE Inhibitors / Diuretics / Digoxin / Others
Anti Hypertensives / Anti Diabetics / Cholesterol Lowering Drugs

V. Family History

VI. Personal History

Nature of Job
Type of Food : Vegetarian / Non - Vegetarian / Mixed
Animal / Meat Consumption

Habits	Duration	Quantity of Consumption
a. Smoking		
b. Alcohol		
c. Pan Parag		
d. Tobbaco Cheweing		
e. Other Forms of Nicotine		
f. Oral Contraception		

VII. Clinical Examination

1.	Temperature	2.	Pulse	3.	Respiration
4.	BP	5.	JVP	6.	Edema / Ascitis
7.	Nicotine Stains	8.	Height	9.	Weight
10.	Body Mass Index	11.	Obesity	12.	Arcus senilis
13.	Xanthoma	14.	Xanthalesma	15.	Carotids
16.	Locomotor brachii	17.	Anemia	18.	Clubbing
19.	Cyanosis	20.	Jaundice	21.	Polycythemia
22.	Fundus	23.	Features of corpulmonale	24.	Others if any

VIII. Systemic Examination

i. Examination of CVS

1. Apical Impulse
2. Sounds
3. Murmurs
4. Rub
5. Knock Sound
6. Additional Events

ii. Examination of Respiratory System

iii. Examination of Abdomen

iv. Examination of CNS

IX. Investigations

1. Urine
2. Blood
3. ECG
4. X-ray Chest
5. Echocardiogram

X. Final Diagnosis

XI. Comment

MASTER CHART

Sl.No.	In Patient Register No.	Age in yrs.	Sex	Urban (U) / Semi Urban (S) / Rural (R)	Sedentary (S) / Mixed (M) / Non Sedentary (N)	Postive (P) / Negative (N) Family History	Vegetarian (V)/ Mixed Diet (M)	SHT	DM_2	Non SHT & Non DM	IHD	COPD	PVD
1	728123	41	M	U	S	P	M	-	+	-	+	-	-
2	728199	58	M	R	S	N	M	-	-	+	-	-	-
3	728230	43	M	R	S	N	M	+	-	-	+	-	-
4	728243	62	M	R	S	N	M	-	-	+	+	-	-
5	728280	34	M	U	S	P	V	+	-	-	+	-	-
6	728301	59	M	R	S	N	M	-	-	+	+	-	-
7	728358	51	M	U	N	N	V	+	-	-	-	-	-
8	728389	55	F	R	N	N	M	+	-	-	+	-	-
9	728452	44	M	R	S	P	M	-	+	-	+	-	-
10	728499	71	M	U	N	P	M	+	-	-	+	-	-
11	728522	56	M	U	S	N	M	-	+	-	-	-	-
12	728572	65	M	R	N	N	M	-	-	+	+	-	-
13	728688	49	M	U	S	N	M	-	+	-	+	-	-
14	728786	56	F	R	N	N	M	+	-	-	+	-	-
15	728792	68	M	R	S	N	M	-	-	+	-	-	-
16	728821	40	M	R	S	N	M	-	+	-	+	-	-
17	728897	58	M	U	S	P	M	+	-	-	+	-	-
18	728992	74	M	U	N	P	V	+	-	-	-	-	-
19	729015	51	M	S	M	N	M	-	+	-	+	-	-
20	729100	70	M	R	N	N	M	-	-	+	-	-	-
21	729170	48	M	U	S	P	M	-	-	+	+	-	-
22	729194	52	M	U	S	P	M	-	-	+	+	-	-
23	729241	65	M	R	M	N	V	-	-	+	+	-	-
24	729282	55	M	R	S	N	M	+	-	-	+	-	-
25	729410	61	M	U	N	N	M	-	-	+	-	-	-
26	729522	58	M	U	M	N	V	+	-	-	+	+	+
27	729821	50	M	U	N	P	M	-	+	-	+	-	-
28	729912	68	M	U	S	N	V	-	-	+	+	+	-
29	730122	53	M	R	S	N	M	-	-	+	+	-	-
30	730280	66	M	R	M	N	M	-	-	+	+	-	-
31	730512	50	M	R	N	P	M	-	+	-	+	-	-
32	730782	76	M	U	M	P	M	-	-	+	+	-	-
33	730920	58	M	R	S	N	M	+	-	-	+	-	-
34	731186	51	F	R	S	N	M	+	-	-	-	-	-

