Today's Treatment

Diseases of the cardiovascular system

Management of acute myocardial infarction

J F PANTRIDGE, J S GEDDES

British Medical Journal, 1976, 2, 168-170

Each year in Britain over 55 000 deaths among people under 70 result from myocardial infarction. Nearly two-thirds of these premature deaths are classified as sudden, as they occur within one hour of the onset of symptoms. Asystolic arrest usually related to acute atrioventricular block is responsible for a few deaths, but more than 90% of the sudden deaths from ischaemic heart disease result from primary ventricular fibrillation. Since the median delay in hospital admission may be more than eight hours, the hospital coronary care unit cannot influence the incidence of sudden death nor will it appreciably reduce the community mortality from the acute coronary attack, which is about 40%. Furthermore, the hospital coronary care unit will not affect the incidence of cardiogenic shock and pump failure, since these complications are manifestations of an extensive infarct. Clinical and experimental evidence indicates that after coronary occlusion there is likely to be a step-by-step increase in the amount of myocardium destroyed and that the size of the infarct is determined in the first few hours. Early therapeutic intervention may limit the magnitude of the infarct.

Since the incidence of ventricular fibrillation is highest immediately after the onset of symptoms and since treatment to limit the size of the infarct and prevent shock and pump failure is likely to be most effective if started at the onset of the coronary episode, the early start of intensive care is clearly imperative.

Ventricular fibrillation

The correction of ventricular fibrillation outside hospital has been shown to be a practical proposition.^{1,2} This has led to the development of mobile coronary care units, many of them staffed by medical personnel. It has been found, however, that paramedical workers are quite capable of correcting ventricular fibrillation. In the USA, in addition to mobile units, life-support stations have been installed in football stadia, skyscraper office blocks, factories, and airports. In Seattle about 25% of the adult population have been instructed in cardiopulmonary resuscitation. In that city (population 400 000) there were 73 long-term survivors among those defibrillated outside hospital by paramedical workers in one year. If this figure is applicable to the cities in Britain some 4000 lives might be saved annually by correcting ventricular fibrillation outside hospital.

The report of a working party of the Royal College of Physicians and British Cardiac Society on the care of the patient with coronary disease³ stated that: "widespread instruction of the public in resuscitation should be encouraged as a further means of rescuing victims stricken by acute heart attacks. Such instruction could be carried out by trained laymen or medical personnel in schools, factories, and large institutions." Further involvement of the public has been suggested, and Friedberg has stated that "possibly every home with a person at risk should have a portable defibrillator next to the television set." In buildings occupied by many people, particularly middle-aged men, it might be appropriate to mount portable defibrillators beside the fire extinguishers. Furthermore, a portable defibrillator should be part of the equipment of general practitioners and doctors who are likely to have to attend medical emergencies.

The major barriers to the widespread availability of portable defibrillators for use outside hospital relate to their cost, bulk, and weight. The claim of Tacker and co-workers that defibrillators should be capable of delivering more energy than they do at present is calculated to heighten the barriers. The view that high-energy direct-current shock is necessary, however, has been challenged and indeed a miniature defibrillator weighing 3.2 kg is available.^{4,5}

PREVENTION

The prevention of ventricular fibrillation presents difficulties. In hospital coronary care units prophylactic antiarrhythmic agents are administered when R on T ectopics are evident or when ventricular ectopics are multifocal, occur in salvos, or occur more frequently than six a minute. This is not a rational policy during the acute phase since it is now recognised that about 40% of patients who develop ventricular fibrillation do so without prior warning dysrhythmias. Ideally, therefore, if an effective prophylactic antiarrhythmic agent were available it should be given to all patients when first seen. It has been claimed that lignocaine reduces the risk of ventricular fibrillation when given as an intravenous bolus of 100 mg followed immediately by an infusion of 3 mg/min. Nevertheless, lignocaine may be less effective during the acute phase than later in the attack. It may fail to control ventricular ectopics and may have an adverse effect when the heart rate is over 90/min.

The high incidence of ventricular fibrillation immediately after the onset of acute infarction and the difficulty in its prevention at that time may relate to the frequency of the autonomic disturbance associated with coronary occlusion. Among patients seen within 30 minutes of the onset of the coronary attack over 80% show clinical evidence of autonomic disturbance.⁶ Nearly half show evidence of parasympathetic overactivity, with bradycardia or hypotension, or both. More

Cardiac Department, Royal Victoria Hospital, Belfast

J F PANTRIDGE, MD, FRCP, physician in charge and professor of cardiology, The Queen's University of Belfast

J S GEDDES, MD, MRCP, physician

than one-third show sympathetic overactivity with tachycardia or transient hypertension, or both.

