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ORIGINALS AND PAPERS

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100 Patients of Acute Myocardial Infarction Treated in an Intensive Coronary Care Unit

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Intensive Coronary Care Units have achieved a significant reduction in the mortality rate of acute myocardial infarction (Brown *et al.*, 1963; Day, 1965; Killip and Kimball, 1965-66; Goble *et al.*, 1966; Lown *et al.*, 1967a; Datey *et al.*, 1968). This has been possible by the prevention and correction of arrhythmias and the early detection and treatment of congestive cardiac failure and shock.

Our experience in the treatment of the first 100 patients of acute myocardial infarction in such a unit is presented.

Material and Method

Patients were admitted serially to the Coronary Care Unit (C.C.U.) at the K. E. M. Hospital, Bombay. There was no deliberate selection, but usually critically ill patients were referred.

100 patients of acute myocardial infarction were admitted, 19 of these were transferred from other medical wards because of complications.

Analgesics, oxygen and sedatives were used freely. Arrhythmias were detected early and treated promptly. Ventricular premature beats (V.P.B.) and ventricular tachycardia were treated with xylocaine and/or quinidine as indicated by one or more of the following conditions:

(a) V.P.B. frequency 5/min. or runs of two or more (b) Multifocal V.P.B.s. (c) QR^1/QT interval 0·85, QR^1 being the interval between Q of the normal and R of the following ectopic beat.

50 to 100 mg. (1 to 2 mg./kg. of the body weight) of xylocaine was given as an initial intravenous bolus and if found effective, an infusion

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of 1-4 mg./min. was maintained. When necessary, quinidine (200 mg. 6 hourly) was used.

Ventricular fibrillation was promptly treated with external cardiac massage, artificial respiration and when necessary by one or more countershocks (50 to 400 J). The resulting metabolic acidosis was treated with 100-400 ml. of 7·5 per cent sodium bicarbonate.

Sinus bradycardia was treated with intramuscular or intravenous atropine so as to maintain the heart rate at about 80/min. First and second degree A-V blocks were treated with corticosteroids (40 mg. or more of prednisolone daily), atropine and oral isoprenaline. High grade A-V blocks were treated with isoprenaline infusions (0·5 mg. per cent) at about 15 drops per minute and when necessary by transvenous catheter pacing.

Ventricular asystole was treated with external cardiac massage and artificial respiration. ½ to 1 ml. of 1: 1000 intracardiac adrenaline was given. 10 ml. of 10 per cent calcium gluconate was given intravenously. Acidosis was treated by 100-400 ml. of 7·5 per cent sodium bicarbonate.

Cardiac failure was treated with diuretics and digitalis. Diuretics were also used in cases where the urine output was diminished even when overt signs of cardiac failure were not present.

Shock was treated by one or more drugs after evaluation of several parameters, viz., blood pressure, urinary output and central venous pressure. The drugs used consisted of one or more of the following, viz., beta stimulators (0·5 mg. per cent isoproterenol infusion), alpha stimulators (noradrenaline), alpha blockers (25 mg. chlorpromazine), corticosteroids 1-2 g. daily, digitalis and diuretics.

Results and Discussion

Age, sex and period of monitoring—There were 90 males and 10 females. Their ages ranged from 34 to 80 years with a mean of 54 years. The average period of monitoring was 5½ days.

Site of infarction—54 had anterior, 44 posterior and 2 had a double infarction.

Rapidity of admission—The interval between the onset of symptoms and admission to hospital is shown in Table I. 64 per cent of patients were admitted within 12 hours of the onset of symptoms.

Table I—Showing the Interval between Onset of Symptoms and Admission

Interval	Percentage of cases
≤6 hours	44
6 to ≤12 hours ...	20
12 to ≤24 hours ...	8
24 to ≤48 hours ...	7
48 hours and more	21

Most of the patients admitted after 48 hours were transferred to the C.C.U. because of complications.

Severity of infarction—The mortality in the acute stage depends upon the severity of infarction. The severity should be graded by standard criteria so that the data in different series could be compared with respect to mortality, treatment and prognosis.

