G.P. Review Article

The Role of the General Practitioner in the Early Management of Acute Myocardial Infarction *

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SUMMARY

The recent literature on intensive coronary care units is briefly reviewed to show that much of the new knowledge should be applied by the general practitioner who is in the most important position, being the first doctor to see the patient. A much greater responsibility now rests on the GP whose attitude and management require considerable re-orientation.

A regime for the use of lignocaine and atropine is suggested for the treatment of the minor premonitory arrhythmias in an attempt to prevent the fatal major arrythmias, which are mainly responsible for the high early death rate.

By better appreciation of the emergency situation, better decision making and the application of a few therapeutic rules to prevent major arrythmias, it is hoped that the GP will play a major role in reducing the mortality of acute myocardial infarction.

S. Afr. Med. J., **45**, 1103 (1971).

In the past decade much new knowledge has been acquired concerning the early management of patients with myocardial infarction. This has stemmed from the Intensive Coronary Care Units (ICCU) where continuous monitoring of ECGs and the opportunity to observe all aspects of the patients very early in the course of the disease has led to many new observations and greatly improved methods of therapy. The time has come for the general practitioner to apply this knowledge. Being the first doctor to see the patient, the general practitioner is in an ideal position to apply the life saving therapy to the vast majority for whom admission to an ICCU is not feasible.

The following facts summarize some of the key points discovered in ICCUs throughout the world and, more recently, also by doctors working in mobile ICCUs who have been able to study patients at home within a few hours after the onset of a myocardial infarct.[22,23,33]

1. Most deaths from myocardial infarction occur within 12 hours from the onset of symptoms.[20,22,23]

2. Among deaths from myocardial infarction, 60% occur in the first hour.[23] The majority of these are due to arrythmias (ventricular fibrillation or cardiac asystole).

3. Arrhythmias of all types occur in about 80% of patients with myocardial infarction.[13,15,19]

4. Sudden deaths from a myocardial infarction are almost always due to a deranged heart rhythm.[1,3,4,5,9,11-14,19,22,23,34]

5. Minor arrhythmias usually precede the major arrhythmias.[1,3,4,5,9,11-14,19,22,23,34]

6. Prompt recognition and effective control of the warning minor arrhythmias greatly reduce the incidence of fatal arrhythmias. These premonitory derangements in rhythm are ventricular premature extrasystoles (VPSs) which are the prodromata of ventricular tachycardia and ventricular fibrillation, and bradycardia which may precede cardiac standstill or asystole.[1,3,4,5,9,11-14,19,22,23,34]

7. It has been claimed that by the detection and prompt treatment of the earlier dysrhythmias in ICCUs the immediate death rate from myocardial infarction has been reduced by one-third.[9,12,15] It is likely that even greater reduction in mortality could be achieved if effective therapy were applied to patients seen earlier in their homes.

In South Africa ICCUs have been established in relatively few centres so that the majority of patients will not have the benefit of this type of service. In Cape Town there are, at present, approximately 8 ICCU beds in provincial hospitals with a similar number in private institutions. There is no specifically equipped and staffed mobile ambulance service. Patients who are fortunate enough to be admitted to one of these beds, have no 'cover' from the time of the suspected infarct until admission to the ICCU.

Delay in admission to the ICCU varies from one institution to another. McDonald[16] states that only 16% are admitted within 4 hours. Mittra[17] and Lown *et al.*[12] report that the average time in their series was 12 hours between the onset of symptoms and admission.

There is no reason to believe that admission time to South African hospitals is faster, so by the time the patient reaches the ICCU, the greatest risk has often passed. The general practitioner therefore has an enormous responsibility and it is the purpose of this article to emphasize the vital role he has to play. By applying many of the new well-proved observations and principles the practitioner is now in a position to act much more effectively in lowering the mortality and morbidity of this disease.

There is little need to emphasize that a patient with a suspected myocardial infarction must rate as one of the most serious medical emergencies a general practitioner has to face. He should always give top priority to the patient in whom he suspects the attack, interrupting whatever he is doing in favour of this emergency.

Delay in onset of therapy can be reduced by earlier summoning of the doctor by the patient (patient-doctor time) and better appreciation by the doctor of the potential

* Date received: 14 May 1971.

emergency situation in any patient with a suspicious history (doctor-patient time).

