The management of acute myocardial infarction

J. P. SHILLINGFORD
M.D., F.R.C.P.

*Medical Research Council's Cardiovascular Research Unit,
Royal Postgraduate Medical School, London, W.12*

UNTIL recently approximately 30% of all patients entering hospital following a myocardial infarction died. This figure is remarkably consistent throughout the hospitals of the world. The majority of deaths occurred within the first few hours and up to 1 week following the infarction.

The cause of death is not always clear but we can place it under the following headings:

1. Arrhythmias.
2. Myocardial weakness and failure.
3. Biochemical disturbances.
4. Embolism.
5. Misuse of drugs.

In 1960, standing at the side of a bed of a patient seriously ill following a myocardial infarction, we felt that we were extremely ignorant of what was happening to the circulation and what effect our therapeutic endeavours were having. As a result we decided to set aside a special room in which a patient could be nursed and continuously observed with facilities for measuring intravenous pressures, output of the heart, respiratory function and renal function. At the same time the electrocardiogram could be continuously recorded on magnetic tape in a central recording room.

Continuous recording of the electrocardiogram on magnetic tape has shown that some form of arrhythmia occurs in over 90% of all patients at some time in the first few days following a myocardial infarction. In fifty cases studied in our Coronary Care Unit the following incidence of arrhythmias was found: supraventricular tachycardia group 14%, supraventricular bradycardia group 24%, nodal rhythm 6%, ventricular tachycardia 4%, ventricular bradycardia 15%, extrasystoles only 20% and ventricular fibrillation 4%.

The arrhythmias are often transient and may last from a few seconds to hours or days. Their unpredictable behaviour often with spontaneous recovery makes assessment of the value of drugs in their treatment difficult. In many cases where the arrhythmia does not signify the risk of the development of more serious complications no specific

treatment is indicated. In others, the right treatment may be life-saving.

For the purposes of discussion the arrhythmias may be classified under the headings of extrasystoles, supraventricular and ventricular bradycardia, supraventricular and ventricular tachycardias and disturbances in atrioventricular conduction and asystole.

Extrasystoles

Atrial and ventricular extrasystoles are common following myocardial infarction and if occurring infrequently may be ignored. If, however, they occur more frequently early in the cardiac cycle with interruption of the T wave, in salvos of two or more, show a multiform configuration or occur in a frequency greater than 5/min, treatment should be given as these may herald the onset of more serious arrhythmias such as ventricular tachycardia or ventricular fibrillation. A single injection of 50 mg of lignocaine is given in the first place. If the arrhythmia is not suppressed a further 50 mg is given. If this treatment is successful in suppressing the ectopic beats a continuous intravenous drip of from 1 to 2 mg of lignocaine, diluted in glucose and water, 1 min is then given. Usually the drip has to be maintained for 24-48 hr when the ectopic beats will have disappeared spontaneously.

In the rare cases where lignocaine is ineffective 50 mg of procainamide may be given every 2 or 3 min up to 250 mg until the arrhythmia is abolished followed by an oral maintenance dose of 1-2 g daily for 2 or 3 days.

Nodal rhythm and supraventricular bradycardia

Atrioventricular or nodal rhythm may develop following a myocardial infarction with consequent loss of atrial transport function. In these rhythms the normal sequence of atrial and ventricular contractions is disturbed to a varying degree, depending on the altered relationship of the P and QRS complexes in the electrocardiogram. In the presence of acute cardiac infarction the loss of atrial transport function

may lead to a fall in blood pressure. In most patients return to normal rhythm occurs spontaneously. In cases of slow nodal rhythm where there is a serious deterioration in the patient's condition, intravenous atropine 0.3–0.6 mg may increase the heart rate and abolish the arrhythmia. In cases of nodal rhythm in association with a rapid heart rate, intravenous lignocaine may be tried or it may be necessary to use direct current countershock in combination with lignocaine.