Table 2 shows the distribution of patients according to the severity of infarction as evaluated by the coronary prognostic index of Peel *et al.* (1962). More than half the patients had moderate to severe myocardial infarction.

Table 2—Showing the Distribution and Mortality

	Peel *et al.*'s (1962) index			
	1-8	9-12	13-16	17 and more
Pre-coronary care era (Peel *et al.*, 1962)				
Distribution %	22·2	38·0	22·4	17·4
Mortality %	4·5	12·5	23·4	64·1
Coronary care era (Daley *et al.*, 1968)				
Distribution %	16·0	28·0	24·0	32·0
Mortality %	—	—	25·0	42·0
Coronary care era (Lown *et al.*, 1967b)				
Distribution %	22·1	26·2	25·5	26·2
Mortality %	1·6	10·8	14·9	42·1

Mortality—The total mortality was 19 per cent. Table 2 shows the mortality in different severity groups. The mortality between the pre-coronary care era (Peel *et al.*, *loc. cit*) and the post coronary care era at the C.C.U. at Peter Bent Brigham Hospital (Lown *et al.*, 1967b) is compared with our own.

Table 3—Showing the Results of Resuscitation

Peel *et al.*'s (1962) index	No. of cases	Number resuscitated	Temporary only	Left hospital alive	Success rate (%)
1—8 ...	4	—	—	4	100
9—12 ...	—	—	—	—	—
13—16 ...	6	2	1	1	16
17 and above	12	7	5	—	—
Total ...	22	9	6	5	23

Resuscitation—Resuscitative measures were applied to both groups of patients, viz., those deve-

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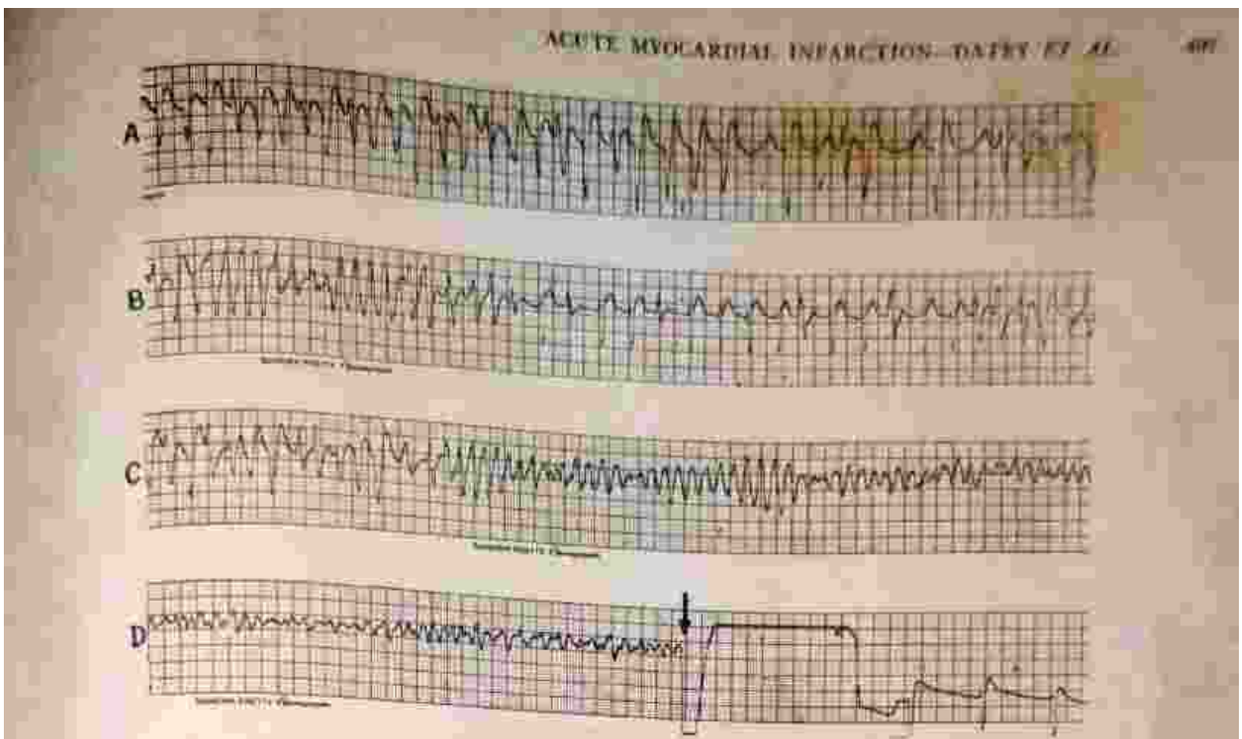