As regards the former, it would be difficult to find a mass-media method of education which would not trigger off panic. In fact, the doctor's practice would become unmanageable due to the spate of 'emergencies' that would follow any such education programme. This does not stop the doctor educating his own patients, especially those in the 'higher risk' category. This article, however, is concerned with reducing the doctor-patient time and the prompt application of more effective management.

DIAGNOSIS

This always depends on careful history-taking, since frequently there are no abnormal signs on examination. All retrosternal, arm, chest, neck, lower jaw and epigastric pain should be treated with greatest suspicion. A practitioner should be fully acquainted with the various presentations and characteristics of myocardial ischaemic pain. Sometimes the presentation is atypical; there may be no pain, simply the onset of acute dyspnoea or vague oppressive discomfort often dismissed as 'indigestion'. Nausea and sweating associated with the symptoms are highly suggestive clues.

The diagnosis depends on a very high index of suspicion and on taking no chances. While ECG confirmations must always be sought, treatment must never be delayed when the diagnosis is reasonably suspect. To defer effective therapy until the consultant arrives to verify the diagnosis could cost the patient his life.

EXAMINATION

The physical examination may or may not be revealing. A careful check is made on the pulse rate and the presence or absence of arrhythmias. This means checking the pulse for a few minutes and repeating these observations frequently. The blood pressure is noted. The state of the peripheral circulation (temperature, sweating, colour) are carefully assessed for the possibility of shock. There must be a careful search for signs of 'pump failure'—respiratory rate, basal crepitations, wheezing, raised jugular venous pressure and hepatojugular reflux should all be checked. The heart is examined for its size, the quality of the sounds, the presence or absence of a triple rhythm, systolic murmurs or pericardial friction rub.

A practitioner may or may not possess an ECG machine. If he does he must appreciate that an ECG may show no abnormality when done very early after the onset of symptoms. Only later may serial ECGs and serial enzyme studies confirm or negate the diagnosis of myocardial infarction.

An ECG is extremely useful, but if the GP does not have this facility, this should make no difference to the early management. In fact a negative ECG could be disastrous if it lulls the doctor into a false sense of security.

The doctor should spend time checking the pulse with the specific aim of detecting the presence of extrasystoles and arrhythmias. This is far better than a hurried initial examination and a return visit several hours later with a consultant.

A full and complete history may be continued if the patient is not too ill or sedated and while this proceeds the pulse rate and rhythm should be frequently observed. Always important are factors such as smoking, anxiety, occupation and past history of ischaemic heart disease, diabetes and hypertension. Full details of drugs taken, must be noted; also adverse reactions to drugs used such as morphia, etc. The presence of lung, hepatic or renal disease should always be excluded before using potent drugs.

In a suspicious case, whenever the aforementioned warning signs are noted, it is often wise to start effective treatment immediately, then proceed with the rest of the examination. Prompt treatment and prevention of the premonitory arrhythmias can be life-saving.[1,3,4,5,11,12,20,32] A doctor should not be fooled by the so-called 'mild coronary attack' and adopt a 'wait and see' policy. It is just this type of patient, whose heart is initially undamaged, who may die suddenly from electrical failure. Any experienced GP will be able to recall the tragedy of an unexpected death in such a situation. A 'mild' coronary can only be a retrospective diagnosis, made a few weeks later.

EARLY TREATMENT OF ACUTE MYOCARDIAL INFARCTION

General

The importance of a calm reassuring attitude will not only benefit the patient psychologically but can also improve the prognosis. Fear is associated with an increased outpouring of catecholamines which have the dangerous effect of causing VPSs which may trigger off lethal ventricular tachycardia and fibrillation. All medical discussion must be couched in encouraging and optimistic terms.[12] A patient may not admit to his fear, but it is rarely absent.

A number of principles regarding the administration of drugs will be mentioned before referring to specific treatment. These are based on the recommendations of Harrison and although these refer to the treatment of shock, the principles hold good for the treatment of acute myocardial infarction.[7]

1. All drugs should be given intravenously (unless specifically indicated otherwise). They should be diluted and given very slowly. The advantages are obvious and include rapidity of action and greater certainty about the quantity of the drug in the bloodstream. It is convenient to use disposable syringes leaving one syringe in the vein all the time. If no drug is being administered, 1 ml saline is flushed through the needle from time to time, thus preventing the needle from getting blocked. If a drug has to be administered, the barrell of the disposable syringe, which has saline in it, is simply exchanged for one containing the required drug.

