MEDICAL PRACTICE

Occasional Survey

Five hundred patients with myocardial infarction monitored within one hour of symptoms

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Abstract

Of 2886 patients monitored during acute myocardial infarction, 500 were observed within one hour of the onset of symptoms. Half of the early admission group were admitted in response to emergency 999 calls and 435 of them travelled in resuscitation ambulances, where surveillance for arrhythmias was instituted. Pulmonary oedema occurred in 130 patients (26%), cardiogenic shock supervened in 60 (12%), and 115 (23%) died in hospital. Ventricular fibrillation was observed in 98 patients (20%). Forty two of them survived to be discharged, including 20 of the 24 with primary fibrillation which had occurred first in hospital. In only one case did primary ventricular fibrillation occur after the first 10 hours of onset of illness. Sinus bradycardia, atrial fibrillation, ventricular tachycardia, and ventricular fibrillation were all observed more frequently in patients admitted within one hour after the onset of symptoms than in those admitted later.

An element of selection is inevitable when early admission is encouraged by the existence of a resuscitation ambulance system; this will depend in part on the early recognition of risk and the geographical location of the attack. These factors may bias the group towards relatively high risk. Nevertheless, prompt admission after myocardial infarction should improve survival by permitting successful management both of ventricular fibrillation and of other arrhythmias which may influence short term and long term prognosis.

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Introduction

Little information is available in world reports on the complications of myocardial infarction when patients come under observation very early after the onset of symptoms. A series representing an unselected population cannot be obtained because data are collected only from those who seek treatment. In 1971 Adgey and colleagues reported[1] on the incidence of arrhythmias among 284 patients observed within one hour in a mobile coronary care unit manned by medical staff and, at that time, intended to operate in response to calls from general practitioners. Selection may not necessarily be similar in a community encouraged to make use of the emergency (999) telephone system for patients with severe chest pain or collapse; the Belfast results may also have been influenced by the availability of medical skills.

Information on the results of early intervention is of particular value because of a resurgence of interest in prehospital care and early hospital admission. In Britain the Department of Health no longer actively discourages the creation of new coronary care and resuscitation ambulance programmes.[2,3] District health authorities considering setting up such schemes within financial constraints will wish to know the potential benefits of early intervention.

Taking advantage of data collected since the creation of an ambulance system[4-6] based on emergency services without direct medical intervention, we have reviewed retrospectively the records of 500 patients with confirmed myocardial infarction observed within 60 minutes of the onset of chest pain, with particular reference to complications and outcome.

Patients and methods

We included all patients with myocardial infarction admitted from early 1973 to the end of 1980, provided that they had been monitored either in hospital or in a resuscitation ambulance within one hour of the onset of symptoms. Patients were not considered if this interval could not be determined with reasonable accuracy. The duration of the retrospective survey was determined by our wish to recruit 500 patients. The diagnosis of myocardial infarction was confirmed on the basis of clinical presentation together with typical evolution of electrocardiographic changes or increase of the serum aspartate transaminase activity to greater than twice normal. Some patients carried in the ambulances were first seen in ventricular fibrillation and resuscitated. We included these only if the diagnosis of myocardial infarction could be substantiated, if the patients reached hospital alive, and if the time of onset of symptoms could be defined. We excluded patients who were resuscitated from sudden unheralded cardiac death and also the few from whom a history could not be obtained.

Arrhythmias occurring in the cardiac ambulances up to 1978 were recorded on magnetic tape for analysis in the cardiac care unit. We found 94% agreement between the ambulancemen's report and the registrar's interpretation.[5] During the last two years of the survey we therefore relied on a written report supported in nearly all instances by a paper record of any rhythm disorders.

Identification of rhythm disorders occurring within the cardiac care unit depended on conventional monitoring with rate alarms. An arrhythmia computer (Reynolds Medical Electronics)[7] was used only in selected patients. Thus some transient rhythm disorders which did not cause haemodynamic disturbance may have been missed.[8]

Continuous tape recordings of heart rhythm were available and were scrutinised if important disturbances were suspected but had not been observed directly. We document all rhythm disorders of clinical relevance by permanent paper records mounted chronologically in the patients' case notes. All but the most obtrusive arrhythmias are likely to be noticed more readily in the cardiac care unit than in a general ward. The median time that patients stayed in the cardiac care unit declined from 47 hours in 1973 to 34 hours in 1980. Those without complications are now discharged from hospital usually five days after admission[9]; a minority with persisting pulmonary oedema or arrhythmias remain for 10 days or more.