TREATMENT

Pain may accentuate or indeed initiate autonomic disturbance. Its relief is therefore of primary importance. Catecholamine release, which is significantly related to the development of ventricular dysrhythmias, may be diminished by the immediate control of pain. Analgesics used in relieving the pain of acute myocardial infarction should also relieve distress and fear. The ideal analgesic should have a rapid effect without causing nausea, vomiting, or haemodynamic disturbance.

Pethidine and pentazocine have potential adverse haemodynamic effects. Narcotic drugs are usually advocated despite their respiratory depressant effect.

Morphine is the drug most commonly used. Administered intravenously, 10 mg will usually relieve pain rapidly and alleviate mental distress. Heroin has a more rapid action and less emetic effect than morphine. When given in the standard intravenous dose of 5 mg it has little haemodynamic effect. After the administration of a narcotic drug hypotension may occur on tilting or during transport with the feet dependent. Every effort should therefore be made to keep the patient supine.

Bradycardia

Sinus bradycardia does not require treatment unless accompanied by hypotension or ventricular extrasystoles. Nevertheless, uncomplicated bradycardia in the acute phase of myocardial infarction is uncommon. Bradycardia immediately after the onset of acute infarction is usually associated with hypotension, which in most cases may be corrected by raising the heart rate. The slow heart rate is associated with a fall in ventricular fibrillation threshold and an increased risk of ventricular fibrillation. Conversely, raising the heart rate may abolish ventricular ectopics.

When atropine is required for correcting sinus bradycardia it is given intravenously in aliquots of 0.3 or 0.6 mg. Careful monitoring is mandatory during administration of the drug since an inappropriate and dangerous increase in the heart rate may result. Early atrioventricular block, second degree or complete, will usually respond to atropine. The dose required for the correction of atrioventricular block, however, is usually much larger than that required for treating sinus bradycardia.

Tachycardia

The tachycardia associated with sympathetic overactivity has an adverse effect on the ventricular fibrillation threshold. In the patient with recent coronary occlusion an increase in the heart rate occurring spontaneously or resulting from the injudicious administration of atropine may precipitate ventricular fibrillation. Antiarrhythmic agents may fail to control ventricular ectopics unless the tachycardia associated with sympathetic overactivity is corrected. An inappropriately rapid heart rate may occur during movement in more than one-third of patients with acute myocardial infarction. This may be relevant to the frequency of arrhythmic death in ambulances. Thus monitoring during transport is imperative.

Tachycardia resulting from sympathetic overactivity may be controlled by a beta-adrenergic blocking agent. Practolol, a selective cardiac beta-blocker, has been used. The drug is administered intravenously in aliquots of 5 to 10 mg and a decrease in the heart rate is apparent within two minutes. Doses of more than 10 mg do not usually result in additional slowing of the heart rate. When the heart rate is greater than 120/min practolol seldom produces a fall below 100/min. Sotalol, which does not possess intrinsic sympathomimetic activity, has a more potent negative chronotropic effect.

Prevention of shock and cardiac failure

Since established cardiogenic shock is associated with massive infarction its treatment is seldom rewarding. Clinical studies^{7 8} and recent experimental evidence, however, indicate that the magnitude of the infarct may be limited. After coronary occlusion irreversible damage begins within 20 minutes in totally ischaemic regions but survival is more prolonged in much of the affected zone. Many cells may recover provided the intensity of ischaemia does not increase. The fate of such jeopardised myocardium is determined by the balance of oxygen supply and demand. It has been rightly said that the quantity of jeopardised myocardium that can be salvaged varies inversely with the time after coronary occlusion that treatment begins. The loss of a critical amount of muscle may result from delay in treatment. This loss will further depress pump function. The resulting less favourable haemodynamic conditions jeopardise new areas and a vicious circle is begun.

That the incidence of shock may be reduced when haemodynamic disturbances secondary to autonomic imbalance are controlled early in the attack was shown six years ago.^{7 8} Only 3.4% of patients aged 70 or less treated within one hour of the onset developed shock, with the result that the hospital mortality was under 10%. This may be contrasted with the prevailing mortality of 15-20% among patients treated in hospital coronary care units.