2. All drugs must be given with greater caution and lesser dosage whenever there is renal or hepatic disease or impaired circulation in the presence of shock.

3. Certain drugs act synergistically and potentiate actions of others. The danger of the potentiating action of the phenothiazines is a specific example.

4. All drugs given must be recorded, with the time of administration and dosage noted. It must also be appreciated that once a drug is given, the *status quo* has been altered. Every drug has side-effects of which the general practitioner should be aware.

Drugs Used

1. Morphia. There is no doubt of the efficacy of morphia for pain. It immediately dulls the pain and allays anxiety, which are the patient's main concern. The drug should be given intravenously. By administering it very slowly, diluted and in small doses ($\frac{1}{8}$ gr. or $\frac{1}{6}$ gr. at a time) the optimal effect can often be reached with the minimal dosage, thereby lessening the side-effects of the drugs.[2,19,20] The aim is to dull the pain, rather than to eradicate it entirely which might lead to overdosage.

The side-effects of morphia are as follows:

(*i*) *Shock*: Beck[2] states that acute circulatory deficiency in myocardial infarction is not necessarily due to severely damaged myocardium. He further states that morphia overdosage is commonly encountered in patients transferred to the ICCU and that a quarter grain of morphia administered intramuscularly may be far too much for some patients.

(*ii*) *Bradycardia*: This is a dangerous side-effect and can precipitate the development of minor and major brady- and tachy-arrhythmias. Atropine counters these effects and it should be administered as a routine with morphia, provided there is no sinus tachycardia at the time.[1,12]

(*iii*) *Hypotension*: This frequently follows the administration of morphia and can sometimes be profound.[1,9] For this reason all patients should be nursed and transported flat after receiving this drug. A frequent check should be made on the blood pressure. If the blood pressure drops excessively, elevation of the foot of the bed often helps to raise it. Naturally if a patient is orthopnoeic from acute left ventricular failure, the head of the bed will have to be elevated.

(*iv*) *Nausea and vomiting*: These side-effects can lead to dehydration and acute circulatory embarrassment.[1] Atropine can help counter this. Phenothiazines are sometimes given for their anti-emetic effect. This is not advised as they can provoke severe and prolonged hypotension.

(*v*) *Respiratory depression*: Acute respiratory depression occasionally follows the administration of morphia. In this event, respiration must be supported and nalorphine, the antidote to morphia, given intravenously.

2. Diazepam. It has an hypotensive effect and can induce respiratory depressive effects (usually only in large doses). In elderly people, particularly, the response to the drug is very varied. It is absorbed quickly by mouth and can be given in divided doses of 2 mg at a time. The oral method of administration will lessen the risk of marked respiratory depression and hypotension. Diazepam has proved to be a very effective and useful drug in the early management of cardiac infarction.

3. Phenothiazines. These should be used with the greatest caution, as indicated above.

TREATMENT AND PROPHYLAXIS OF MINOR ARRHYTHMIAS

General

It is now well established that of all the rhythm disturbances that may occur, the most significant are sinus bradycardia and ventricular premature systoles (VPS).[1,3,4,5,9,11-14,19,22,23,24]

Brady-arrhythmias are very frequent following inferior myocardial infarction and if not dealt with may lead to various degrees of heart block and asystole.[1,9] A brady-arrhythmia frequently also encourages the development of VPS and the more dangerous tachy-arrhythmias which the VPS is the most frequent.[12] In the presence of an ischaemic left ventricle the ventricular fibrillation threshold is much lowered so that a VPS which occurs very early and which strikes the summit of the T wave can trigger off a ventricular tachycardia or fibrillation.[12]

The object of therapy is thus to promptly eliminate the minor dysrhythmias which can be the warning signal of forthcoming catastrophic electrical failure of the heart.[12]

