Treatment of coronary heart disease

Part 4: A review of the management of Myocardial Infarction in general practice

- E L Murray

Summary

In this final part of a review of coronary heart disease, the management of Myocardial Infarction and its complications are discussed. The initial steps in rehabilitation are also given from the perspective of general practice.

KEYWORDS: Coronary disease; Myocardial infarction; Pulmonary oedema; Drug therapy; Rehabilitation

As more than two thirds of the deaths from acute MI occur before the patient reaches hospital and more than 40% of deaths occur in the first hour, this is where any reduction in mortality is going to be achieved. More than 90% of early deaths are due to VF. Our patients must be trained to seek medical attention when they have symptoms of a MI. The public must be instructed in CPR. The doctor must get to the patient as soon as possible (whether it is quicker to get to the patient at home or to the patient in hospital must be determined) and defibrillation equipment must be available. All of the above proposals have problems.

The patients will persist in taking antacid tablets for their "indigestion" or in waiting for the pain to pass. They are reluctant to worry us and these major events "never happen to us personally, but always to the next man".

More than 40% of deaths occur in the first hour

The National Heart Foundation has for four years now been trying to interest the general public in CPR. There are a few first aiders and paramedics trained, but the public is reluctant to get involved. In Seattle in the USA, which has a population of 500 000, more than 100 patients with VF that occurred outside the hospital have been resuscitated. The lay public are well trained in this, the "CPR capital of the world." It is reported to be the best place in the world to have a MI, but the worst in which to suffer a vasovagal attack!

The sooner the GP gets to the patient the sooner treatment can be started and clinical evidence indicates that after a coronary occlusion the size of the infarction is likely to be determined within the first few hours and that early therapeutic intervention may have a salutary effect on the size. The extent or size of the infarct is the single most important determinant of survival after infarct.

The aim of initial treatment is control of arrhythmias, relief of pain and the correction of autonomic disturbances.

VENTRICULAR ARRHYTHMIAS

Ventricular ectopics in patients with a bradycardia may be abolished by raising the heart rate with atropine.

Ventricular ectopics need to be suppressed if they are

(i) more than 6 per minute
(ii) multifocal
(iii) occurring on a T wave (R on T phenomenon)
(iv) runs of two or more
(v) coupled beats.

Lignocaine is the drug of choice and is given as a bolus of 100 mg (1-2 mg/Kg body wt.) slowly over 1 to 2 minutes. This bolus dose may be repeated if necessary after 5 minutes, after which a continuous infusion is necessary. This is given at 4 mg/minute for 30 min. (4 ml lignocaine 10% in 200 ml, 5% DW with a 15 drops/ml drip set at 30 drops/minute). This rate is halved for the next two hours and if satisfactory, continued at 1 mg/min (7 drops/min.).

If Lignocaine fails, the following agents can be tried.

a) Procainamide given in an initial loading dose of 100 mg IV repeated at 5 minute intervals until controlled, or until a maximum of 1 gm. Maintenance infusion runs at 2-5 mg/minute.
b) Mexiletine is started with an IV loading dose of 200 mg given over 10 minutes. Then infuse at a rate of 4 mg/min for 1 hour, then 2 mg/min.

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If the patient is not accompanied to the hospital or an IV line cannot be put up before he leaves for hospital prophylactic lignocaine should be given as an IM dose of 300 mg.

A note must be made of all drugs given and the route used so that the subsequent management can be planned and note taken of the effect of IM injections on the serum enzymes.

An ECG should be recorded as early as possible because it is the ECG changes that might occur later that are important in the diagnosis.

PAIN

Relief of pain is a very important part of treatment of the acute MI. The autonomic disturbances present may also respond to the relief of pain. Analgesics will also help to control the distress and fear, reduce sympathetic activity and therefore help reduce oxygen demand and so decrease infarct size.

Ventricular fibrillation causes 90% of early deaths

Morphine combined with an antiemetic agent is the most effective analgesic. The dose must be given IV to be rapidly effective and be given slowly in a diluted form. Cylomorph 10 is diluted to 10 ml with N. saline and injected in 2 mg portions watching all the time to give the minimum dose necessary. A fall in BP due to morphine responds well to elevation of legs and administration of IV fluids. The cyclizene which is combined with the morphine for its antiemetic effect does also have a symphathomimetic effect and might induce or aggravate a tachycardia. Metoclopramide is an alternative antiemetic.

