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PROBLEMS IN MANAGEMENT OF ACUTE MYOCARDIAL INFARCTION*

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Acute myocardial infarction varies greatly in severity from patient to patient. Its course is treacherous, its complications are frequent and serious, and its supervision is often testing and exacting. Five aspects of the medical management of these patients are worthy of special consideration in the light of current experience.

1. Diagnosis

Successful management depends primarily on early and precise diagnosis. The illness can then be more accurately assessed, the probability of complications more readily anticipated, and appropriate therapy begun from the outset. The first problem is the necessity to establish the diagnosis accurately and early.

In assuming responsibility for the management of acute myocardial infarction, the doctor should not hesitate to enlist the diagnostic help which technical procedures can provide. Many conditions masquerade as myocardial infarction. Electrocardiograms and other tests—transaminase levels, sedimentation rates—are not infallible, but in the hands of experienced observers they can provide a standard of accuracy of diagnosis with which the unaided clinician cannot compete. Paton (1957), working in this hospital, had a 94% accuracy of electrocardiographic diagnosis as judged by post-mortem control. This compared with a 35% accuracy amongst a group of patients in whom death occurred after days or weeks of observation by the physician who elected to place reliance purely on his clinical judgment, without the diagnostic help which electrocardiograms can provide. The moral is obvious.

2. Home or Institutional Care

An early decision must be taken on the advisability of home or institutional care. Routine medical supervision under home conditions was justified in the past, when treatment was commonly limited to rest in bed and appropriate sedation. Times have changed. To-day, domiciliary treatment is more exacting. Daily blood samples, laboratory visits, and the regulation of anticoagulant dosage impose a considerable burden on the single-handed doctor.

On account of the facilities for handling shocked patients, the benefit of adjacent laboratories, the provision of skilled nursing throughout the 24 hours, and the resources to deal promptly with sudden emergencies, the hospital has advantages which far outweigh home care for the great majority of these patients.

Nevertheless, domiciliary supervision may be necessitated by geographical considerations, an efficient medical clinic with reliable laboratories being too remote, or by a shortage of suitable medical bed accommodation. Some elderly people, resisting the disruption which a hospital imposes on their daily routine, will prefer to remain at home. The severity of the illness and the degree of shock at the onset may render transfer to hospital unjustifiable. Some patients will always require home care.

A major problem confronting the doctor is the justification for moving to hospital severely shocked patients, for whom the immediate risk of transfer is great. The practitioner, weighing the risks, knowing the time which must elapse before the patient can be expected to arrive in the hospital, should not, so far as is possible, postpone transport by ambulance. An opiate should be given, a pressor stimulant drug injected to counteract shock, and oxygen made available *en route*. The risk of leaving the gravely ill patient in his own home, at the mercy of unskilled attention, may be greater than the danger of his transport to the hospital.

The first two or three days are the most vital in treatment. No time should be lost in giving heparin: the earlier it is used and oral anticoagulation thereafter maintained, the better the prospects for that individual. Whether treated at home or in hospital, the patient requires skilled nursing. A nurse is required to act as a protector—a protector from relations, friends, and business associates. It is helpful to have a nurse accustomed to anticoagulant therapy and trained to record blood-pressure. Her understanding means much to the patient. She must be firm enough to insulate the patient from over-solicitous visitors, and yet not so strict as to foster such feelings of inadequacy that chronic invalidism is the result.

3. Bed Rest and Early Ambulation

Rest is a traditional principle in the treatment of active disease. On the average, six weeks are required for the acute myocardial infarct to be converted into a firm fibrous scar. There are wide variations in the rate of healing. The more protracted the pain, the more severe the shock, and the more numerous the complications, the longer will that patient require to be confined to bed. The advantages of complete rest in bed must be weighed against the deterioration of

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morale, the discomforts of constipation, abdominal distension, and urinary infections, the liability to peripheral venous thrombosis, the loss of muscular tone, and the development of arthritic adhesions.

