CARDIAC ARRHYTHMIAS IN ACUTE MYOCARDIAL INFARCTION

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INTRODUCTION

THE reduction in mortality in acute myocardial infarction from 30-35% to 20-25% is attributed to the early recognition and treatment of cardiac arrhythmias in the early phases of the acute coronary event (Kimball and Killip, 1968; Pantridge *et al.*, 1975). The more frequent use of continuous electrocardiographic monitoring, artificial cardiac pacing and electrical cardioversion has contributed in this reduction in mortality. In addition, there is now a better understanding of life-threatening arrhythmias and its role in the genesis or aggravation of cardiac failure and shock. In contrast, mortality from cardiogenic shock in acute myocardial infarction has however not changed despite modern facilities and drugs.

It is common to develop one or more types of arrhythmias following acute myocardial infarction. An incidence of 75%-95% of patients has been reported to develop some disorder of rate, rhythm or conduction as a complication (Julian *et al.*, 1964; Meltzer and Kitchell, 1966; Hurwitz and Eliot, 1964). Time lapse between onset of symptoms and hospital admission and the duration of monitoring are two important factors that influence arrhythmia detection.

This report is a prospective study of 164 patients with acute myocardial infarction seen between October 1977 and July 1979 in the Universiti Kebangsaan Division of the Coronary Care Unit, General Hospital, Kuala Lumpur. By continuous electrocardiographic monitoring for 24 hours, we report our experience of the incidence of the various types of arrhythmias in acute myocardial infarction in the local population.

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PATIENTS AND METHOD

All patients admitted with history and symptoms suggestive of acute myocardial infarction were included in the study. No selection was made regarding age, sex, race and severity of illness. The diagnosis was established by a) typical clinical history b) electrocardiographic abnormalities consisting of either new Q waves or typical evolutionary ST-T changes c) serum glutamate oxaloacetate transaminase rise of at least twice normal. Patients not fulfilling the above criteria and re-admissions were excluded from the study. Every patient was seen by one of us within 12 hours of admission. A standard 12 lead electrocardiogram was done on admission and the patient was started on continuous electrocardiographic monitoring without delay.

All arrhythmias detected on the monitors were noted and where possible, recorded. Deaths and complications of cardiac failure and shock occurring in the coronary care unit were documented. Cardiac failure was diagnosed in the presence of a) raised jugular venous pressure b) crepitations in the lung bases c) chest X-ray changes consistent with cardiac failure. Shock was diagnosed when the systolic blood pressure was less than 90mm. of Hg. in the presence of peripheral cyanosis, cold and sweaty skin and oliguria. Patients with cardiogenic shock and cardiac failure were classified as shock.

Monitoring system: Continuous electrocardiographic monitoring was obtained using the Hewlett Packard Model 78220 A/B Arrhythmia Monitoring System. Each bedside monitor was

interfaced to the central console in the nursing station allowing arrhythmia detection from both places. An alarm system sensitive to changes in heart rates, ectopics and life-threatening arrhythmias permits direct recording during appropriate events. The system in addition could display, on request, of a patient's heart rate and ectopics trends over the past 9 hours thus permitting evaluation of preceeding events and response to treatment.

RESULTS

There were 146 men (89%) and 18 women (11%) who fulfilled the criteria in the study. Table I shows the distribution of the 164 cases by race and sex. The age range was 26 years to 81 years, with a mean of 53.5 years.

Site of Infarction, Complications and Deaths: The site of infarction and its frequency by sex is shown in Table II. In this study, there were more anterior (69.8%) than inferior (40.2%) infarctions. In 120 patients (73.2%) no complications of cardiac failure and cardiogenic shock developed. These complications were more frequent with anterior (30.6%) than inferior (21.2%) infarctions (Table III). There were 16 deaths (9.8%), 11 patients with anterior and 5 with inferior infarctions, within 24 hours of admission. Of the 8 patients who developed cardiogenic shock, 5 died within 24 hours. There were 6 deaths (16.7%) out of the 36 patients who developed cardiac failure on admission. Refractory ventricular tachycardia and/or ventricular fibrillation and complete heart block were the immediate causes of death in the other 5 patients. This relatively low incidence of deaths seen with this series was because deaths occurring after 24 hours were excluded from the study.

Arrhythmias: 132 of the 164 patients (80.5%) monitored developed some disorder of rate, rhythm or conduction (Table IV). The incidence of arrhythmias varied with the clinical status of the patients. All patients with cardiogenic shock, 94.4% of patients with cardiac failure and 75% of patients with no complications developed arrhythmias. The occurrence of arrhythmias in relation to the site of infarction is shown in Table V.