AUTONOMIC DISTURBANCE

Sinus bradycardia does not require treatment unless it is associated with hypotension or ventricular irritability. The bradycardia may be treated with IV atropine in 0,5 mg aliquots. The heart rate must be monitored to ensure that it is not raised unduly by the atropine. The atropine might need to be repeated at 5 minute intervals up to a dose of about 2,0 mg. If a greater dose is necessary a temporary pacemaker is indicated. A BP at 90 mm Hg systolic will keep the myocardium oxygenated and prevent extension of the infarct.

Acute pulmonary oedema

Acute pulmonary oedema occurs when the LV is unable to maintain its normal stroke volume. The diastolic pressure in the LV rises causing an increase in the L atrial and then pulmonary venous pressure and this results in interstitial and intra-alveolar oedema. This is a very distressing condition to the patient who struggles to breathe.

The patient should be sat up or nursed in Fowler's position as this

(i) reduces the venous return to the heart and thus pulmonary congestion
(ii) decreases the work of breathing and increases the vital capacity.

Drugs

(a) Oxygen should be supplied by a 28% mask or nasal catheter.

(b) Morphine is life-saving in this condition.

Early intervention may reduce eventual size of infarct

I vividly recall a patient reputedly allergic to morphine who, after all the drug therapy at my disposal, was dying on me, only to be saved by a "last ditch" stand of giving morphine.

The mechanism of action is

(i) Venodilatation reducing the venous return
(ii) Blunting of the respiratory reflex and reduction in pain and anxiety reduces the hyperventilation.

Small doses are given (5-10 mg) IV because of the poor peripheral perfusion, but be wary of hypotension and respiratory depression.

Aim to: control arrhythmias relieve pain correct autonomic disturbance

(c) Vasodilator therapy: Sublingual nitrates in the form of ISDN 5 mg are given sublingually every 30 minutes for 2 hours and then every 2 hours while the patient is dyspnoic. Watch the BP and keep the systolic pressure $\geq$ 90 mn Hg. ISDN is very effective because of the preload reduction due to venodilatation.

IV vasodilators I feel is a physician's drug and should only be used on the advice of one of them unless the GP has had sufficient experience in their use.

(d) Diuretics Furosemide is the diuretic of choice as it is a rapidly acting diuretic resulting in eventual mobilisation of alveolar and interstitial fluid in an hour or two, but there is also a more rapid reduction in pulmonary arterial pressure occurring in 5-15 minutes, probably related to venodilatation.

(e) Digitalis Because of the danger of toxic rhythm disturbances and debate as to whether digoxin increases or decreases the myocardium's oxygen requirement and possible increase in infarction size, an attempt should be made to control the cardiac failure with diuretics alone.

If intropic support is considered necessary use Dobutamine; 250 mg of Dobutamine is reconstituted with 10 ml sterile water and added to 200 ml 5% D.W. Start with a dose of 5 microgrammes per kg per minute and increase to a maximum of 50 microgrammes per kg per minute or until desired effect obtained. After 48 hours wean off the Dobutamine and start digoxin orally.

Autonomic responses may respond to pain relief

Aminophyllin is contra-indicated in acute pulmonary oedema due to acute MI because of its potential to produce arrhythmias.

Venous Occlusion

Venous occlusion applied in rotation to three of the four extremities might be a useful adjunct to drug therapy. There must be no arterial occlusion and occlusion is rotated every 15 minutes. This therapy should not be necessary as sublingual nitrates perform as a medical venous occlusion.

Ventilation

Physicians tend to ventilate these patients very early. If their patients do not respond to diuretics and nitrates they put them on a pump. Elevating the pO_2 oxygenates the kidneys resulting in a massive diuresis.

Morphine with an antiemetic is the most effective analgesic

Depending upon your location and your expertise, you might develop a lower threshold for ventilating.

There appears to be a delay in the onset of action of the drugs in acute pulmonary oedema, the morphine taking 10-15 minutes to start and the diuresis from furosemide taking up to 60 minutes to start. I have on two occasions called for help from physician colleagues only to have the patient respond before he arrives.

INPATIENT TREATMENT

GPs do not normally have access to Intensive Coronary Care Units (ICCU) but the doctor in the country will have to treat his uncomplicated MI - but help is never further than the telephone away. Of the patients who arrive at hospital 70% will survive no matter what we do, 15% will die no matter what we do and 15% are available for medical modification of their risks.

Ideally all patients with MI should be admitted to an ICCU where they can be monitored for the dangerous first 72 hour period. Depending upon the GP's ability and interest, the condition of the patient and how hungry the physician is, an uncomplicated case can be handed back to the GP after the ICCU phase for further in-hospital and then rehabilitation care.