The tradition of strict immobilization is difficult to overcome; for one reason, the old-fashioned hospitals in this country are not readily adapted to the care of convalescent or semi-convalescent patients, who, when out of bed, are too often regarded as a nuisance. More mobility is justifiable. These patients should be encouraged to feed themselves within a day or two of the onset, to sit up in bed and use their limbs to some extent, even before the end of the first week. By raising the head of the bed on blocks 12–18 in. (30–45 cm.) for a few hours each day, the patient is encouraged to use his legs to overcome gravity. The maintenance of skeletal muscular tone helps to shorten convalescence, reduces the risks of venous thrombosis, and lessens the sense of disability in the convalescent period.

So-called "chair treatment" should not be mistaken for ambulant or active treatment. Anyone who has had experience of sitting, even in a semi-reclining position, for 8 to 12 hours at a stretch, as in long-distance flying, soon appreciates that sitting is more tiring than lying and that sleep is less satisfying in the upright position. Sitting in a chair by the bedside for patients who are relaxed and comfortable and not fatigued by so doing may be encouraged from the third week onwards. Physiotherapy may be started as soon as the acute phase of the illness is past.

After four weeks in hospital, the majority should be ready for four weeks' convalescence at home. By the end of that month most should be sufficiently rehabilitated to return to work.

4. Prevention of Complications

The main complications of acute myocardial infarction are four in number: (1) shock, (2) left ventricular failure, including also congestive heart failure, (3) thromboembolic phenomena, and (4) the arrhythmias. The first three often occur in sequence in that order, and the doctor will be wise to anticipate difficulties. Consideration must be given to the selection of remedies designed to forestall or reverse these disorders.

Shock

A major problem is to know when to start energetic treatment and what form this should take. As there is no easy quantitative measurement of shock, its severity has to be assessed in a rough-and-ready way on clinical grounds, taking into account the patient's blood-pressure, pulse rate, and state of the skin as regards pallor, moisture, and cyanosis. The earlier reliable remedies are employed the more easily is shock arrested. It is better to assume that it will be severe than to wait for it to develop.

There are two factors for consideration in the treatment of cardiogenic shock: (1) the progressive reduction in cardiac output, the ill effects of which can be overcome to some extent by a sufficient rise in the

total peripheral resistance; and (2) the development of pulmonary oedema in an acute or subacute form, the consequence of left ventricular failure. In the presence of shock, distension of the neck veins and moisture in the lungs are an ominous combination.

The indications are to increase the coronary perfusion pressure and thus promote the efficiency of the surviving as opposed to the necrotic heart muscle. To accomplish

FIG. 1.—Response of blood-pressure to vasopressor drugs in a man aged 58 who sustained an acute infarction shortly after admission to hospital. Shock developed rapidly and was not arrested until a noradrenaline drip was used. The hypotension was temporarily overcorrected (B.P. 170/106) by this means—fortunately without harm.

these objectives, in addition to the usual symptomatic measures designed to control pain, restlessness, chilling, and anxiety, the prompt use of vasopressor drugs in sufficient quantity to support the peripheral pressure is indicated. Systematic blood-pressure recordings are essential for the proper control and regulation of pressor drugs. Employed early and repeated as necessary, they may be all that are required to reverse the shock syndrome. An example of the response of the blood-pressure to their use is shown in Fig. 1, constructed from the records of a patient in whom shock developed with great rapidity. This man was already in the hospital when the acute infarct occurred. He made a good recovery.

More often than not, by the time the shocked patient arrives in hospital a greater or less degree of left ventricular failure complicates the situation. This is shown by the detection of an increased respiratory rate, neck-vein distension, and moist sounds at the lung bases. In these circumstances, prompt and thorough digitalization may prove to be the first measure really effective. Should an intravenous drip of a pressor drug prove necessary, the digitalized heart is more likely to tolerate the increased circulatory load. The correction of delayed cardiogenic shock by the use of intravenous, and later oral, digoxin is shown in Fig. 2.