Blood flow to ischaemic myocardium through collateral vessels occurs mainly during diastole and is very sensitive to changes in arterial pressure. Hypotension or tachycardia therefore reduces flow. Oxygen requirement in contracting myocardium is also related to arterial pressure but to a lesser extent than blood flow. An increase in the blood pressure from hypotensive to normotensive levels may therefore have a favourable effect on myocardial survival. Oxygen requirement is also affected by the heart rate, the contractility of the myocardium, and the ventricular diastolic or filling pressure. A rise in the last, indicative of left ventricular failure, decreases blood flow by compressing the subendocardial zone. The associated dilatation of the left ventricle results in an increase in the work required to maintain the arterial pressure.

The early vagal overactivity of acute myocardial infarction is usually complicated by hypotension, which is frequently profound. Among patients seen within 30 minutes, 22% will have a systolic blood pressure not over 80 mm Hg.⁸ Hypotension and resultant reduction in blood flow through ischaemic areas extend the boundaries of the developing infarct. Correction of early vagal overactivity and its associated bradycardia and hypotension with appropriate doses of atropine improves myocardial perfusion and prevents or limits extension of the infarct.

Sympathetic overactivity is associated with sinus tachycardia, with increased contractility, and not infrequently with transient hypertension. The resulting stress on the left ventricle may precipitate failure which is often clinically unobtrusive. Reduced diastolic compliance (increased stiffness) of the ischaemic zone aggravates this effect. Thus sympathetic overactivity increases oxygen demand and curtails supply. Beta-adrenergic sympathetic blocking drugs control tachycardia and excessive contractility with little effect on ventricular filling pressure or arterial blood pressure (unless this is greatly increased). The longer diastolic intervals permit an increase in collateral flow. The viability of jeopardised cells is improved.

Hypotension in the absence of bradycardia and simulating cardiogenic shock is sometimes associated with inadequate venous return and a low ventricular filling pressure. When signs of left ventricular failure are absent 200 ml of a solution of 5% dextrose are infused rapidly, and if an increase in arterial pressure follows a volume expander such as low molecular weight dextran in 5% dextrose is infused for a few hours at a rate sufficient to maintain the systolic blood pressure at a level above 90 mm Hg.

Other therapeutic methods directed towards limiting infarct size are at present undergoing intense investigation.

Infusions of vasodilator drugs such as nitroglycerine and intravenous administration of frusemide effectively lower the ventricular filling pressure when this is raised, and provided the effects are monitored carefully a harmful reduction in blood pressure may be avoided. A reduction in S-T segment elevation after nitroglycerine in one clinical study suggested that ischaemia had been reduced.

Myocardial oedema secondary to ischaemia increases vascular resistance and contributes to extension of the infarct. It may be reduced by administering mannitol, an osmotically active agent. The enzyme hyaluronidase may improve the transfer of substrates between the circulation and tissues, and a preliminary clinical trial of this agent has produced encouraging results. Glucose and glucose-insulin-potassium mixtures may provide metabolic support even in the absence of improved oxygenation. Corticosteroids when administered in high doses prevent the disruption of intracellular structures and may allow ischaemic tissues to survive until better haemodynamic conditions prevail.

Fortunately it is already possible to influence the incidence of shock by such simple measures as relieving pain and correcting the autonomic disturbance. It does not seem over-optimistic to expect that treatment begun soon after the onset of the attack with one or more of the recently introduced agents will help to limit the size of the infarct.

Prehospital coronary care

Although most of the unnecessary deaths from acute myocardial infarction are sudden and occur outside hospital, and although most of the myocardial damage occurs in the first three hours, little attempt has been made in Britain to provide prehospital coronary care. The lack of enthusiasm for mobile coronary care units in this country despite their proliferation in the USA requires comment. Undoubtedly the concept of prehospital and hospital coronary care has been influenced adversely by the misunderstanding of the findings of the Bristol Study. The report of the Bristol study⁹ did not, of course, question the unassailable fact that most early deaths from acute myocardial infarction result from ventricular fibrillation which may be corrected or prevented. Nor did that report question the fact that infarct size might be limited and cardiogenic shock and pump failure prevented.

The Bristol study purported to show that for some patients with myocardial infarction treatment at home might be satisfactory. This claim is not unrealistic. Intensive care has little to offer the patient who has survived the acute phase of the infarct. It is, however, salutary to consider the fate of patients in the Bristol study who sought medical advice within one hour but who presumably did not get intensive care at that time. The mortality was 26%. This is to be contrasted with a mortality of less than 10% in comparable patients who received intensive care within one hour of the onset of symptoms.^{5 6}

It is unfortunate that prehospital coronary care schemes are assumed to be expensive in terms of personnel and equipment. The mobile coronary care unit may be integrated into the existing emergency system and the addition of a prehospital coronary care scheme to the hospital coronary care unit should result in only a marginal increase in the cost, apart from that increase resulting from admitting those whose death outside hospital has been prevented. This latter cost may be diminished when attention is directed to the earlier discharge of patients no longer at risk. One-third of patients with acute myocardial infarction may be discharged on the third day.¹⁰ When a higher proportion of patients get intensive care early, fewer will have large infarcts and, therefore, more will qualify for early discharge.