Mortality recedes exponentially after the first day, but deaths still occur after the discharge from the ICCU. Most of these deaths are related to LV dysfunction, a sizable minority are sudden and unexpected, due to arrhythmias, so extended monitoring is desirable.

In the smaller hospitals without physicians and no ICCU the GP must handle the case on his own.

A BP of 90 mmHg systolic will keep the myocardium oxygenated

All cases must be monitored, actively watched by the nursing staff and reliance not placed on the alarm system of the monitor. The patient must be assessed for any signs of complications. His heart must be regularly checked for a gallop and the neck veins and lung bases checked for CCF and pulse rate and BP regularly measured. Ensure that the IV line is adequate so that IV drugs can be given when necessary, in a hurry. Adequate analgesia must be provided by repeating morphine with cyclizine diluted and given slowly as required, using the smallest effective dose. He should be sedated with a benzodiazepine as all patients with MI are anxious and few will ask for sedation.

While confined to bed he should have prophylactic heparin; 5000 units subcutaneously every 8 hours will be adequate after a loading dose of 10000 units IV. Alternatively heparin can be given IV which is less traumatic; 6250 units IV 6-hourly as a constant infusion after 10000 units bolus.

He will need continuous oxygen by nasal catheter for the first 6 hours. This can be discontinued if no failure or further pain. A mild aperient and a bedside

commode will make things easier for him. He should have regular active leg and breathing exercises. A mobile chest x-ray will help in detecting the earliest signs of CCF. Cardiac enzymes are done and repeated as necessary to make the diagnosis or to look for signs of extension of the infarct. The ECG is repeated daily for 3 or 4 days.

In the first few days the patient must be seen twice daily and specifically questioned and examined with respect to

- pain
- arrhythmias
- BP
- CCF
- DVT

All pain is not myocardial in origin, but should be appropriately treated. The pericardial pain which is often present responds quite well to Indomethacin.

The intermediate phase

This is the phase after the patient has been discharged from the ICCU and is nursed in the general ward. All MI patients should be nursed in the same area of the ward where the surveillance is that degree higher than the rest of the ward. Monitors should be available, ECG machines should be on hand and a defibrillator should be on the spot. The patient is sat out of bed on about the third day provided he has no complications, and the stage of rehabilitation starts while he is in the hospital. He is gradually allowed more time out of bed and is allowed to walk about the ward. His anticoagulation is stopped. Provided there are no contra-indications, a cardio selective beta-blocker is started.

Aminophyllin is contra-indicated in acute pulmonary oedema due to acute MI

He is discharged on about the tenth day having been given a copy of "Taking Care of Yourself at Home after a Heart Attack" which originated from Groote Schuur Hospital, and an appointment is made for stress testing at 3 weeks after the infarct.

TREATMENT OF COMPLICATIONS OF MI

1. ***The arrhythmias***

The correct diagnosis is most important as this determines the necessity and mode of treatment. Important to treat the patient and not the arrhythmia and to try and determine the cause of the arrhythmia as treating this is more effective than treating the result.

(a) *Sinus tachycardia*

Exclude underlying cause viz: incipient cardiac failure, fever, pain, pericarditis or anxiety. If no underlying cause found it should be treated as it causes increased myocardial oxygen consumption and possible increase in infarct size. Best treated by beta-blocker, but beware of LVF. X-ray of chest to exclude CCF is mandatory before commencing beta-blockers.

(b) *Atrial extrasystoles*

In themselves not important but they may lead to atrial fibrillation.

(c) *Supraventricular tachycardia*

Not common arrhythmia after MI. Provided no contra-indications (hypotension or cerebrovascular disease) carotid sinus massage may reverse it. Can also try the Valsalva manoeuvre.

Drug therapy:

i) No CCF or hypotension: Verapamil slowly, IV (if not on a beta blocker) the dose is 10 mg over 10 minutes

ii) Boluses of undiluted IV Verapamil are contra-indicated in CCF, and in patients who have been taking digitalis and/or beta-blockers but it is safe to give an infusion.

Elevating the PO_2 accelerates diuresis in acute pulmonary oedema

30 mg is diluted in 200 ml: use 60 drops per ml dropper, start at 10 drops and increase until rate comes down to about 100 or tachycardia breaks.

Note: Any fall in BP due to IV Verapamil is reversible by giving IV Calcium as a bolus.

(d) *Atrial fibrillation*

Rapid ventricular response controlled with IV Verapamil as in 1(c) above. Patient can be digitalised orally after 48 hours if deemed necessary.