Fig. 1.—R. R., Male, 45 Years, Acute Myocardial Infarction. Continuous Strip of ECG showing (A) Multiple Ventricular Ectopic Beats, (B) Short Runs of Ventricular Tachycardia, (C) Ventricular Tachycardia Leading to Flutter and Fibrillation, (D) Ventricular Fibrillation being Terminated by 75 J Countershock (Arrow Shows Delivery of Shock)

loping "sudden" cardiac arrest or "primary electrical failure" due to serious arrhythmias and those with electrical failure secondary to cardiac decompensation or "pump failure" (congestive cardiac failure, shock and pulmonary oedema). The results in the former group were very gratifying whereas those in the latter, though initially encouraging, were eventually unsuccessful. Resuscitation in this group of patients is usually not compatible with long term survival (Nachlas and Miller, 1965; Goble *et al., loc. cit.*; Datey *et al., loc. cit.*). As shown in Table 3 though resuscitation was successful in 57 per cent of the cases, only 23 per cent survived to leave the hospital. All 5 cases of "primary electrical failure" who survived (left the hospital alive) had ventricular fibrillation. A representative case is shown in Fig. 1. No case of ventricular asystole survived. More or less similar results have been shown by other workers (Nachlas and Miller, *loc. cit.*; Goble *et al., loc. cit.*).

Resuscitation

Arrhythmias—84 per cent of patients had arrhythmia. Table 4 shows the incidence and mortality in patients who had major arrhythmias. Several patients had more than one arrhythmia.

Xylocaine was found to be safe and effective in 11 patients; however, it failed to control ectopic beats in 2 patients who later responded to quinidine.

Table 4—Showing the Incidence of Major Arrhythmias

	Primary		Secondary	
	No. of cases	Mortality rate (%)	No. of cases	Mortality rate (%)
Ventricular tachycardia	8	12	4	50
Complete heart block	5	20	5	80
Second degree A-V block	8	—	2	50
Sinus bradycardia	11	10	1	100
Multiple, multifocal V.P.B.s	25	4	3	33

12 patients with sinus bradycardia were treated with atropine, but the heart rate failed to increase in 3 patients. Side effects were not rare, specially difficulty in evacuating the bladder. One patient developed paralytic ileus.

Worsening of A-V block can usually be prevented with drugs (corticosteroids, atropine and isoprenaline). However, should a high grade A-V block ensue, transvenous pacing seems to be the only safe and effective method for increasing the ventricular rate. A representative case is shown in Fig. 2. With the semi-floating pacing catheters which can be introduced percutaneously, there is no need for sophisticated fluoroscopic equipment.

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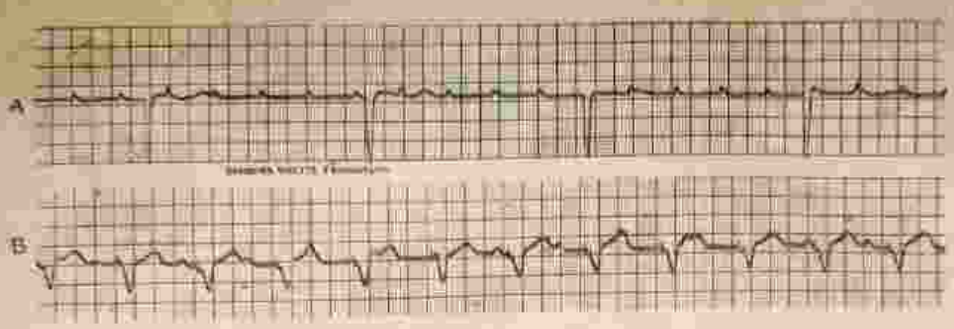

FIG. 2—S. B., FEMALE, 64 YEARS, ANTERIOR MYOCARDIAL INFARCTION WITH STOKES-ADAMS ATTACKS. ECG STRIP SHOWING (A) COMPLETE HEART BLOCK WITH ATRIAL RATE 150/MIN. AND VENTRICULAR RATE 27/MIN. (B) TRANSVENOUS PACING AT 75/MIN.; EACH QRS IS PRECEDED BY A PACEMAKER STIMULUS

To prevent "competition arrhythmias" the use of a "demand" pacemaker has been advocated.