Supraventricular Arrhythmias occurred in 54.8% of patients. Sinus bradycardia was more common in inferior infarctions whereas supraventricular tachycardia was seen more frequently in anterior infarctions. Bradycardia arising from sinus bradycardia, second degree heart block and complete heart block was seen in 23.2% of the patients. This was more common in inferior (13.5%) than anterior (9.7%) infarctions. Premature atrial contractions, atrial flutter and atrial fibrillation were less frequent, usually transient and did not require specific treatment.

Ventricular Arrhythmias was seen in 34.7% of patients. Unifocal premature ventricular contractions was the most common type seen (23.2%). Treatment of premature ventricular contractions with Lignocaine (Xylocard), either in a bolus injection or as a continuous infusion, was

Table I
Racial distribution of 164 Patients

Race	Sex				Total	
	Male		Female			
	No.	%	No.	%	No.	%
Malay	52	31.7	8	4.9	60	36.6
Chinese	34	20.7	6	3.7	40	24.4
Indian	58	35.4	4	2.4	62	37.8
Others	2	1.2	0	0	2	1.2
Total	146	89.0	18	11.0	164	100.

Table II
Distribution of patients by sex and site of infarction

Site of Infarction	Sex of Patient				Total	
	Male		Female			
	No.	%	No.	%	No.	%
antero-septal	62	37.8	10	6.1	72	43.9
Extensive Anterior	20	12.2	6	3.7	26	15.9
Inferior	64	39.0	2	1.2	66	40.2
Total	146	89.0	18	11.0	164	100

Table III

Incidence of Complications in relation to site of Infarction

Clinical Status of Patient	Site of Infarction			Total	
	Antero-septal	Ext. Anterior	Inferior	No.	%
No Cardiac Failure	50(0)	18(3)	52(2)	120	73.2
Cardiac Failure	18(3)	6(2)	12(1)	36	22.0
Cardiogenic Shock	4(1)	2(2)	2(2)	8	4.8
Total	72(4)	26(7)	66(5)	164	100

( ) Figure in brackets indicate number of deaths.

Table IV

Incidence of Arrhythmias [all types] in relation to the Clinical Status of the Patient

Clinical Status of Patient	Total Number of Patients	Patients with Arrhythmias	
		No.	%
No Cardiac Failure	120	90	75
Cardiac Failure	36	34	94.4
Cardiogenic Shock	8	8	100
Total	164	132	80.5

usually satisfactory. Mexiletene (Mexitil) was used when an unsatisfactory response was obtained. Ventricular tachycardia or fibrillation was seen infrequently (3.6%) in anterior infarctions only. Of the 6 patients who developed this arrhythmia, 2 showed it as a terminal event and was refractory to treatment.

Conduction Defects developed in 29.8% of the patients studied. It was more frequent in inferior infarctions (17.1%) than anterior infarctions (12.7%). 9 patients developed complete heart block, 3 of whom died.

DISCUSSION

It is now well recognised that arrhythmias of various types occur in 75%-95% of patients with acute myocardial infarction. In the pre-monitoring era, arrhythmias were considered to occur in only about 20% of patients with acute myocardial infarction, rather than the almost 100% incidence that is now reported (Master *et al.*, 1937). Early mortality in acute myocardial infarction which occurs within a few hours of the onset of symptoms is attributed to arrhythmias. Attempts to reduce this mortality rate should thus be directed to the early detection and treatment of arrhythmias. This became apparent after the widespread establishment of coronary care units with continuous electrocardiographic monitoring facilities. It is also recognised that the earlier the patient is admitted to the coronary care unit, the greater will be the incidence of arrhythmias detected. Certain arrhythmias are considered life-threatening which predispose to early deaths. They include severe bradycardia, advanced heart block, frequent ectopics, ventricular tachycardia and ventricular fibrillation. Treatment of arrhythmias in general serve to prevent this mortality and in addition prevents extension of infarct size and aggravation of cardiac failure.

