New Concepts in the Management of Acute Myocardial Infarction

M. Hafizullah,* M.B.,B.S., M.R.C.P.,
Postgraduate Medical Institute, Lady Reading Hospital, Peshawar, Pakistan

Summary

Management of acute myocardial infarction is individualised to the demands of the patient. In general, pain is alleviated, hypoxaemia corrected and physical activity curtailed. Early detection of arrhythmia and correction by drugs or D.C. conversion has improved in-hospital mortality. Left ventricular failure and cardiogenic shock adversely effect the prognosis and merit early treatment. Recently attempts have been made to contain myocardial infarct size by either reducing oxygen metabolic demand or increasing perfusion, by employing drugs or surgical techniques. Secondary prevention entails correction of risk factors and use of beta blockers. By-pass surgery is indicated in symptomatic patients.

Introduction

Nystagmatic eye motion while watching cardiac monitor, eyes popping out in the excitement of catching an ectopic, tightly holding syringes loaded with Lignocaine, Atropine and Verapamil in one hand and clinging to Dopamine and Dobutamine infusions with the other hand and a pacemaker lead wrapped around the neck — such is a state in which a coronary care nurse looks after an acute infarction patient.

Due to the unpredictable course, management varies from analgesics and monitoring on one hand to emergency cardiac surgery on the other hand. More than sixty per cent of deaths associated with acute myocardial infarction (A.M.I.) occur within the first one hour.[1] Since the inception of coronary care units, there has been a sharp decline in the in-hospital mortality from 30% to 15%.[2]

The management broadly entails firstly general measures, secondly prompt detection and correction of arrhythmia, thirdly correction of haemodynamic disturbances, fourthly measures to limit the size of the infarct, fifthly steps to be undertaken for secondary prevention of infarction and sudden death.

*Senior Registrar, Deptt. of Cardiology, Postgraduate Medical Institute, Lady Reading Hospital.

1. General Measures

Myocardial infarction usually announces itself with severe capricious pain; therefore, alleviation of pain is the primary concern. Morphine administered intravenously is still the most effective and remains the drug of choice.[3] An anti-emetic, for example Prochlorperazine or Metoclopramide, may be used simultaneously to counteract the emetic effect of Morphine. Patients with myocardial infarction commonly have hypoxaemia which can be corrected by the administration of oxygen at 2–4 litres/minute for twenty four to forty eight hours by a nasal cannula or a mask.[4] There is ample evidence now to suggest that it protects myocardial ischaemia.[5] Heart rate, arterial blood pressure, cardiac output and myocardial contractility are increased during activity or exercise: this can adversely effect myocardial infarct size by increasing the work of the heart. Physical activity is restricted for twenty four to thirty six hours in uncomplicated infarction and for more time in complicated infarction. Patients are gradually mobilised before leaving the hospital. In some centres, patients are required to undergo a submaximal effort tolerance test before discharge for psychological support and as a prognostic indicator.

2. Detection and Correction of Arrhythmia

Ninty per cent of patients have some sort of ectopic activity.[6] Development of continuous electrocardiographic monitoring, invention of synchronised electrical defibrillator and introduction of new anti-arrhythmic drugs have contributed greatly to the control of lethal arrhythmias.

1. *Premature Ventricular Contractions (P.V.C.)* constitute the commonest occurring arrhythmia following acute myocardial infarction (A.M.I.). Infrequent sporadic P.V.C. occur in almost all patients and do not require any specific therapy.[6] Indications for treating frequent P.V.C. differ from centre to centre. In general if P.V.C. are more than one in ten sinus beats, multifocal in origin, couplets or triplets — they merit treatment. Aggressive approach is desired when an ectopic 'R' wave is super-imposed on a preceding 'T' wave (i.e. early part of diastole) as this can trigger ventricular tachycardia or fibrillation. The ectopic activity can usually be controlled with time-tested lignocaine administered intravenously.[7] A loading dose of 1 mg/kg is given followed by infusion. A second dose may be tried if the arrhythmia persists. If Lignocaine is unsuccessful, twenty minutes later another anti-arrhythmic of group I or group III should be tried. Hypokalaemia should be on the top of check list if the treatment fails to abort P.V.C. Usually oral maintenance therapy is not indicated.