Congestive cardiac failure—49 patients showed evidence of cardiac decompensation. Diuretics and digitalis were used. The mortality was 34 per cent. This high mortality is probably due to the severity of the infarction and to the extensive myocardial damage.

Gallop rhythm without any other evidence of decompensation was present in 8 patients. Only 1 of these died, the mortality being 12·5 per cent. When this is compared to the overall mortality of 19 per cent, we feel that gallop rhythm (unaccompanied by other signs of congestive cardiac failure) need not be considered as an ominous sign.

Diminished urine output was not an unusual finding even in the absence of other signs of cardiac decompensation. We considered this as early evidence of incipient cardiac failure and gave diuretics. In many cases the clinical improvement was perceptible and several episodes of overt cardiac decompensation were probably thus prevented (Fig. 3). In our opinion, the danger from the use of diuretics has been exaggerated. In case of marked diuresis, with replacement of potassium orally, hypokalaemia can be prevented and ventricular arrhythmia is unlikely to be precipitated. In our patients, even with a systolic blood pressure of 100 mm. Hg, the B.P. increased instead of falling after the diuresis. None of these patients developed thromboembolic phenomenon and we feel that the danger of increased blood viscosity and liability to thrombosis is probably only a theoretical consideration.

Shock—Shock was considered present with a systolic B.P. of less than 90 mm. Hg accompanied by oliguria and other signs of peripheral vasoconstriction. Table 5 shows the incidence and mortality in cases of shock.

The mortality due to shock is itself alarmingly high and when associated with congestive cardiac failure it becomes higher still.

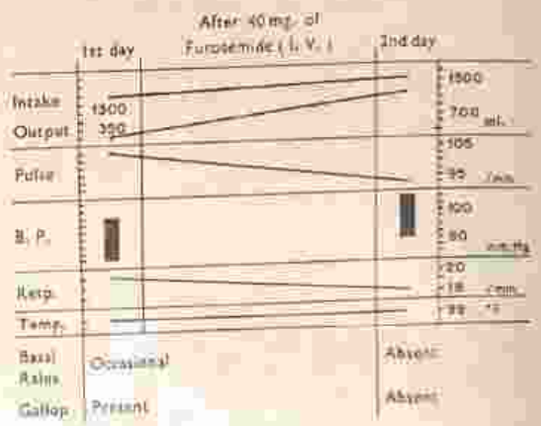

FIG. 3—B. P., MALE, 50 YEARS, ACUTE ANTERIOR MYOCARDIAL INFARCTION, SHOWING IMPROVEMENT FOLLOWING 40 MG. OF I.V. FRUSEMIDE IN SEVERAL PARAMETERS (URINE, PULSE, B. P., RESPIRATION, BASAL RALES AND GALLOP)

TABLE 5—SHOWING THE INCIDENCE OF SHOCK AND MORTALITY THEREIN

	No. of cases	Mortality rate (%)
Shock	10	60
Combination of congestive cardiac failure and shock	7	86

Late deaths—Of the 19 patients, 7 (35 per cent) died (Table 6) after the first 5 days, i.e., after transfer into the General Medical Ward. 2 of these died suddenly, presumably due to primary electrical failure. Some form of monitoring is therefore necessary for the first 2 to 3 weeks.

Future of coronary care units—The therapeutic focus has shifted from resuscitation of cardiac arrest to its prevention. With this, electrical failures will cease to take their toll of life. Power failure still remains the major hurdle to be surmounted. The possible avenues of research may be directed

Comments on Archive

Dr Ruchit Shah, Dr BR Bansode, Dr Nihar Mehta

Dear friends, it gives me immense pleasure to write a commentary on an article published way back in 1969. The importance of this write up is in understanding the milestones that have finely crafted the management of ST elevation myocardial infarction (STEMI).