All surviving patients undergo portable chest radiography and have a 12 lead electrocardiogram recorded shortly after arrival in hospital. All films are checked by a consultant cardiologist; the presence or absence of pulmonary oedema is noted routinely on the admission form. Cardiogenic shock is defined clinically as a systolic blood pressure below 90 mm Hg together with evidence of peripheral vasoconstriction and oliguria. The Norris prognostic index[10] is used as a measure of severity in those patients who survive to undergo chest radiography.

Since 1973 we have recorded the precise times of all the important events relating to episodes of infarction for patients carried in the resuscitation ambulances or treated in the cardiac care unit. These times and relevant clinical details are collated and checked before entry on to a computer. The system was unchanged throughout the period of our study. The information presented here was also checked against original hospital records.

The technique of analysis of proportion was used to compare the incidence of arrhythmias occurring in the patients seen within one hour with that in the patients first seen later than one hour after the onset of symptoms.

Results

Over the seven years under review a total of 2886 patients were entered on to our computerised index with a discharge summary of acute myocardial infarction. Their mean age was 62 years (± SD 13) and the male to female ratio 2·7:1. The median delay between the onset of major symptoms and monitoring varied from 115 minutes in 1973 to 89 minutes in 1980.

In the subset of 500 patients known to have been monitored within one hour of the onset of the major symptom of myocardial infarction (usually pain) the mean age was 60 years (± SD 13). The male to female ratio (4:1) was higher than that of the total series. The resuscitation ambulances brought to hospital 435 (87%) of the patients in this subset. Emergency 999 calls prompted admission in 260 cases, and 190 (38%) were referred through general practitioners. Of the remaining 50 patients, most were self referrals to the accident and emergency department. The site of infarction was considered on electrocardiographic evidence to be predominantly anterior or lateral in 235 patients (47%), predominantly inferior or posterior in 225 (45%), and indeterminate in 40 (8%). This distribution did not differ appreciably from that in patients observed later than one hour, for whom the respective percentages were 46%, 43%, and 11%.

Within the early admission group 130 patients (26%) had pulmonary oedema at some stage during their hospital stay. This diagnosis was based on the chest radiographs or on characteristic clinical evidence in a few who did not survive to have radiography. Sixty patients with pulmonary oedema also had cardiogenic shock as defined by our clinical criteria; 19 of these (32%) survived.

A total of 98 of the 500 patients developed ventricular fibrillation either before or during hospital admission. Primary ventricular fibrillation occurring in the absence of cardiogenic shock or clinically apparent left ventricular failure was seen in 31 patients in the ambulance and 24 patients in hospital. In all but one patient primary ventricular fibrillation occurred within 10 hours of the onset of symptoms (fig 1); he sustained a cardiac arrest at 11 days, after admission had been prolonged for social reasons only. In his case prompt attempts at resuscitation failed. Necropsy was not carried out and a possibility of pulmonary embolism cannot be excluded. In 43 instances (nine in the ambulance and 34 in hospital) ventricular fibrillation supervened in those who already had evidence of left ventricular failure.

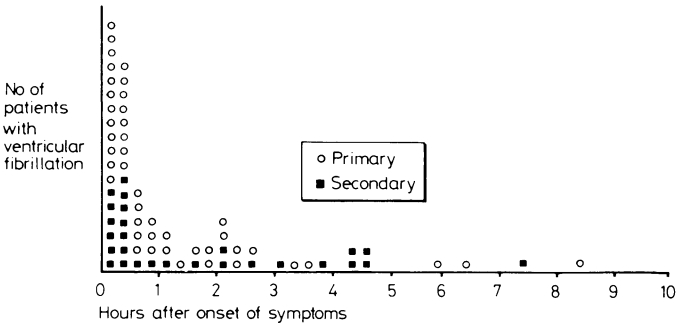

FIG 1—Incidence of ventricular fibrillation in first 10 hours of illness in 500 patients with acute myocardial infarction monitored within one hour of onset of symptoms. Further 26 cases of late secondary ventricular fibrillation and one of late primary ventricular fibrillation occurred outside time scale of graph.

Overall 42 of the 98 patients with ventricular fibrillation survived. Forty patients were resuscitated before admission to hospital, of whom eight developed the arrhythmia after the ambulance had arrived. Fifteen of these (38%) were eventually discharged from hospital. The survival rate from primary ventricular fibrillation (83% in hospital cases) was notably better than when the arrhythmia occurred in the setting of left ventricular failure; only seven patients (16%) in this group survived.

Information on ventricular tachycardia was less definite because episodes without serious haemodynamic disturbance may not have been detected. The arrhythmia was recognised in 121 patients, but 34 of these also had ventricular fibrillation at some stage. The course was not always benign in the remaining 87 patients even in the absence of ventricular fibrillation; 13 died in asystole or electromechanical dissociation, 12 of them after the development of pulmonary oedema or cardiogenic shock.