Bradyarrhythmias are common after acute myocardial infarction (Jewitt *et al.*, 1967; Cristal *et al.*, 1975). The prevalence of bradycardia is dependent on the time monitoring is started. When seen within 30 minutes after infarction, Webb *et al.* (1972) found 77% of patients had bradycardia. Pantridge *et al.* (1975) in their series noted the incidence decreased to 34% when seen within 1 hour of symptoms. In this study, 23.2% of patients developed bradycardia and was more frequent in the inferior infarc-

tions. Delay in hospital admission is contributory to the low incidence. In our experience, the mean dealy in hospital admissions is 11.7 hours (Ng *et al.*, 1979). The significance of the bradyarrhythmias, sinus bradycardia or advanced heart blocks, is their association with an increased morbidity and mortality. This is attributed to hypotension with its adverse haemodynamic sequelae, enlargement of infarct size and causing electrical instability that may provoke genesis of escape rhythms such as ventricular tachycardia and ventricular fibrillation (Moroko *et al.*, 1971). Bradycardia is significantly more common in patients with inferior myocardial infarction than in those with infarctions of other sites (Adgey *et al.*, 1968; Grauer *et al.*, 1973). In the early phases of acute myocardial infarction, hypotension and electrical instability secondary to bradycardia frequently respond to an increase in heart rate. This is possible with atropine or the use of a pacemaker when the drug is ineffective.

Tachyarrhythmias in acute myocardial infarction are due to sinus tachycardia, paroxysmal atrial tachycardia, atrial flutter, atrial fibrillation or ventricular tachycardia and fibrillation. Sinus tachycardia is reported to occur in 20%-53% of patients with acute myocardial infarction (Julian *et al.*, 1964; Jewitt *et al.*, 1967). This arrhythmia can arise from anxiety, pain or extensive myocardial damage resulting in cardiac failure or shock. As a reflection of the extent of myocardial dysfunction, it is seen more frequently with anterior than inferior infarctions where the occurrence of such complications are higher. Mortality is therefore higher in these patients. With supraventricular tachycardia, paroxysmal atrial tachycardia and paroxysmal junctional tachycardia, the occurrence is infrequent. It is usually transient lasting from several munutes to a few hours. Atrial fibrillation and atrial flutter are uncommon. They arise from a concommitent infarction of the atrium or secondary to left ventricular failure. When associated with rapid ventricular rates, the prognosis may be worsened by the undesirable effects on cardiac output either precipitating or aggravating cardiac failure and also because severe cardiac damage is usually present. In 24 hours monitoring of the 164 patients in this study, sinus tachycardia was observed in 33 patients (20.1%). Less than 5% of patients developed atrial fibrillation (4.9%) and atrial flutter (2.4%). Ventricular tachycardia and ventricular fibrillation occur in 2%-6.4% of patients. Frequency of detection is dependent on delay in admission time. Their occurrence is increased in cases complicated by cardiac failure or shock and thus seen usually as a terminal event preceeding "cardiac arrest".

The incidence of developing premature ventricular contractions (all types) ranges from 58%-93% depending on interval from onset of symptoms and duration of monitoring (Pantridge *et al.*, 1975). In this study, 31.1% of the patients developed premature ventricular contractions. This low incidence in comparison to other reported series may be attributed to the short duration of monitoring. Non-detection of this arrhythmia by the observer is also contributory. Certain types of premature ventricular contractions are now identified as those that preceed ventricular tachycardia or ventricular fibrillation. They include; ectopic beats occurring greater than 5 per minute, multifocal nature of the beats, coupled beats or salvos (three or more in a row) and ectopics that show a "R on T" phenonmenon (Lown *et al.*, 1967). Suppression of these arrhythmias may prevent the high risk of developing ventricular tachycardia or fibrillation. This forms the basis for immediate or prophylactic anti-arrhythmic therapy in the attempt to reduce coronary care mortality. Prophylactic use of Mexiletene (Achuff *et al.*, 1977) and Disopyramide (Zainal *et al.*, 1976) have been shown to effectively reduce ventricular arrhythmias following myocardial infarction.

Continuous electrocardiographic monitoring has increased awareness to early arrhythmia detection with its clinical implications in acute myocardial infarction. Being an observer monitoring system, and despite well trained coronary care unit nurses, non-detection of warning arrhythmias occur. Vetter and Julian (1975) demonstrated in their study using a computer analysis system, the detection of 99% of potentially serious ventricular arrhythmias as compared to the detection of less than 50% of these arrhythmias in the conventional observer monitoring system. This limitation results in patients not receiving anti-arrhythmic therapy or delayed because of detection failure.

SUMMARY

Cardiac arrhythmias occur in the majority of patients after acute myocardial infarction. The incidence of arrhythmias is affected by the time delay before hospital admission and the duration of monitoring carried out. 164 patients with acute myocardial infarction are studied with reference to the types and incidence of arrhythmias occurring in 24 hours of continuous monitoring. The local experience is compared with other series and the significance of certain types of arrhythmias is discussed.

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