Drugs Used

1. Lignocaine. Lown *et. al.*[12,24] were the first to use lignocaine extensively as the drug of choice in eliminating the VPS. They also found it highly effective in the treatment of ventricular tachycardia. In their initial communication where they treated 130 unselected cases of myocardial infarction, they found that none of the patients developed ventricular fibrillation.[12] This they attributed to the ability of lignocaine to suppress the VPS in all cases of their series. However, Lawrie and Bennett, as well as later articles by Lown and others, showed that not all VPS are eliminated by lignocaine. When this drug fails, other anti-arrhythmic drugs must be resorted to.[3,11]

Nevertheless, a vast cumulative experience from ICCUs have confirmed that lignocaine is the drug of choice in the suppression of VPS.[3,4,8,9,14,22]

Lawrie found that 80% of all patients with proved ventricular fibrillation developed this complication in the first few hours. Thus the prompt administration of the drug is mandatory when an ectopic beat or beats are recognized by the GP.

Kostuck and Beanlands[10] state that infusion of lignocaine immediately after acute myocardial infarction is undoubtedly of benefit in the prophylaxis of ventricular arrhythmias. Morgenson's[18] results show that prophylactic treatment with lignocaine significantly reduces the incidence of ventricular tachycardia and ventricular ectopic beats. Valentine *et al.*[24] have initiated a double-blind trial where all patients with suspected acute cardiac infarction are given lignocaine or a placebo by the general practitioner. Their preliminary results show that none of the patients who received an intravenous bolus and intramuscular dose

of lignocaine died in the first 2 hours following the onset of symptoms.[24]

Scott *et al.* point out the usefulness of maintaining blood levels of lignocaine by intramuscular injection when the patient has not immediate access to an ICCU[30,31] Harrison and Alderman[6] claim that prophylactic lignocaine has been effective in reducing the frequency of attacks of ventricular tachycardia in 75% of patients with blood levels of lignocaine greater than 1·2 μg/ml.

Pitt *et al.*[26] found that significant arrhythmias were 3 times more frequent in patients who had not received lignocaine prophylactically after acute myocardial infarction as compared with patients who had been given lignocaine.

These workers support the strong argument for the routine administration of lignocaine for the prophylaxis of the VPS and their sequelae especially when the patient is to be transported with no monitor cover. It is suggested that lignocaine be given as a routine when the pulse rate is above 60, whether VPS is observed or not. It must be noted, however, that Pantridge has recently expressed doubts as to lignocaine's efficacy in the prevention of ventricular arrhythmias in the first hour following a myocardial infarct.[21,27]

The low toxicity of lignocaine has been adequately documented.[3,4,8,9,11,14,27,32] CNS effects recorded include transient drowsiness, twitching, blurred vision, sensations of hot and cold and numbness and with larger doses, apprehension, disorientation and fits have been seen.[3,4,8,9,27,32] However, no circulatory depression or hypotension has been encountered[32] In fact, Bennett[3] states that the sedative and analgesic actions of lignocaine are desirable therapeutic features.

Used in the recommended dosages, the drug is highly effective with the almost total absence of side-effects. There need be no hesitation about administering lignocaine immediately to a patient with a suspicious history and before diagnostic confirmation, provided there is no bradycardia or cardiogenic shock. The life-saving benefit of the drug far outweighs any slight side-effects.

The recommended intravenous dose is to a bolus of 60 - 100 mg (3 - 5 ml of 2% solution).[26,30,31] While the effect on the myocardium is apparent within seconds, the suppressive effect lasts only 10 - 20 minutes. However, 200 mg given intramuscularly (5 ml of 2% lignocaine into each buttock) has been shown to provide effective blood levels for 2 hours.[27,30,31] As lignocaine is metabolized in the liver, it should be given cautiously if any hepatic disease is suspected.

2. Atropine. This drug has proved remarkably effective in the treatment of minor brady-arrhythmias, especially when these are due to excessive vagal action occuring soon after cardiac infarction. This is particularly so after inferior myocardial infarction and when morphia is used.[12,14,22,23,33]

The more advanced degrees of heart block due to ischaemic change to the conducting pathways tend to develop later and may be minimized by more effective therapy earlier on.