Supraventricular bradycardia, or slowing of the heart in sinus rhythm occurs at some stage in 20% of all patients following an acute infarction and in the more severe cases may lead to a profound shock syndrome. This form of bradycardia may occur spontaneously and at times appears to be potentiated by the administration of morphine or other analgesic drugs. When the bradycardia is moderate and transient it is usually of little clinical significance. More serious consequences follow if it is profound, and particularly if ventricular function is poor with low stroke volume. Symptoms may be limited to a sensation of weakness and nausea while lying flat but with dizziness, pallor and sweating after sitting up or standing. The general appearance resembles that of a common 'vasovagal' faint. If the bradycardia progresses, mental confusion may be succeeded by loss of consciousness, associated with the clinical picture of profound circulatory shock. Other arrhythmias or cardiac arrest may follow.

Treatment is indicated in most patients at an early stage in view of the potential haemodynamic deterioration and also because of the increased incidence of serious arrhythmias occurring in patients in whom bradycardia is allowed to persist. Mild cases may be managed conservatively by maintaining the patient flat and raising the foot of the bed. If the heart rate does not increase and the blood pressure remains low, atropine sulphate should be injected intravenously. In the first place 0.3 mg is given and the pulse rate recorded for 5 min. If, at the end of this time, it has not reached 80–90 beats/min, a further 0.3 mg is given with similar increments, if necessary up to a total of 2 mg. Subsequent doses may be necessary 2 or 3 hourly until the heart rate is maintained at normal levels. It must be emphasized that the drug should be given intravenously as absorption from subcutaneous or intramuscular injection is too slow and unpredictable under conditions of circulatory failure.

Heart-block

Incomplete or complete heart-block is a not infrequent complication of myocardial infarction and, where complete, may be serious. Measurement of the stroke output of the heart under these conditions has shown that the damaged ventricle is not able to

compensate for the slow heart rate by increasing the amount of blood expelled at each beat; the resultant falling cardiac output rapidly leads to circulatory impairment and the shock syndrome. Usually first and second degree heart block cause little haemodynamic change and specific treatment other than careful and continuous observation of the patient is not necessary.

In cases of complete heart block with associated impairment of an adequate circulation treatment will be necessary. In some cases the administration of isoprotenerol hydrochloride at the rate of approximately $1 \mu\text{g}/\text{min}$ may raise the ventricular rate to approximately 50–60 beats a minute and thus maintain an adequate minute cardiac output. Care should be taken not to give too much isoprotenerol which may produce ectopic beats and herald the onset of ventricular fibrillation. In most patients, however, it is more satisfactory and indeed may be essential to pace the heart electrically by passing an electrode catheter into the right ventricle via an arm or neck vein.

Atrial fibrillation

Atrial fibrillation in patients with myocardial infarction is a not infrequent finding. It is often paroxysmal in nature and may last for a few seconds up to hours or days. Usually this arrhythmia spontaneously reverts on improvement of the patient's general condition. More rarely it may persist following the patient's recovery. Treatment depends on the general condition of the patient and how adequately the circulation is being maintained. In most cases the fast irregular rate will lead to impairment of the cardiac output and digitalis should be administered to slow the cardiac rate. Only very rarely will it be necessary to have to resort to direct current countershock to restore normal rhythm.

Supraventricular tachycardia

As in the case of atrial fibrillation supraventricular tachycardia may occur in the acute stages of the illness and often spontaneously reverts to sinus rhythm. Vagal stimulation by pressure on the carotid bulb or the eyeball, may, in a few cases, be successful. It may be possible to slow the heart by the use of digitalis; beta-blocking agents such as propranolol may be tried but caution must be exercised in their use in the presence of heart failure. If these remedies fail and the evaluation is inadequate then cardioversion by direct current countershock must be carried out.

Ventricular tachycardia

Ventricular tachycardia is not infrequent and one of the most serious arrhythmias which, if persistent, demands immediate treatment. Often, however, it presents as brief paroxysms which are self-limiting.

Treatment is the same as for ventricular ectopic beats and calls for the administration of 50 mg injection of lignocaine followed by a 100 mg injection if the first does not succeed in establishing sinus rhythm. If the ventricular tachycardia persists, electrical direct current countershock must be employed.