(e) *Ventricular extrasystoles*

Dealt with earlier under Emergency Treatment.

Only 70% of those who arrive at hospital will survive

(f) *Ventricular tachycardia*

This is defined as 3 or more consecutive ventricular ectopics at a rate above 100 beats/min. Treatment is initially by drugs, using the same sequence of drugs as for ventricular ectopics. If the onset is associated

with cardiovascular collapse and unconsciousness, then treatment is by CPR and DC shock.

(g) *Ventricular fibrillation*

VF must be treated by CPR until DC defibrillation can be performed.

During CPR the arterial pressure during chest compression is about 80 mm Hg and between compressions it is 10 to 20 mm Hg. The mean arterial pressure is thus below 70 mm Hg, the figure below which there is unlikely to be adequate perfusion of the ischaemic myocardium.

Mortality recedes exponentially after the first day

The longer the delay in restoring the normal heart beat, the less the chance of survival. Defibrillation should therefore be initiated as soon as humanly possible.

Shock is given at 400 joules and if not successful initially it should be repeated. Synchronisation is not necessary in VF. If the second shock is not successful CPR is continued with the patient intubated and oxygenated. 100 mg Lignocaine is run in and 200 ml of 4,2% sodium bicarbonate is run in rapidly (and 100 ml repeated every 10 minutes). The defibrillation is attempted again and if again not successful, the patient is given 100-250 mg procainamide IV. If not converted the DC defibrillation is repeated.

Fine fibrillation can be converted to coarse fibrillation by giving 1 mg adrenalin IV or by intracardiac injection. The high amplitude fibrillation is more readily reverted electrically. Once the patient has been converted continue with Lignocaine maintenance (4 ml of 10% soln. in 200 ml N.saline at 2 ml/min.).

2. ***Cardiac failure***

Oxygen is given to all cases. A diuretic is given if there is radiological or clinical evidence of fluid retention. Start will 40 mg furesemide IV and double dose in one hour if necessary. If daily diuretics are needed monitor the blood urea and electrolytes in case potassium supplementation is necessary.

Vasodilators such as ISDN or prazosin may be useful if diuretics are inadequate and provided the patient is not hypotensive. If the above measures fail Dobutamine is the drug of choice.

THE PROGNOSIS AFTER MI

We are now in a better position than before to define the low risk survivors of a MI. These are the patients that the GP will care for and help to rehabilitate.

Of 100 patients who survive their hospital stay 10 will have acute episodes of ischaemic pain 24 or more hours after the infarct. Their mortality exceeds 20% in the first year and they need angiography for possible PTCA or CABG. Also during the first five days in hospital another 10 will have evidence of severe LV pump failure and their first year mortality figure exceeds 30%. Of the 80 remaining, 10 will show severe exercise induced myocardial ischaemia on treadmill testing to 70% of max. heart rate (70% HRM) at 7 days or later. Their first year mortality is about 15%.

A further 10 patients can be detected at this stage with severe LV dysfunction by radionuclide venticulography showing a LV ejection fraction of 35%. This examination is not readily available other than at the big centres.

Of the 60 patients appearing fit at discharge, a further 10 will manifest severe exercise ischaemia when they have their symptom limited exercise test at 3 to 6 weeks after infarction. These patients would have a 1st year mortality of about 10%. Those 50 patients who pass all the tests will have a 1st year mortality rate of 2%.

Angiography is less sensitive than exercise testing in determining subsequent mortality, but has its place in those 10 patients with severe ischaemia at rest in the first five days and those 20 patients who showed marked exercise induced ischaemia on exercise testing, where it will indicate those patients needing angioplasty or CABG after which their prognosis is significantly improved.

Rehabilitation starts in hospital

Exercise testing is effective in establishing individual guidelines for the resumption of normal activities in low-risk patients and obviates much of the medically unwarranted disability that can follow an acute MI.

This incidence of exercise induced ECG changes, angina and ventricular ectopic activity is similar at the same heart rate - systolic pressure product under all conditions.

Functional capacity increases substantially in the first 6 months after an uncomplicated MI even in the absence of exercise training. Individually prescribed progressive exercise training starting at 3 weeks after the uncomplicated infarct augments this normal functional increase. Exercise is done at the prescribed heart rate for 20 to 30 minutes three times a week.

An American estimate of 15% of post infarctive patients participate in rehabilitation programmes,

whereas 50% are low risk patients, and could do so.

A symptom-limited stress test done at 6 weeks after infarction and showing no ischaemic changes is the green light for resuming any occupation.