Adgey *et al.*[1] found that 61% of cases with posterior myocardial infarction were complicated with brady-arrhythmias when seen within the first hour of onset of symptoms. Moreover, these patients often had severe types of bradycardia such as nodal bradycardia and atrioventricular block of all grades. Atropine given promptly was shown to be highly effective in eliminating all forms of bradycardia and even complete A-V block reverted to normal conduction. By removing a dangerous brady-arrhythmia, many deaths from either A-V block, asystole and secondary tachy-arrhythmias can be prevented.

The immediate administration of atropine sulphate is recommended whenever there is brady-arrhythmia.[1] It should be given intravenously and slowly, in doses of 0·6 mg until a satisfactory ventricular rate is obtained. Often one or two doses suffice but up to 3 mg can be administered. Initially there may be a slight cardiac slowing if small doses are used but acceleration of the pulse usually occurs after 1·0 mg and is maximal after 2·0 mg. The effect of atropine often persists for 2 - 4 hours and if bradycardia returns, the drug may be given again.[1]

It is accepted that atropine is the treatment of choice when VPS is associated with bradycardia, the VPS usually disappearing when a sinus tachycardia is induced. However, if VPS still persists after an atropine-induced tachycardia, lignocaine should be administered.

As indicated earlier, atropine should be given as a routine with morphia in order to block the untoward vagal effects of the drug.

The side-effects of atropine include dryness of the mouth, blurring of near vision, slurred speech, aggravation of glaucoma and urinary retention. None of these are serious enough to contra-indicate giving atropine when there is a bradycardia with a suspected myocardial infarction.

Isoprenaline is also effective in bradycardia but should not be given by the GP because of the great danger of provoking myocardial irritability.

TREATMENT OF MAJOR ARRHYTHMIAS

Diagnosis

A major arrhythmia may be found when the patient is first seen. Without an ECG machine, accurate diagnosis is seldom possible, except for atrial fibrillation. A tachycardia with regular rhythm may be supraventricular or ventricular in origin. There are clinical bedside features to differentiate between the two but these are difficult for the GP to assess, and accurate diagnosis always depends on the ECG.

Schrire and Vogelpoel[29] have, however, made a few useful observations that may help the observant and experienced practitioner to differentiate between these two groups of rapid regular tachycardias. Supraventricular tachycardia can be diagnosed with confidence if the heart sounds are single or normally split, whereas in ventricular tachycardia, there is wide splitting of both sounds. This is because the QRS complex is not widened in the former but very much so in the latter, resulting in asynchronous ventricular contraction. If independent irregular 'Cannon A' waves are present in the jugular venous pulse, as well as splitting of both heart sounds and variation of intensity of the first sound, the diagnosis of ventricular tachycardia can be made with assurance on clinical grounds.[29]

A. Supraventricular Tachyarrhythmias

Supraventricular arrhythmias are usually transient, self-limiting and relatively benign.[19] However it must be remembered that supraventricular tachycardias are the usual dysrhythmias associated with pump failure.

1. Sinus tachycardia. Without an ECG it may be difficult to distinguish this condition from paroxysmal atrial tachycardia or atrial flutter. It often occurs as part of the shock syndrome. Lignocaine will have no effect but will do no harm. No specific therapy is advised. If the condition is due to fear, sedation is indicated. If there is congestive cardiac failure, digoxin should be given.

2. Atrial fibrillation. This is the most important supraventricular tachycardia. Lignocaine has been found to be of little use but will cause no harm.[8] Atrial fibrillation often reverts spontaneously. Intravenous digoxin given diluted and slowly will usually correct or control this arrhythmia. Occasionally electrical cardioversion will have to be used if the ventricular rate cannot be slowed and circulatory embarrassment persists.

3. Atrial flutter and paroxysmal atrial tachycardia. Lignocaine is not usually successful in the treatment of these arrhythmias which often revert spontaneously.[13] Various anti-arrhythmic drugs may be used (digoxin, Practolol, tensilon, quinidine, etc.) but electrical cardioversion is by far the most effective and safest form of treatment for reverting a persistent supraventricular tachycardia which is causing circulatory embarrassment.

B. Ventricular Tachyarrhythmias

Major ventricular arrhythmias carry a far more serious prognosis.