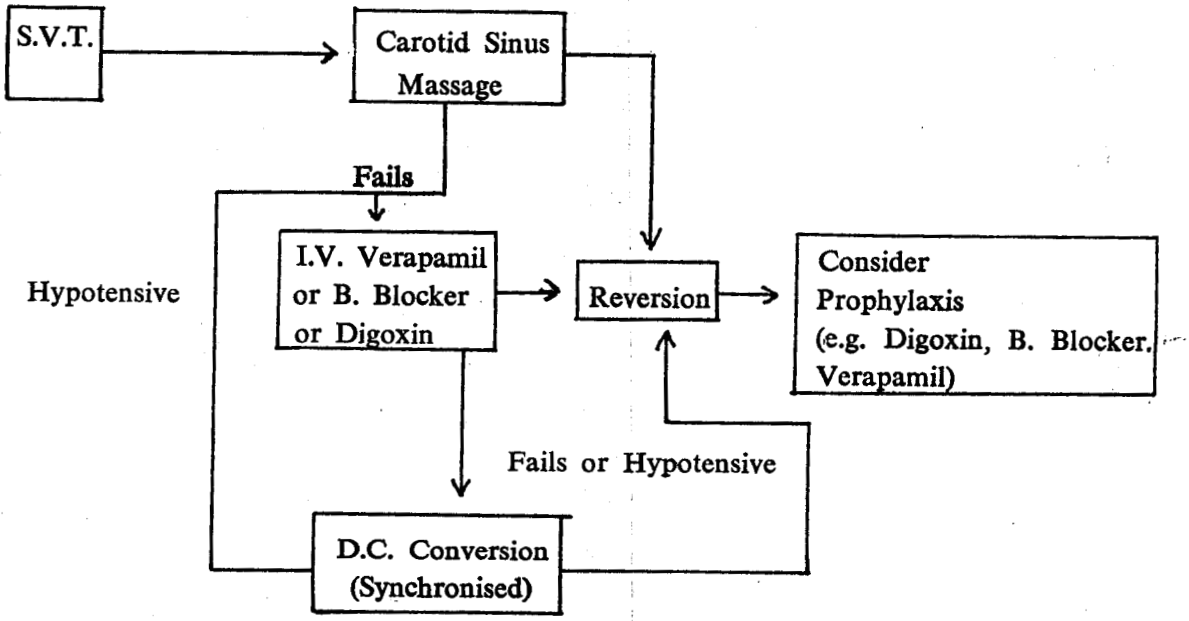
2. *Sinus Bradycardia.* Upto 40% of patients experience sinus bradycardia, more frequently seen in patients sustaining inferior and posterior wall A.M.I.[8]

Treatment is indicated if the patient becomes symptomatic like hypotensive. Atropine 0.5–1.5 mg may be used intravenously.

3. *Sinus Tachycardia* is common and may be due to anxiety, pain, fever, pericarditis or left ventricular failure. It has been recognized as a poor prognostic factor in patients with extensive myocardial infarction. Fifty per cent of patients with persistent sinus tachycardia over 100 beats per minute die in hospital.[9]

4. *Supraventricular Tachycardia (S.V.T.)* increases heart work and can unfavourably effect myocardial infarct size. In case of haemodynamic deterioration or persistent pain, synchronised D.C. conversion is the treatment of choice. Cautious and determined carotid sinus message should be always tried first, before using intravenous Verapamil (5–10 mg), a beta-blocker (e.g. Inderal 1–5 mg) or Digoxin. The latter usually takes 20–30 min. to show its effect[9] (Table I).

TABLE I MANAGEMENT OF SUPRA VENTRICULAR TACHYCARDIA IN A.M.I.