Sl.No.	In Patient Register No.	Age in yrs.	Sex	Urban (U) / Semi Urban (S) / Rural (R)	Sedentary (S) / Mixed (M) / Non Sedentary (N)	Postive (P) / Negative (N) Family History	Vegetarian (V)/ Mixed Diet (M)	SHT	DM_2	Non SHT & Non DM	IHD	COPD	PVD
35	731271	59	F	R	N	N	M	+	-	-	-	-	-
36	731428	63	F	U	M	P	M	-	-	+	-	-	-
37	731589	36	M	U	S	P	M	-	+	-	+	-	-
38	731732	52	M	U	S	P	M	+	-	-	+	-	-
39	731928	63	M	S	S	P	M	+	-	-	-	-	-
40	732418	58	M	U	N	P	M	+	-	-	+	-	-
41	732621	49	M	U	S	N	M	+	-	-	-	-	-
42	732860	50	F	U	N	P	M	+	-	-	+	-	-
43	733168	41	M	U	M	N	V	-	+	-	+	-	-
44	733519	65	M	U	S	P	M	-	+	-	-	-	+
45	733928	60	M	U	N	P	M	+	-	-	+	-	-
46	734019	38	M	U	S	P	M	+	-	-	+	-	-
47	734377	73	M	S	M	N	M	-	-	+	-	-	-
48	734811	52	M	U	S	P	V	-	-	+	-	-	-
49	735008	69	M	U	S	P	V	+	-	-	-	-	-
50	735184	59	M	U	S	N	M	+	-	-	+	+	+
51	735721	50	M	U	S	P	M	-	-	+	+	-	-
52	735988	71	M	S	M	N	M	+	-	-	+	-	-
53	736168	51	M	R	N	N	M	-	-	+	-	-	-
54	736729	48	M	R	M	N	M	+	-	-	+	+	-
55	736954	58	M	U	S	P	V	-	+	-	+	+	-
56	737122	67	M	U	S	P	M	-	+	-	+	-	-
57	737480	51	M	U	N	N	M	-	-	+	-	-	-
58	737501	28	M	R	N	N	M	-	+	-	+	-	-
59	737808	53	M	U	S	P	M	+	-	-	+	-	-
60	738148	72	M	U	M	N	M	+	-	-	+	-	-
61	738803	59	M	U	M	N	M	+	-	-	+	+	-
62	738951	42	M	U	S	N	V	-	+	-	+	-	-
63	739825	58	M	S	N	N	M	-	+	-	-	-	-
64	739911	65	M	S	S	P	M	+	-	-	-	-	-
65	740461	49	M	U	S	N	M	+	-	-	+	+	-
66	740802	74	M	R	S	N	M	+	-	-	+	+	-
67	741820	51	M	U	N	P	M	-	-	+	+	-	-
68	742119	37	M	R	S	P	M	+	-	-	+	-	-

Sl.No.	In Patient Register No.	Age in yrs.	Sex	Urban (U) / Semi Urban (S) / Rural (R)	Sedentary (S) / Mixed (M) / Non Sedentary (N)	Postive (P) / Negative (N) Family History	Vegetarian (V)/ Mixed Diet (M)	SHT	DM_2	Non SHT & Non DM	IHD	COPD	PVD
69	742535	61	M	U	M	N	M	-	-	+	-	-	-
70	742712	56	M	R	N	P	M	+	-	-	+	-	-
71	743358	41	M	U	S	N	M	-	+	-	-	-	-
72	744579	52	F	S	S	N	M	+	-	-	+	+	-
73	745620	58	M	U	N	N	M	+	-	-	+	-	-
74	745731	62	M	R	N	P	V	-	-	+	-	-	-
75	746127	39	M	U	N	P	M	+	-	-	-	-	-
76	747100	52	M	U	S	N	M	+	-	-	-	-	-
77	748619	43	M	U	S	P	M	+	-	-	+	+	+
78	749118	68	M	U	N	P	M	-	-	+	+	-	-
79	749940	55	M	U	M	N	M	-	-	+	+	+	-
80	750830	61	M	R	N	N	M	-	-	+	+	-	-
81	751656	74	M	S	S	P	M	+	-	-	+	-	-
82	752117	49	M	U	S	N	V	-	+	-	+	-	-
83	753548	58	F	U	S	N	M	-	-	+	+	+	-
84	754356	60	M	S	S	N	M	+	-	-	+	-	-
85	755670	66	M	U	S	N	V	+	-	-	+	-	-
86	756889	35	M	U	S	P	M	+	-	-	-	-	-
87	757282	42	F	S	S	N	V	-	+	-	+	-	-
88	758103	50	M	U	N	P	M	+	-	-	-	-	-
89	759418	67	M	S	S	P	M	+	-	-	+	+	+
90	760769	59	M	R	S	N	V	+	-	-	+	-	-
91	761628	30	M	R	N	N	M	-	+	-	+	-	-
92	762540	75	M	S	S	P	V	+	-	-	+	-	-
93	763119	47	M	S	S	N	V	+	-	-	-	-	-
94	767008	66	F	U	N	P	V	-	-	+	+	-	-
95	774134	52	M	U	S	N	V	-	+	-	+	+	-
96	780222	68	M	S	N	N	M	-	+	-	+	-	-
97	781618	33	M	U	N	N	V	+	-	-	+	-	-
98	783785	42	M	S	M	P	V	-	+	-	-	+	+
99	784839	78	M	R	S	P	M	+	-	-	+	+	-
100	786287	45	M	S	M	N	M	-	+	-	-	-	-