When the cost of prehospital coronary care is under consideration attention should be directed to the cost of the hospital coronary care unit and to the fact that in the absence of an associated prehospital coronary care scheme the hospital unit will have only a small impact on community mortality.^{5 11} It is also salutary to remember the resources wasted in the unsuccessful

treatment of cardiogenic shock. It is not unreasonable to suggest that much of the effort and expenditure directed to the treatment of cardiogenic shock by elaborate medical and surgical procedures might be redirected towards the prehospital phase of acute myocardial infarction and the prevention of these complications.

The report of the Working Party of the Royal College of Physicians of London and the British Cardiac Society³ stated that "in view of the high death rate in the first three hours after an attack, the facilities that are generally available are inadequate. First, the public should be further instructed on the indications to call without delay upon emergency skilled help, and mobile coronary care units should be further developed. The exact means must depend on local conditions and requirements. Thus, their implementation should be on a local or regional basis, with the encouragement of the Department of Health and Social Security and the other national health authorities."

References

- 1 Pantridge, J F, and Geddes, J S, *Lancet*, 1966, **1**, 807.
- 2 Pantridge, J F, and Geddes, J S, *Lancet*, 1967, **2**, 271.
- 3 Working Party of the Royal College of Physicians and the British Cardiac Society, *Journal of the Royal College of Physicians*, 1975, **10**, 5.
- 4 Pantridge, J F, et al, *British Medical Journal*, 1975, **2**, 313.
- 5 Pantridge, J F, et al, *The Acute Coronary Attack*. Tunbridge Wells, Pitman Medical, 1975.
- 6 Pantridge, J F, et al, in *Progress in Cardiology*, vol 3, ed P N Yu, and J F Goodwin, p 173. Philadelphia, Lea and Febiger, 1974.
- 7 Pantridge, J F, *Quarterly Journal of Medicine*, 1970, **39**, 621.
- 8 Pantridge, J F, World Health Organisation, *Mobile Coronary Care Units*, EUR 5020. Copenhagen, WHO Regional Office for Europe, 1970.
- 9 Mather, H G, et al, *British Medical Journal*, 1971, **3**, 334.
- 10 Wilson, C, and Pantridge, J F, *Lancet*, 1973, **2**, 1284.
- 11 Pantridge, J F, *British Heart Journal*, 1974, **36**, 233.

What treatment could help a woman of 60 with severe and painful osteoarthritis of both knees?

This is an important question because it is an extremely common condition and there is no straightforward answer. Joint disease and arthritis connote "crippling" to many patients, particularly the elderly who have a real fear of losing independence in daily living activities. It is important, therefore, to emphasise the relatively benign prognosis for function in most sufferers, and orthodox management by rest during the acute phase, sensible home exercises, weight reduction in the obese, and the appropriate use of simple analgesics and anti-rheumatic agents are all important and enable most patients to cope. In those with more severe disease, with joint instability and deformity as well as continuous or more severe pain and loss of function and ability, the advice of an orthopaedic surgeon should be sought. Osteotomy, as pioneered by Benjamin,¹ is valuable, particularly when combined with correction of deformity and relief of pain, and increase in function may be expected to follow in most suitable cases. More advanced procedures such as joint replacement arthroplasty² have not yet achieved the success of total hip replacement for largely technical and anatomical reasons peculiar to the knee joint. At present it is reasonable to advise these procedures in those patients who have gross deformity, severe pain, or destructive changes such that their function is very severely limited.

¹ Benjamin, A, *Journal of Bone and Joint Surgery*, 1969, **51B**, 4, 694.

² Smillie, I S, *Diseases of the Knee Joint*. Edinburgh, Churchill Livingstone, 1974.

What value have barrier creams in protecting the skin of foundry workers?

Barrier creams are only of limited value in protecting the skin from contact with acids, alkalis, detergents, and other irritants. They are helpful not because they prevent contact of the irritant with the skin but because they allow the irritant to be removed more easily. If mechanisation cannot eliminate contact with irritants, then there is no substitute for adequate protective clothing.