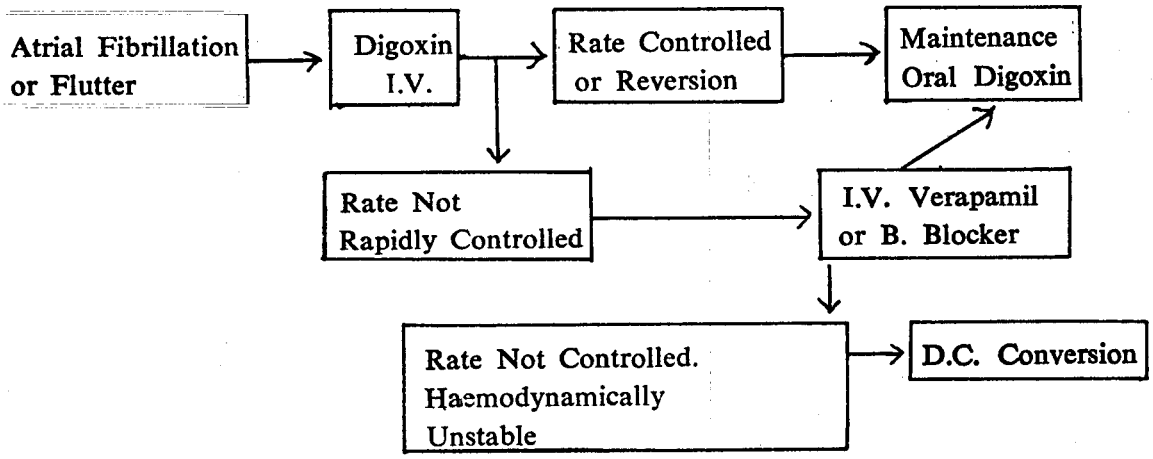


5. *Atrial Fibrillation* occurs in 10% of patients, mostly those with left ventricular failure. Commonly it lasts for 2–4 hours and recurs in 50% of patients.[10] If ventricular response is rapid, similar course of treatment as for S.V.T. is meted out (Table II).

6. *Nodal Rhythm* is uncommon and rarely of any clinical significance.[9]

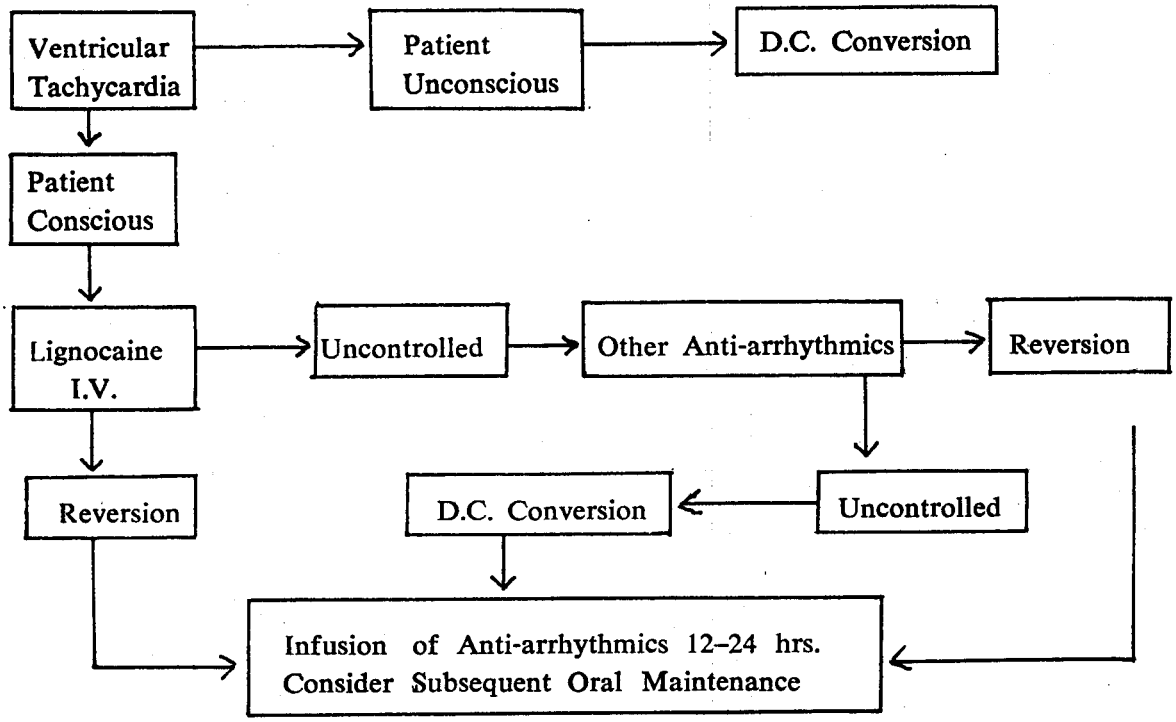
7. *Idio-Ventricular Tachycardia:* ventricular rate is 60–100 beats/min. and is rarely of any haemodynamic consequence. However there are higher chances of this arrhythmia converting into ventricular tachycardia or fibrillation.[9]