Complete atrioventricular block with an escape rhythm was seen in 21 patients in the early admission group. Inferior infarction was present in 17 patients, of whom 16 survived, and anterior infarction was present in three patients, of whom two survived. One other surviving patient had an indeterminate infarct site (left bundle branch block). Both patients who died were in cardiogenic shock and died despite pacing.

Twenty six patients developed asystole apparently as a primary event without prior observed tachycardia, defibrillation, or suppressant treatment. Four of these remained without a detectable cardiac output despite ventricular capture by pacing stimuli. Only two survived to leave hospital. Interestingly, none of the 22 patients with neither QRS complexes nor identifiable P waves regained a cardiac output despite full resuscitation attempts which included not only pacing but also the use of adrenaline and calcium chloride.

We compared the incidence of major disorders of rhythm in the patients observed in less than one hour after the onset of symptoms and those first observed after a longer delay (fig 2). Sinus bradycardia was significantly more common ($p < 0.001$) in the early group, as were

atrial fibrillation ($p < 0.01$), ventricular tachycardia ($p < 0.001$), and ventricular fibrillation ($p < 0.001$). There was little difference in the incidence of sinus tachycardia.

The overall mortality of patients observed within one hour of infarction was 23% (115 cases). The Norris prognostic index was recorded at the time of admission in 406 patients, and the table compares the expected and actual mortalities.

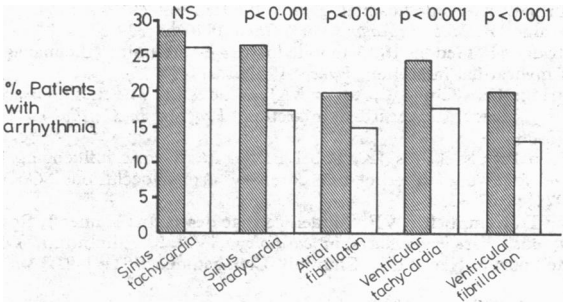

FIG 2—Comparison of incidence of major disorders of cardiac rhythm in patients with acute myocardial infarction observed in less than one hour after onset of symptoms (▨)—and those first observed after longer delay (□).

Mortality and coronary prognostic index. Risk categories and expected mortality are derived from the data of Norris et al[10]

Coronary prognostic index (Calculated risk (%))	No of patients	Expected mortality (No)	Actual mortality (No (%))
3	45	1	1 (2)
8	56	4	2 (4)
22	137	30	6 (4)
38	77	29	13 (17)
65	50	33	10 (20)
78	41	32	29 (71)
Not measurable	94*		54

*It was not possible to calculate the index in many of these patients who did not survive to undergo chest radiography.

Discussion

Much of the course of acute myocardial infarction remains obscure and cannot readily be defined accurately. Even mortality rates are conjectural. Community based surveys in Britain[11 12] and in the United States[13 14] suggest that about half of the victims die in the early weeks and that half of those deaths occur within an hour or two of the onset of symptoms. At that stage postmortem evidence of infarction is inconclusive and the condition may be indistinguishable from that of an unheralded fatal arrhythmia.[15] Complication rates are also unknown because many patients die before clinical observations are made. Moreover, of those who survive, over one fifth have had no recognised symptoms[16] and diagnosis can be made only retrospectively from electrocardiographic evidence; others who do have symptoms receive no specialised care.

The widespread concern that patients should be treated sooner after the onset of symptoms than at present seems well founded. Most early deaths are believed to be due to ventricular fibrillation,[17] which is potentially correctable. Life threatening complications such as cardiogenic shock may also occur less commonly in patients treated early.[1 18] Moreover, the pattern of complications in patients diagnosed and treated promptly may differ from that seen under more conventional arrangements for medical care.[18]

The 500 patients observed within an hour of the onset of major symptoms were not a representative sample of those suffering acute myocardial infarction. Most people with severe cardiac pain either are slow to recognise the importance of their symptoms or take an optimistic view of the outcome, which is not always justified.[1 18] Hence the tendency is for patients to die before medical help reaches them or to be treated after the period of greatest risk has passed. In Brighton resuscitation ambulances appreciably shorten the delay before monitored care can be given. For the majority who avail themselves of this service the median delay is less than 90 minutes.[5] This delay is unusually short, though comparable with that in Belfast.[18 19] Even in Brighton and Belfast, however, only a minority of patients are seen within an hour.