Haemodynamic changes occurring in myocardial infarction in the presence of sinus rhythm and their management

Systemic hypotension is frequently an outstanding feature of cardiovascular failure in patients with acute myocardial infarction. A low level of blood pressure is often used as an index of the patient's clinical condition and as a guide to the necessity for therapeutic support. Haemodynamic studies have shown that several haemodynamic patterns may be associated with a fall in the arterial pressure and that in the clinical assessment of patients with a low arterial pressure, a rigid interpretation based on the low cardiac output theory would sometimes be erroneous. Similarly the cause-and-effect relationship between low cardiac output and the shock state is not clear cut. An example of this is the common association of low arterial blood pressure with low stroke volume and cardiac output which occurred in a patient with the clinical features of shock. The peripheral resistance was very high. The further fall of 20 mmHg in arterial pressure between the first and second days was apparently related to a fall in peripheral resistance rather than stroke volume; in fact the cardiac output and stroke volume increased slightly. During convalescence progressive increase of the arterial blood pressure was associated with a rise of stroke volume to approximately three times the initial level. Peripheral resistance returned to the normal range.

The haemodynamic pattern is in marked contrast to that found in another patient in whom the same low initial arterial blood pressure was associated with a stroke volume and cardiac output three times as great as in the first patient, and within the normal range; the peripheral resistance was low. The rise in blood pressure during convalescence was related to a rise in peripheral resistance.

These patients emphasize the important point that one cannot be guided entirely by the level of the blood pressure in management but must take into account the patient's condition as a whole.

Systemic hypotension with adequate organ perfusion as assessed by normal skin temperature and colour, cerebral function and the passage of normal amounts of urine may be treated conservatively or by raising the foot of the bed.

Pulmonary oedema should be treated by diuretics, digitalis and oxygen. Infusions of glucose solutions

or alpha-blocking drugs have been recommended in advanced cardiovascular failure but no clear-cut haemodynamic advantages have to date, been recorded.

Management of the shock syndrome

The clinical picture of 'cardiogenic shock' is well recognized. The patient presents with cold sweating peripheries, cerebral confusion, irritability or unconsciousness and hypotension. Its mechanism of production is not always clear but in the great majority of cases in myocardial infarction is associated with failure of the heart as a pump. This failure may be, and is often associated with, the development of an arrhythmia; other causes of failure may be due to an excessively large infarct involving a considerable portion of the functioning myocardium, rupture of the interventricular septum, development of mitral incompetence due to involvement of the papillary muscle, disturbances in electrolytes or pH or the misuse of drugs. Any, or a combination of these factors may be responsible for the picture of 'shock'. Each cause needs a specific treatment and there is no one treatment for 'shock'. In fact, it would probably be better to talk of the 'shock syndrome' with the realization that essentially it is a series of clinical signs and symptoms with varying underlying causes. The institution of any treatment should, therefore, be governed by the patient's overall clinical condition and a clear understanding of the various possible mechanisms which may lead to a failure of the circulation following myocardial infarction. The indiscriminate use of one therapeutic agent such as a pressor drug for hypotension, could well lead to deterioration in the general condition of some patients.

From the practical point of view the following questions must be asked: What is the patient's rhythm? If irregular, or abnormally slow as in sinus bradycardia, treatment must be directed to this. Is there an associated disturbance in electrolyte balance or pH? In cases following cardiac arrest there may be a change in acid-base ratio towards acidosis which must be corrected with sodium bicarbonate. Has the patient developed mitral incompetence or ventricular septal defect? Both these can lead to cardiac failure and the shock syndrome. Is there associated respiratory failure with a very low oxygen tension? The administration of oxygen is important in these patients. In the absence of any of these complications the patient may exhibit the 'shock' syndrome in sinus rhythm due to the size of the damaged area of muscle itself being such that the heart is unable to maintain an adequate stroke output. Treatment has to be directed towards improving, if possible, the function of the remaining heart muscle. In some cases inotropic agents may be of value but are very variable in their effect. Digitalis may be given or, in

more extreme cases, isoprotenerol hydrochloride in glucose solution may be continuously administered at the rate of 1 $\mu\text{g}/\text{min}$.