TABLE II. MANAGEMENT OF ATRIAL FLUTTER/FIBRILLATION IN A.M.I.



3. *Ventricular Tachycardia (V.T.).* Slow V.T. without impairment of consciousness can be treated like frequent P.V.C. If drugs fail or there is haemodynamic deterioration, then it requires D.C. conversion followed by intravenous anti-arrhythmic usually Lignocaine[11] (Table III).

TABLE III MANAGEMENT OF VENTRICULAR TACHYCARDIA



4. *Ventricular Fibrillation (V.F.)* — commonest lethal arrhythmia: should be immediately treated with asynchronous D.C. conversion.[12]

Temporary Bipolar Transvenous Pacemaker should be inserted in :–

(a) complete heart block in anterior A.M.I. and in inferior A.M.I. if there is associated haemodynamic deterioration.

(b) second degree block in anterior A.M.I. and to be considered in inferior A.M.I. with haemodrynamic deterioration.

(c) alternating left and right bundle branch blocks;

(d) prophylactically in patients with bifascicular block especially left posterior hemi-block with right bundle branch block[13],[14] and

(e) sinus or junctional bradycardia, not responsive to Atropine.

3. Correction of Haemodynamic Disturbances

Prognosis of a patient can be reasonably predicted taking in account the haemodynamic status. Killip[15] divided patients in four groups (Table IV) :–

Class 1: no signs of failure — hospital mortality 0–5%.

Class 2: signs of left ventricular failure (L.V.F.) as tachypnoea, tachycardia, gallop rhythm and basal crepitation — hospital mortality 10–20%.

Class 3: severe heart failure — hospital mortality 35–45%.

Class 4: heart failure with shock, systolic pressure less than 90 mm Hg — hospital mortality 85–95%.

TABLE IV KILLIP CLASSIFICATION OF PATIENTS WITH ACUTE MYOCARDIAL INFARCTION

Class	Definition	Percentage of Patients in C.C.U.	Approximate Hospital Mortality
I	Absence of rales over the lung fields and absence of S_3	30 – 40	0 – 5
II	Rales over 50% or less of the lung fields or presence of S_3	30 – 50	10 – 20
III	Rales over more than 50% of the lung fields (frequently pulmonary oedema)	5 – 10	35 – 45
IV	Shock	10	85 – 95

Patients presenting with left ventricular failure should be ideally treated so as to reduce pre-load and after-load and augment inotropic state. Pre-load can be reduced by employing either diuretics[16] orally or parenterally, or nitrates used orally, intravenously or transdermally.[17,18] After-load can be decreased by using arteriolar dilators orally or intravenously e.g. Hydralazine, Nitroprusside[19] etc. Blood pressure is to be monitored for any unwarranted fall. Positive inotropic support is offered with guard as it may increase infarct size: Dopamine, Dobutamine or Digoxin are effective therapeutic agents.