Our experience with mephentermine, given intravenously as a priming dose and repeated thereafter intramuscularly, has on the whole been disappointing. Its effects are unpredictable and usually too transient to be really reliable and effective. Metaraminol ("aramine"), according to Weil and Spink (1955), has the advantage that intermittent intramuscular injections every half to two hours will often support or restore a falling pressure, thus circumventing some of the

problems inherent in the use of a continuous intravenous drip. A single intramuscular injection of metaraminol in a dose of 5 to 10 mg. yields a maximum pressor response in a few minutes, the rise in pressure persisting for 30 to 40 minutes. As a temporary expedient—for instance, to prepare a patient for transport to hospital—or as a temporary measure while a noradrenaline drip is being got ready, with subsequent intramuscular

and well diluted (say, 20 ml. over a period of 5 to 10 minutes), on account of the risk of overstimulation and rupture of the ventricle at the site of the infarct. Digoxin is a safer drug in this emergency.

Experience shows that congestive heart failure is prone to develop within two or three weeks in patients surviving the immediate upheavals of shock and acute left ventricular failure. A reasonable degree of digitalization should therefore be maintained for some weeks after the acute emergency has subsided.

FIG. 2.—Correction of delayed shock in patient aged 67 after an acute infarct by use of intravenous digoxin after other remedies had failed to check the fall in pressure.

injections if necessary, these drugs have a useful but limited place in treatment.

If the response to successive injections of vasopressor drugs is unsatisfactory there should be no hesitation in using digoxin intravenously (0.5 to 1.5 mg.) and setting up a continuous noradrenaline drip.

The effective dose of noradrenaline can be determined only by regulating the rate of flow according to the blood-pressure response. The problem is to maintain an effective and relatively steady systolic pressure, preferably between 100 and 120 mm., without the addition of too great a volume of fluid to the circulation. The dangers are acute hypertension and possibly ventricular rupture on the one hand, and acute pulmonary oedema on the other. Constant supervision of the drip, with pressure readings every 20 minutes, makes this an exacting form of treatment. A concentration of 4 mg. of noradrenaline in 400–800 ml. of 5% glucose, run over a period of four to eight hours or longer, can restore and maintain pressure, and abolish the severer grades of shock, if used promptly before irreversible damage has been done.

Left Ventricular Failure

Acute left ventricular failure does not present much of a problem, provided it is treated energetically with morphine and rapid digitalization by the intravenous route from the outset. Many fairy stories persist to the present day about the dangers of digitalis. Clinical experience indicates that digitalization is justified when cardiogenic shock or acute left ventricular failure supervenes as a complication of an acute infarction. A dose of 1 mg. digoxin intravenously, and diluted with 10 ml. of normal saline, is often effective within half an hour. If aminophylline is used, it should be given slowly

hospitals in this country at the present time. Fig. 3, reproduced from their paper, brings into contrast the yearly mortality rates in their two groups. The conclusion is warranted that anticoagulants make a substantial contribution to the treatment of acute myocardial infarction. Until some simpler and more effective remedy is available these drugs are indicated from the outset in all cases, with very few exceptions.

Paroxysmal Arrhythmias

Fortunately the paroxysmal arrhythmias are comparatively uncommon. Fibrillation and flutter can usually be controlled by thorough digitalization and seldom give rise to difficulty.

Paroxysmal ventricular tachycardia predisposes to congestive failure, to a greater or less degree of shock, and to ventricular fibrillation, which is commonly fatal.

The problem is to bring the ventricular rate, usually 180 or more, under effective control as soon as possible. Quinidine sulphate is the most reliable remedy available, and, provided portal congestion has

Thrombo-embolic Phenomena

To my mind there is no problem here. Objective statistical studies show a consistent advantage in favour of those receiving anticoagulants during the acute phase of a myocardial infarct. If anticoagulant therapy were a less exacting routine, devoid of daily blood-sampling and less dependent on a relatively crude laboratory technique, there would be less hesitation and less opposition to its routine use. Striking support for the earlier conclusions regarding its efficacy (Tulloch and Gilchrist, 1950) comes from the report of McCluskie and Seaton (1959), who from a five-year study conclude that anticoagulants yield a reduction in mortality rate and in incidence of thrombo-embolic complications of approximately one-half—a result thought to be representative of most