The relatively short delay for the patients in this series may have reflected not only their location away from home at the onset of their attacks but also an unusual severity of symptoms—including collapse leading to 999 calls. The proportion of patients with severe attacks was relatively high in the early admission subset, 22% of all our patients falling into the two most severe categories of the Norris prognostic index.[10] The "expected" mortality based on Norris's data was 129 of the 406 patients in our early admission group for whom sufficient data were available to generate a score, but the observed mortality was only 61 in this group. It might be suggested that early treatment improved the prognosis, but there was another factor. The Norris index places heavy weighting on the blood pressure on admission. Blood pressure tends to be low in the early stages of infarction, especially when it affects the inferior wall and is associated with bradycardia. Though prompt treatment doubtless helps such patients, many recover spontaneously. The index may therefore suggest an inappropriately poor prognosis if applied exclusively to patients admitted early.

The incidence of ventricular fibrillation and tachycardia in this series was similar to that observed by Adgey *et al.*[1] The timing of the occurrence of ventricular fibrillation shows that most of these fatal arrhythmias would not have occurred under monitored conditions had patients come under care even two hours after the onset of symptoms, which is still an early response by most standards. In the event, 42 of the 98 patients who had ventricular fibrillation survived. In hospital, primary ventricular fibrillation had an 83% survival after prompt resuscitation. Secondary ventricular fibrillation had a much worse prognosis.

Early admission can improve prognosis in the short and longer terms in a second way. Both survival and quality of life after infarction depend critically on the extent of myocardial necrosis, which may be influenced by drug treatment. Effective treatment of bradyarrhythmia and tachyarrhythmia is believed to limit the size of the infarct by a favourable influence on the balance between supply and demand for metabolic requirements.[20 21] Treatment could be started promptly for our patients observed within one hour of the onset of symptoms. Moreover, figure 2 shows that the proportion with arrhythmias was unusually high in this subset. Whether this reflects selection of a high risk group or the usual course of the early phase of the illness, important treatment could be provided for a higher than usual proportion of patients with infarction. The later the admission after the onset of symptoms the more likely it is that expensive specialised units will be used to provide accommodation rather than useful treatment.

Though the importance of early monitored care has been emphasised many times before,[1 19 20 22] our observations strengthen the belief that a new sense of urgency in arrangements for admission of patients with myocardial infarction is long overdue. The data will be of value to those who must weigh the advantages and the cost of any system designed to bring coronary care to patients during the period of greatest risk.

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Aviation Medicine

Problems of altitude

I: Hypoxia and hyperventilation

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"Even modern aircraft still expose their passengers to some risks from lowered atmospheric pressure."[1]

Concorde commonly cruises at altitudes of 50 000-60 000 ft (15 240-18 288 m) where, if unprotected, its occupants would be unconscious within 15 seconds and dead four to six minutes later from lack of oxygen.[2] Even at the more usual cruising altitudes of commercial aircraft—30 000-40 000 ft (9144-12 192 m)—unprotected passengers and crew would rapidly succumb. Of course this does not happen, thanks to the protection of the aircraft pressure cabin. The well being of the air traveller within this artificial environment is, however, always threatened by the problems of altitude extending beyond lack of oxygen to decompression sickness, cold, and simple pressure effects.

Physics of the atmosphere

Ascent to altitude is associated with a fall in air pressure parallel by decreases in density and temperature. Thus at 18 000 ft (5486 m) atmospheric pressure has fallen to half its value at sea level and the ambient temperature to about −20°C (fig 1). The fall in total atmospheric pressure and the consequent reduction in the partial pressure of oxygen (Po_2) poses the greatest single threat to anyone who flies, hypoxia. Fortunately, the relationship between oxygen saturation of haemoglobin and oxygen tension, reflected in the shape of the oxygen dissociation curve, minimises the effect. The plateau represents an inbuilt reserve, which is exploited by aircraft designers, and provides protection against hypoxia up to an altitude of 10 000 ft (3048 m) (fig 2). Ascent to this altitude produces a fall in alveolar Po_2 from the normal 13·7 kPa (103 mm Hg) to 8·0 kPa (60 mm Hg) but only a slight fall in percentage saturation of haemoglobin with oxygen. As the altitude progressively rises above 10 000 ft (3048 m) the percentage saturation of haemoglobin falls precipitously and results in hypoxia.

Cabin pressurisation and decompression

Commercial aircraft cabins are pressurised to below 10 000 ft (3048 m)—usually between 5000 and 7000 ft (1524-2134 m)—both as a safety margin for passengers (see previous article, 16 April, p 1229) and because psychomotor performance at novel tasks, which is of relevance to aircrew, deteriorates at altitudes of 8000 ft (2438 m).[3] Pressurisation to sea level, though ideal, is not cost effective.

Aircraft maintain a positive ambient pressure within their cabins by drawing in external air and delivering it compressed to the cabin. The outflow of cabin air is then controlled so as to maintain the required pressure differential. The through flow also ventilates the cabin and provides a means by which the ambient temperature may be controlled. As long as the pressurisation system and the aircraft remain intact, protection is provi-

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