Patients with cardiac damage more than 40% may present with cardiogenic shock. Such patients should ideally be monitored, using Swan Ganz catheter to measure left ventricular filling pressure indirectly and cardiac output by thermodilution.[20] Sympathomimetic amines are used to provide inotropic support and maintain systemic arterial pressure: Dopamine and Dobutamine[21], [22, 23] infusion form the mainstay of such therapy. To decrease left heart filling pressure (pre-load), diuretics[16] or nitrates[17,18] may be used. Intra-aortic balloon counter-pulsation[24] mechanically assists circulation by inflating balloon in diastole and deflating in systole. It has been proved to be of great help in such a situation.

POOR PROGNOSTIC FACTORS

- ☐ *Persistent cardiac failure*
- ☐ *Cardiomegaly*
- ☐ *Persistent sinus tachycardia > 100 beats/min.*
- ☐ *Transient atrial fibrillation*
- ☐ *Late 'malignant' ventricular arrhythmia*
- ☐ *Low blood pressure < 90 m.m.Hg.*
- ☐ *New bundle branch block*

Mechanical problems as rupture of mitral valve or intra-ventricular septum[25,26] require close monitoring and surgery at appropriate time.

Right ventricular infarction is seen commonly with inferior wall infarction[27] presenting as hypotension and low cardiac output: unlike in left ventricular infarction; plasma expanders are employed to increase pre-load and hence cardiac output. Arteriolar dilators are used to reduce right ventricular outflow impedance and reduce left heart filling pressure and pulmonary artery pressure, thereby increasing right ventricular cardiac output.

4. Limitation of Infarct Size

Myocardial infarct size is the most important prognostic factor.[28] Development of infarction is a dynamic process passing through a stage of reversible ischaemia before having rendered as irreversibly necrotic. Any physiological or pharmacological intervention to decrease oxygen metabolic demand or improve perfusion of jeopardized myocardium may help in limiting infarct size.

Reduction in Oxygen Metabolic Demand

1. *Beta Adrenoceptor Blocking Drugs.* A.M.I. is associated with reflex sympathetic stimulation,[29] which can be offset by blockade of beta adrenergic neurone receptors.[30] Due to negative inotropic and chronotropic effects, it decreases heart rate, blood pressure, cardiac index, stroke index and tension time index, hence reducing some of the major determinants of myocardial oxygen consumption.[31,32] Beta blockers improve metabolism of ischaemic myocardium[32] and possess anti-dysrhythmic activity.[33] Objective evidence in support of their effect on limiting infarct size, when used early in post-infarct period, has been reported by many investigators using various parameters for infarct size measurement such as electrocardiographic mapping, creatine–kinase (M.B.) peak and cumulative enzyme release.[34] When used in patients with suspected A.M.I. within four hours of onset of symptoms, beta blockers show significant decrease in chest pain experienced and myocardial infarct evolution.[35]

2. *Vasodilators.* Vasodilators reduce left ventricular end-diastolic pressure (LVEDP) and volume, resulting in a fall in left ventricular wall tension. This results in decrease of myocardial oxygen demand hence containing infarct size. Nitroglycerine[36,37] administered intravenously for 48 hours in prospective randomized trials has shown reduction of enzyme release, reduction in ST segment and preservation of 'R' wave and calculated infarct size. Serious ventricular arrhythmias were abated in patients with L.V.F. Frequent and large doses of sublingual nitroglycerine[38,39] used within four hours of chest pain showed electrocardiographic reduction of myocardial infarct size.

3. *Calcium Antagonists.* Due to vasodilatory effect and inhibition of intracellular calcium accumulation, calcium antagonists should theoretically limit infarct size. Earlier reports are encouraging but many more studies are being conducted and results awaited.[40]

4. *Glucose-Insulin-Potassium.* Administration of a solution of Glucose 300 gm, Insulin 50 units and Potassium chloride 80 mg in one litre administered 1.5 ml/kg/hour has been shown to improve haemodynamics and reduce ventricular arrhythmias.[41] However further studies are required to see the effect on infarct size and mortality.