FIG. 3.—Yearly mortality rates observed by McCluskie and Seaton (1959), in two simultaneous groups of acute myocardial infarction, treated with and without anticoagulants. (Reproduced from the *Scottish Medical Journal*.)

not resulted in nausea and vomiting, its oral administration is the method of choice. The aim is to reduce gradually the ventricular rate from say 200 to 120 or 110. By so doing the congestive failure and the peripheral circulatory collapse can be relieved as the heart rate slows, even before normal rhythm is finally restored. Electrocardiograms, heart rates, and blood-pressure readings should be recorded at two- or three-hourly intervals and quinidine administered, at first in a large single dose of 0.6 to 1 g., in the form of capsules or cachets, and subsequently in regular doses of 0.2 to 0.4 g. at two- or three-hourly intervals. A slight slowing of the ventricular rate, counted at the apex beat, is usually observed within a couple of hours. Thereafter, as oral doses are repeated, the rate continues to decline progressively.

When the ventricular rate has fallen to the neighbourhood of 110–100 the drug should be discontinued—that is, if normal rhythm has not already replaced the ventricular arrhythmia. Electrocardiograms and apex counts should be continued, and usually within the next three or four hours the records will show the abrupt resumption of normal rhythm. Serious toxic effects in the electrocardiogram may warrant a reduction or a discontinuance of the drug. No reliance can be placed on a single dose of quinidine or other remedy to control ventricular tachycardia. The cumulative effect of systemic dosage, outlined above, at two- or three-hourly intervals and regulated by repeated electrocardiograms and counts of the ventricular rate is illustrated in Fig. 4. It is the method most likely to meet with success in the treatment of this serious complication of acute infarction.

In desperate situations the intravenous use of quinidine can be life-saving. Nausea, vomiting, diarrhoea, and collapse may preclude oral doses. In these circumstances, during the past 20 years, I have frequently employed the method devised by Hepburn and Rykert (1937). By vigorous shaking, 50 to 60 gr. (3.2 to 3.9 g.) of

quinidine sulphate can be dissolved in 500 ml. of 5% glucose. After filtering and sterilizing, the solution is given by intravenous drip at a rate of 100 to 120 ml. per hour. Heart counts, blood-pressure readings from the opposite arm, and electrocardiograms should be recorded repeatedly during the drug's administration.

FIG. 5.—Response of the heart rate to intravenous quinidine sulphate in the treatment of ventricular tachycardia, complicating acute myocardial infarction, in a man aged 72.

Fig. 5 shows the response of the ventricular rate to intravenous quinidine in a man aged 72. The drip was discontinued after 25 hours, by which time his congestive failure was well under control. Normal sinus rhythm was not restored until eight and a half hours later. It is only the seriousness of the situation which justifies intravenous quinidine.

Supraventricular tachycardia is a less frequent complication of myocardial infarction. When it is unresponsive to full digitalization and other vagotonic drugs, congestive heart failure supervenes and tends to grow worse. On the basis of its power to reduce local oedema, cortisone has been used successfully in the treatment of complete heart-block, a consequence of an acute myocardial infarct invading the ventricular septum. Similar analogies suggested its employment in a man of 57, whose auricular tachycardia persisted for 20 days and was responsible for an increasing degree of congestive failure. He had sustained an acute infarct a few days before the onset of the arrhythmia. The response to the procedures used is shown in Fig. 6.

FIG. 6.—Response of the heart rate to various remedies used to correct an auricular tachycardia of 20 days' duration, occurring after an acute myocardial infarct in a man aged 57.

FIG. 4.—Effect of oral quinidine on heart rate in three separate attacks of ventricular tachycardia, in a man aged 54, shortly after an acute myocardial infarct. In the first attack he received a total of 10.6 g. in 58 hours before normal rhythm was restored. He was remarkably tolerant of quinidine, receiving on another occasion as much as 0.65 g. per hour for 12 doses, by which time his heart rate fell from 150 to 100, normal rhythm being restored 16 hours later. The circles indicate the ventricular rate calculated from electrocardiograms; the squares, the rate during normal rhythm.