In practical terms these low risk patients are the ones for the GP to handle, to get to rehabilitation programmes and to get back to work. Our practice is to stress test all uncomplicated MI at 3 weeks. The test is a low-level test with maximal heart rate of 70% HRM. The test is of course terminated if there are ECG changes before this heart rate or the patient feels uncomfortable in any way. We are able to divide the patients into those who will enter the rehabilitation programme and those who need to be referred for angiography.

Of those entering the programme, some have no ECG changes at all and they are entered at a pulse rate of 70% HRM. Others with ECG changes that are not conclusive of ischaemia are prescribed a pulse rate of 85% of the pulse rate at which the changes occur or at which symptoms develop.

REHABILITATION PROGRAMME

The programme is organised by the Heart Foundation and takes place in a school gymmasium and rugby field from 6 to 7 am on three mornings a week. There is a volunteer PT instructor who runs an initial 15 minute stretch sessions, a nursing sister employed by the foundation who brings the defibrillator, suction apparatus and drug boxes. She records BP, weight and attendance. There is a roster of two physicians and sixteen GPs who take it in turn to attend in case of accidents and we all join in the exercise sessions and then move out onto the field with the patients who walk or jog around the field trying to get their heart rates up to and maintained at their prescription figure. The last 10 minutes are taken up by a cooling down game of volley ball.

CPR is unlikely to perfuse ischaemic myocardium adequately

The patients return at 6 weeks for a symptom limited stress test where they are stressed to 90%. HRM. and again a negative test allows the patient a higher pulse rate prescription, a positive test is referred for angiography and an equivocal test has the pulse rate at which changes occur or symptoms develop, modified to 85% of this figure.

Where a patient is on a beta-blocker this alters the pulse rate. He is requested to perform his stress test on his full medication. The test is symptom limited, so that he usually tires long before reaching 70 or 90%. HRM and this beta-blocked pulse rate is used for the pulse rate prescription.

PROPHYLACTIC BETA-BLOCKERS

It has been shown that the use of beta-blockers after an infarction markedly improves the prognosis. The patients are not beta-blocked but are given a dose of a beta-blocker. Suggested dosages are

Timolol 10 mg bd
Metoprolol 100 mg bd
or
Propanolol 60-80 mg tds.

The patients are put on this dose for a year and if considered a patient at risk and he suffers no side effects he might continue on this medication indefinitely.

High amplitude fibrillation is more readily reverted electrically

THE LYSIS OF CORONARY THOMBI

When the patient is seen very soon after the infarction, as happens often in GP practice, there is a chance of recanalising the occluded artery.

Ideally up to one hour after the occlusion but the limit for initiating therapy is four hours. the current protocol followed is to give 200 mg hydrocortisone IV. This is followed by three infusions of Streptokinase 600 000 units IV over 20 minutes each. This method reported to recanalise 50% of the occluded vessels. If given by intracardiac catheterisation up to 75% of vessels are recanalised.

The contra-indications to the IV therapy are numerous viz:

Hypertension, recent stroke, haemorrhagic diathesis, active haemorrhage, prolonged CPR, recent streptococcal infection and recent streptokinase treatment.

Side effects include haemorrhage, anaphylaxis and febrile reactions. The results look very promising and with the newer experimental drugs such as tissue plasminogen activator and urokinase being tried, perhaps we will have less side effects. This is possibly a trend towards more active treatment of the infarct.

At time of writing streptokinase costs R230,26 for 600 000 units.

In conclusion I would like to thank Elwyn Lloyd very sincerely who has read through my articles and given advice and encouragement and sent me articles that I as a GP would never normally see. We need this contact with experts of whom he is one of the best.

Treatment of myocardial infarction

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Book Review

Infertility: A Practical Guide for the Physician
(Second Edition)

Editors: Mary G Hammond, MD
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Published by: Medical Economics Books, Oradell New Jersey

Price: R72,00 + GST

This book is published by the staff of the University of North Carolina School of Medicine Infertility Clinic in an attempt to standardise treatment of infertility patients. Invited authors have contributed on all aspects on the evaluation and management of the infertile couple. Subject matter covers diagnostic evaluation through to the doctor-patient relationship in infertility therapy and the usual factors associated with infertility. The chapters dealing with the doctor-patient relationship and the role of the nursing officer are comprehensively covered and have an interesting approach to the problem.

The general standard of the book is high and it would be useful to the general family practitioner dealing with couples with this problem. It is a book to be highly recommended.

Dr Alan Alperstein