Article summary

This article was published in JIMA in 1969. It speaks about the management of 100 patients of Acute Myocardial Infarction (AMI) at KEM hospital, Bombay. These patients were admitted in a coronary care unit (CCU) and administered analgesics, oxygen and sedatives. Ventricular arrhythmias were treated with xylocaine, quinidine and cardioversion. Sinus bradycardia was treated with intravenous or intramuscular atropine. First and second degree atrioventricular (AV) blocks were given oral corticosteroids, injectable atropine and isoprenaline. Temporary pacing was done for high grade AV blocks at bedside with semi floating catheter without fluoroscopy. Ventricular asystole was treated with external cardiac massage, artificial respiration, intracardiac adrenaline, intravenous calcium gluconate and intravenous sodium bicarbonate for acidosis. Cardiac failure was monitored clinically with auscultation of gallop rhythm, crepitations and decreased urine output. Digitalis and diuretics were given to treat it. Shock patients underwent blood pressure, urine output and central venous pressure (CVP) monitoring. Shock was treated with isoproterenol infusion, noradrenaline, digitalis, diuretics, oral chlorpromazine and corticosteroids. They predicted that future in management would be in monitoring myocardial contractility by mechanical and biochemical means. Arrhythmia prone patients would be managed by radio telemetry. Research should be focussed on newer drugs, mechanical and surgical procedures to manage power failure.

Evolution of AMI (Table 1)

Phase 1 (1912-61) Bed rest and expectant treatment - Initially, patients were offered bed rest for six weeks which was reduced to five days and hospitalisation reduced to one month. Drugs were administered as a supportive measure. Arrhythmias were detected by clinical auscultation. Most of the deaths were due to arrhythmias.

Phase 2 (1961-75) Coronary care units (CCU) Patients underwent cardiac monitoring with ECG monitors, with round the clock staff available for cardiac resuscitation. Even ambulances were equipped with pre hospital resuscitation

The article above crisply describes every aspect of management in CCU. The focus was on mortality reduction due to arrhythmias. A lot of morbidity and mortality was due to pump failure. Hence the next phase focused on salvaging myocardium.

Phase 3 (1975 till date) Myocardial reperfusion This phase marks a metamorphosis in the management of STEMI as compared to the previous phase. There are striking differences as compared to the nostalgic article.

- 12 lead ECG, which is a century old modality still remains the gold standard for diagnosing STEMI.
- ECG monitoring with defibrillation when required is the norm since the CCU era.
- Previously Oxygen supplement was given routinely. Now it is advised only if SaO2<90% or PaO2<60%. Opioid analgesics or benzodiazepines may be given to alleviate pain and anxiety.
- Pre hospital care In the CCU era, staff was trained in pre hospital resuscitation. Cardiac ambulances are now equipped with ECG and radio telemetry as rightly predicted by the authors in 1969. Today, trained staff can correctly identify STEMI; administer pre hospital thrombolysis and defibrillation if needed.
- Revascularization (Table 2) In the CCU phase, there was no option of revascularization.

With focus on restoring the flow of infarct related artery, primary PCI emerged as the therapy of choice (balloon angioplasty bare metal stents new generation drug eluting stents). It was observed that the earlier the infarct related artery is revascularised, more the mortality benefit. Thus, came the saying “time is muscle”. Attempts were made to reduce time delays by reducing symptom onset/ first medical contact (FMC)/ STEMI diagnosis to revascularisation time. In a country like India, with multiple logistic issues and non-uniform availability of the cathlab; we have developed STEMI systems of care. This system involves extensive training of staff, standardised protocols, improvement of emergency services and developing a network of the hub and spoke model. The hub and spoke model of India involves multiple ambulances with trained personnel. These personnel take ECG at first medical contact and transmit it for interpretation via radio telemetry/ smartphones to the hub. If the patient is diagnosed as STEMI, he is transferred to pre designated hospitals for treatment. If primary PCI is feasible in the designated time, he undergoes the procedure; else he is fibrinolysed and moved from spoke to hub for early PCI. The cost of STEMI treatment is a huge expenditure on the family especially when it is out of pocket. The present government insurance schemes are a big relief to patients.