Other points in the management of acute myocardial infarction

Bed rest

Serial observations of the state of the myocardium in those patients who have died at different periods of time following the acute attack have shown that maximum necrosis and softening of the myocardium occurs between the first and second weeks and healing in the form of fibroblasts and collagen fibres starts to take place at the end of 3 weeks and continues for several more weeks or months. A firm scar is formed by the end of 6 weeks. It would seem reasonable to keep the cardiac output from rising unnecessarily high during the first few weeks and for this reason we keep our patients at rest in bed for 2-3 weeks. Visitors are limited to close relatives for a few minutes during the first few days. The patient is washed and shaved during this time. A light, low salt diet, is given with adequate fluids. He is allowed to sit on a bedside commode for bowel movements. Towards the end of the second week, he sits out of bed and is slowly mobilized and leaves hospital at the end of 3 weeks to spend a further 3 weeks at home or in a convalescent home. He is allowed to return to work at the end of 6-8 weeks in all, providing that the cardiac function appears to have returned to normal.

Anti-coagulants

It is not our policy to routinely use anticoagulant drugs in our patients in the coronary care unit and it is our impression that their routine use does not significantly alter the mortality rate. If, however, a patient develops evidence of a venous thrombosis, with or without evidence of pulmonary infarction, anticoagulation treatment is instituted.

Prevention of venous thrombosis and respiratory complications

Simple leg exercises are of value in preventing venous thrombosis; simple movements of the foot up and down ten times can greatly increase the blood flow in the legs. The nurse, therefore, instructs the patient to move his feet up and down ten times every time she takes the pulse and blood pressure. At the same time she encourages him to take deep breaths to prevent atelectasis and increase the blood oxygen tension.

The coronary care unit

It is essential that, if the sudden development of arrhythmias is to be treated effectively, the patient must be under continuous observation by trained nursing staff, and expert medical assistance must be

immediately available over the whole 24 hr. On account of this the concept of the coronary care unit has been developed over the past few years. The basic principles of such a unit are quite simple, namely that an area should be set aside where patients in the acute stages of myocardial infarction may be under constant supervision. For an average size hospital the number of beds can be from two to six. The coronary unit should preferably be light and airy and quiet to enable patients to have adequate rest and quiet. There must be about double the space normally allotted to hospital beds around each patient. The patients may be in separate rooms or heavy screens should divide the beds in the event of a cardiac arrest. An oscilloscope to record the electrocardiogram is valuable to monitor the electrical activity of the heart and one can be placed in proximity to each patient. It may be convenient and helpful to have a large monitor oscilloscope at the nursing station to monitor more than one patient at once. In our experience the value of alarm systems for denoting tachycardia, bradycardia or cardiac arrest is doubtful and any noise or alarm may disturb the patient. Facilities for defibrillation by direct current countershock and for electrical pacing of the heart must be available with trained staff to operate them. It is a considerable advantage to have a switch at the side of each bed with a direct connection to the telephone exchange which sends out the alarm to the resuscitation team in the event of an emergency.

As a result of our experience over the last 7 years the coronary care ward of three beds at Hammer Smith Hospital is now incorporated, as far as the nursing service is concerned, as an integral part and extension of the general acute medical ward. The Unit is under the overall administration of the head nurse responsible for the whole ward. There is always one nurse trained in coronary care on duty in the ward but junior nurses take their turn in looking after coronary patients. This system of integrating the coronary unit into the medical ward means that when the patients are moved from the coronary unit into the main ward, they remain under the same nursing and medical care and in the event of an emergency can be quickly moved back into the coronary care unit. The training of the nurses is undertaken by the medical and nursing staff by a series of weekly lectures and demonstrations and it is our object to eventually train a large number of nurses in coronary care so that in the event of an emergency there is a pool of trained nurses within the hospital on whom one can call.

The medical staff are similarly trained and the resuscitation team on call for the whole hospital are used for purposes of cardiac resuscitation. At this time nurses do not perform electrical defibrillation themselves.