GLOSSARY

ACEI	-	Angiotensin converting enzyme inhibitor
AF	-	Atrial fibrillation
AIDS	-	Acquired immunodeficiency syndrome
ARB	-	Angiotensin receptor blocker
AV	-	Atrioventricular
BNP	-	Brain natreuretic peptide
BP	-	Blood pressure
CABG	-	Coronary artery bypass graft surgery
CAHD	-	Coronary arterial heart disease
CCF	-	Congestive cardiac failure
CMP	-	Cardiomyopathy
COPD	-	Chronic obstructive pulmonary disease
COR.	-	Coronary
2D	-	Two dimentional
DCMP / DCM	-	Dilated cardiomyopathy
DM/DM_2	-	Diabetes melitus / type 2 diabetes melitus
ECG	-	Electrocardiogram
EDV / P	-	End diastolic volume / pressure
EF	-	Ejection fraction
ESV / P	-	End Systolic volume / pressure
HF	-	Heart failure
IHD	-	Ischemic heart disease
IV	-	Interventricular
IW	-	Inferior wall
JVP	-	Jugular venous pressure
L / Lt	-	Left
LA	-	Left atrium
LAE	-	Left atrial enlargement
LAFB	-	Left anterior fascicular block
LAP	-	Left atrial pressure
LBB	-	Left bundle branch
LBBB	-	Left bundle branch block
LICS	-	Left intercostal space
LPFB	-	Left posterior fascicular block
LV	-	Left ventricle
LVF	-	Left ventricular failure
LVH	-	Left ventricular hypertrophy
MI	-	Myocardial infarction
MR	-	Mitral regurgitation
MRI	-	Magnetic resonance imaging
NIDDM	-	Noninsulin dependent diabetes melitus
NO.	-	Number
NSAIDS	-	Nonsteroidal antinflammatory drugs

NYHA	-	Newyork heart association
PAP	-	Pulmonary arterial pressure
PCW/PCWP	-	Pulmonary capillary wedge pressure
PIF	-	Post infarction failure
PMD	-	Papillary muscle dysfunction
PND	-	Paroxysmal nocturnal dyspnoea
PVD	-	Peripheral vascular (arterial) disease
R/Rt	-	Right
RA	-	Right atrium
RAE	-	Right atrial enlargement
RBBB	-	Right bundle branch block
RBC	-	Red Blood cell
RCA	-	Right coronary artery
RV	-	Right ventricle
RVF	-	Right ventricular failure
RVH	-	Right ventricular hypertrophy
S_3	-	Third heart sound
SA Node	-	Sino atrial node
SHT	-	Systemic hypertension
S/Sl No.	-	Serial Number
STEMI	-	ST elevation myocardial infarction
SV	-	Stroke volume
SVC	-	Superior vena cava
SVT	-	Supra ventricular tachycardia
VDRL	-	Venereal disease research laboratory test for syphilis
Vent.	-	Ventricular
VPC	-	Ventricular premature contraction
VSD	-	Ventricular septal defect
VSR	-	Ventricular septal rupture
Yrs	-	Years
1° / 2° / 3°	-	First / Second / Third Degree
<	-	Less than
>	-	More than
+	-	Present
_	-	Absent