- Hospital logistics Contrary to the CCU era, early ambulation (≤24 hours) and early discharge (≤72hours) is done in uncomplicated cases.
- Imaging - There was no luxury of imaging in the CCU era. Presently, echocardiography is used to assess resting left/ right ventricle function and mechanical

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complications. A follow up echocardiography can be done at 6-12 weeks after the STEMI. Cardiac MRI, SPECT, PET are also available if further information is desired.

- Cardiac biomarkers Biomarkers to detect cardiac injury have been in use since more than 50-60 years. Aspartate transaminase was the first clinically used biomarker in 1954. Then came creatine kinase, lactate dehydrogenase and CK-MB. They were not specific. Presently, quantitative serial high sensitive troponin testing is used to determine infarct size and prognosis.
- Pharmacotherapy Intensive pharmacotherapy in the form of high dose statins, beta blockers, ACE inhibitors, ARB (in ACE inhibitor intolerant patients) and mineralocorticoid receptor antagonists (MRA) have mortality benefits. These classes of drugs were not available in the CCU era.
- Arrhythmias The management of atrial and ventricular arrhythmias in the CCU era was primitive. Apart from temporary pacing for high grade AV blocks and cardioversion for ventricular arrhythmias, everything has been revolutionised.

i) Atrial fibrillation (AF) Intravenous beta blockers, amiodarone and electrical cardioversion is used. Intravenous digitalis is used only if there is associated heart failure and hypotension. In patients with long term AF, CHADS2VaSc score is calculated and oral anticoagulation is considered.

ii) Ventricular arrhythmias Repeated electrical cardioversion, complete revascularisation and correction of electrolytes is the most important. Beta blockers, amiodarone and overdrive pacing can also be done. In case of an electrical storm, intubation with deep sedation and radiofrequency ablation can be done. Implantable cardioverter defibrillator is given to patients at a high risk of sudden cardiac arrest.

iii) AV blocks Urgent PCI, temporary pacemaker and positive chronotropic agents (atropine, epinephrine, vasopressine) are given.

- Heart failure In the CCU era, heart failure was managed clinically with diuretics and digitalis. Now bedside echocardiography and invasive hemodynamic monitoring has revolutionised the management heart failure. Loop diuretics and nitrates are used for symptomatic management. ACE inhibitors, ARB (in ACE intolerant), beta blockers and MRA have mortality benefit in patients with LVEF≤40% and/or heart failure. Oxygen and mechanical ventilation is done if required.
- Cardiogenic shock - In the CCU era, shock patients underwent clinical and CVP monitoring. They were managed conservatively. Today, doppler echocardiography and invasive hemodynamic monitoring is available. Immediate PCI is done if anatomy is suitable. If mechanical complications are there, heart team discussion followed by emergency CABG/ hybrid procedure is done. Inotropes, intra-aortic balloon pump, short term mechanical circulatory support, ultrafiltration and mechanical ventilation can be lifesaving.
- Cardiac arrest Now, primary PCI gives an added chance of survival. If patient remains unresponsive after resuscitation, targeted temperature management (32-36 degrees C) is strongly recommended.

Phase 4 (The future) The future lies in prevention of lethal myocardial injury and use of biologicals.

Mortality

As science progressed and technology evolved, we saw a drop in mortality over the decades. The mortality rate was 30% (1912-61), 15% (1961-75) which reduced to 3-8% in the present day. A Danish cohort studied the mortality risk in acute myocardial infarction patients less than 50 years old between 1980-2009. The 30 day mortality was 12.5%. 8.4% and 3.2 % in 1980-89, 90-99, 2000-09 respectively.

Summary

We have seen a paradigm shift in the management of AMI since the above article was published. The authors had rightly predicted that arrhythmias could be monitored by radio telemetry and the future research should be focussed on newer drugs and better procedures. Primary PCI, systems of care for STEMI, aggressive CCU management, drugs with trials for mortality benefit, better thrombolytics, heart team approach and mechanical circulatory devices have helped to significantly reduce the mortality associated with AMI.