5. *Corticosteroids.* Methyl prednisolone given intravenously causes persistent creatine elevation, higher incidence of ventricular rupture and mortality.[42] This could be due to inhibitory effect on healing.

Increase in Oxygen Perfusion

Myocardial infarction is a consequence of obstruction in one of coronary arteries blood flow. Reperfusion by any technique improves haemodynamics and limits myocardial infarct size. The benefit derived is directly proportional to rapidity with which it is accomplished.

1. *Surgical reperfusion* can be carried out by coronary artery by-pass grafting in post-A.M.I. with low mortality.[43] Indeed such an intervention is contraindicated four hours after the onset of chest pain, thereby severely limiting this technique.[44] Percutaneous transluminal coronary angioplasty can be employed for reperfusion, however time factor, expertise and availability of facilities limit its widespread use.

2. *Thrombolytics* like Urokinase, Streptokinase, when administered in coronary arteries, can restore angiographic patency in 70–80% of patients;[45,46] whereas given intravenously in large doses, these are successful in 50% of cases.[47] The procedure can cause serious arrhythmia and allergic reaction in some cases.[48] Inspite of apparent success, many issues remain unanswered: like interpretation of electrocardiographic changes and enzyme release, effect on survival, optimal course of treatment after reperfusion, establishing diagnosis of infarction before the procedure etc. Till these issues are resolved and measures are undertaken for prompt transferal of patients to the hospital, this technique will remain confined to a few centres.

5. Secondary Prevention

Secondary prevention of infarction and sudden death primarily revolves around reduction of multiple risk factors. Many controlled randomized trials have shown reduction in long term mortality in patients who undergo regular exercise and rehabilitation programmes.[49,50] Every effort should be made to make them give up smoking, as those who continue to smoke have twice the mortality of those who stop[51] (Table V). Weight reduction positively contributes in reducing other risk factors. High serum cholesterol with an increase in low density lipoproteins accelerates atherosclerosis and should be reduced by dietary measures or failing that by using therapeutic agents. Adequate control of hypertension improves left ventricular function and retards disease progression. Platelets inhibiting drugs,[52] Aspirin, Persantin and Sulphinpyrazone, and anticoagulants[53] all have been used in secondary prevention with varying results.

TABLE V EFFECT OF SMOKING STATUS ON MORTALITY AFTER 5 YEARS FROM CORONARY HEART DISEASE (CHD) AND TOTAL MORTALITY

Cigarette Smoking Status	No. of Cases	CHD Deaths		Total Deaths	
		No.	%	No.	%
Stopped	89	11	12.4	13	14.6
Reduced	42	6	14.3	6	14.3
Continued	59	17	28.8	17	28.8
Total	190	34	17.9	36	18.9

Role of beta blockers in secondary prevention has been the main focus of medical research in recent past. Most of the deaths in post-infarction period are due to either arrhythmia or re-infarction. Clinical interest in the use of beta blockers is centred on their potential to reduce infarct size and abort arrhythmia. Some of the recent, placebo controlled, prospective long term trials employing large population have provided ample statistical evidence in support of significant fall in total mortality, cardiovascular mortality, including sudden and non-sudden cardiac deaths, and on the incidence of non-fatal re-infarction. Drugs which have been used in such a setting are Timolol,[54] Propranolol,[55] Metoprolol,[56] Practolol,[57] Alprenolol[58] and Atenolol.[35] Obstructive lung disease, hypotension, bradycardia, A–V block and left ventricular failure are obvious contraindications. It has been recommended that treatment should be instituted in immediate post-infarction period, be continued for long time (18–24 months) and not stopped abruptly. Ancillary pharmacological properties probably do not have any significant bearing on the long terms results.

Patients experiencing post-infarction angina or who develop left ventricular aneurysm or left ventricular failure need objective assessment in the form of exercise tolerance test, radio-nuclide exercise test and left heart catheterisation with coronary angiography with a view to subjection to by-pass grafting[59] and appropriate surgery if required.

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