5. Return to Work

On his discharge from hospital, having recovered from the acute phase of the illness, the problem of

the patient's resumption of work then confronts the doctor. In general an early return to work is to be encouraged.

So long as disabling symptoms persist—exertional pain or dyspnoea—a resumption of occupation may be out of the question. For those handicapped by a residual angina of effort, or even minor degrees of congestive failure, retirement from active work may be the only alternative. Recovering satisfactorily from the acute attack and having mild or only trivial symptoms when ambulant, the patient should in general be able to resume some form of active work after a month's convalescence—that is, in approximately eight weeks from the onset. The weeks elapsing after leaving hospital should be devoted to the recovery of physical fitness and improved muscular tone, skeletal and cardiac. Rehabilitation has its psychological as well as its physical aspects. The doctor can contribute much to the recovery of confidence, which should be a constant aim.

There is nothing to be gained and much to be lost by confining a symptomless patient to bed for two to three months and then subscribing to such a graduated convalescence that little or no attempt is made to resume an active working life, even after a further three to four months' rest. The tendency to impose a great restriction of activities is lessening, but patients are still seen who have received much advice of a negative character, such as, "Don't stoop, don't lift, don't strain, don't hurry, don't walk for more than five minutes, don't take more than five steps on the stair," and so on.

The main principle of rehabilitation is to encourage rather than to restrict. The cardiac patient should be advised to do as much, or a little more, than he thinks he can—that is, short of the production of disagreeable symptoms. In brief, the attitude of his adviser should be not "so little" but "so much." Confident reassurance throughout the illness and convalescence can counteract the development of a neurosis, which is all too apt to prove in the long run a greater handicap than the structural damage.

Admittedly much depends on the nature of the employment to which the patient has been accustomed, his mental and physical stamina, and the ease with which symptoms are induced. I have known miners, within a couple of months after an acute myocardial infarction, return to work at the coal-face without harm to themselves. Busy executives may well resume full and increasing responsibility. Capacity for work can be determined for the individual only by the graduated method of trial and error. Fortunately, the great majority recover from the acute attack and are able to lead useful and productive lives, thus dispelling the notion that a damaged heart necessarily leads to permanent disability and incapacity.

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BLOOD COAGULATION AND FIBRINOLYSIS IN RELATION TO CORONARY HEART DISEASE

A COMPARATIVE STUDY OF NORMAL WHITE
MEN, WHITE MEN WITH OVERT CORONARY
HEART DISEASE, AND NORMAL BANTU MEN

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Since the middle of the nineteenth century there have been two main theories regarding the pathogenesis of atherosclerosis. Virchow (1856) postulated that atheromatous lesions are produced by the imbibition of lipids from the circulating blood, while Rokitansky (1841) favoured the view that the primary lesion is a deposition of fibrin on the arterial intima. These two theories continue to dominate modern thought on atherogenesis, and they have led to many studies of the blood lipids and of blood coagulation and fibrinolysis in relation to coronary heart disease.

In Southern Africa, research in this field has been facilitated by comparative interracial studies among the White population, in whom the death rate from coronary heart disease is very high, and the Bantu population, in whom deaths from this disease are extremely rare. Previous reports from this department have dealt with differences in serum-cholesterol levels in white and Bantu men and with the role of dietary fat intake in their production (Bronte-Stewart *et al.*, 1955, 1956; Brock and Bronte-Stewart, 1955). The present report is concerned with a comparison of blood coagulation and fibrinolysis in apparently healthy men of these two races and in white men with clinical evidence of coronary heart disease.

Material of the Investigation

The three groups of subjects who were studied are shown in Table I. The normal white men ("controls") were mainly white-collar workers from a large Cape-town insurance company; a number of professional

TABLE I.—Material of Investigation

	No.	Age (Years)		Mean
		Minimum	Maximum	
Controls	80	20	65	39.8
Coronary	48	38	69	52.3
Bantu	53	20	65	33.8