1. Ventricular tachycardias. If a GP is suspicious, but uncertain, that a tachycardia is ventricular in origin, he should have no hesitation in first using lignocaine.[9] Ventricular tachycardia is highly dangerous and the longer it persists the greater the danger of cardiac failure shock and ventricular fibrillation. Lignocaine frequently reverts this dysrhythmia provided adequate doses are given. If it fails, the next drug to use is procaine amide which is best given slowly intravenously, 100 mg (1 ml) at a time up to a total of 1 g. Hypotension is usually induced and may be severe if large doses are used.[22] Before resorting to this and other drugs, the GP should make arrangements for an urgent consultant opinion with ECG. Pennington *et al.*[24] have shown that a thump on the chest can sometimes terminate a ventricular tachycardia and this should be tried.

If ventricular tachycardia is confirmed, it is often best to advise urgent electrical cardioversion and admission to an ICCU for monitoring because paroxysmal ventricular tachycardia often recurs and requires continuous lignocaine infusion.

2. Ventricular fibrillation: This is the most common cause of death. Very occasionally a thump on the praecordium may revert this arrhythmia but without a defibrillator, the outcome is almost always fatal. Any hope of salvage would depend on outstanding resuscitative measures including closed chest massage, mouth to mouth respiration, etc., until a defibrillator arrives.

3. Ventricular asystole: In the absence of an ECG recording, it is impossible to distinguish cardiac arrest due to ventricular fibrillation or asystole. In the circumstance of finding a patient with no palpable pulses and no registrable blood pressure the first procedure should be a thump on the praecordium. Regular thumping has occasionally been proved effective in stimulating electrical activity. If any success is achieved, effective supportive measures must be maintained until an Isoprenaline drip is set up or a pacing electrode inserted into the right ventricle.

Ventricular fibrillation and asystole are terminal events and if any successful reversion is to be achieved, it usually requires an expert team with the necessary facilities for resuscitation, defibrillation, pacing, etc. Even in the best of hands the outcome is disappointing.

The GP is powerless to deal with a cardiac arrest, hence the need to stress that his major philosophy should be prevention of terminal electrical failure by aggressive treatment of the prodromal minor arrhythmias. The occurrence of electrical failure should be regarded as a failure in medical management.

Beta-Adrenergic Blocking Agents

There is no doubt that beta-receptor blocking drugs have earned a place in the treatment of dysrhythmias.[5] However, in the early management of a myocardial infarction, the GP should be fully aware of the actions and untoward side-effects of these drugs.

Beta-adrenergic blocking agents cause depression of the myocardium and can precipitate heart failure in a damaged or ischaemic myocardium. They also cause bronchospasm by their action on the smooth muscle of the bronchi which can be highly dangerous in subjects prone to asthma. Should shock or cardiac failure complicate myocardial infarction, the ventricle requires catecholamines to maintain critical contractile functions. The presence of beta-blocking agents in this situation can be most harmful.[5]

Since one is always dealing with an unstable unpredictable situation in every patient with myocardial infarction, propanolol is contra-indicated in the early management. Any drug which can cause ventricular failure and hypoxia is therefore to be avoided, especially if there are safer agents for treating the dysrhythmias.

Practolol and oxprenolol have an intrinsic sympathomimetic action with a decreased likelihood of depressing myocardial function and both have been successfully used in the treatment of supraventricular tachycardias and the suppression of ventricular ectopic activity associated with acute myocardial ischaemia. Practolol is to be preferred because of its cardioselective action and by virtue of the fact that it, as opposed to oxprenolol, has not been found to cause hypotension.[5,28]

Lignocaine still remains the first drug of choice in the prevention and treatment of ventricular ectopic beats.[5]

If a general practitioner has made a confident diagnosis of persistent supraventricular tachycardia and the circumstances are such that immediate treatment is indicated, then practolol is the drug of choice.[5]

CONGESTIVE CARDIAC FAILURE OF PULMONARY OEDEMA

In pump failure, the patient should be nursed upright.

Drugs Used

1. Digoxin: This is a most valuable drug in improving myocardial contractibility, but must be given with considerable care. Digoxin should be given slowly, diluted and intravenously. Digitalis toxicity must be avoided because of its tendency to provoke ectopic beats. By giving smaller intravenous doses slowly (up to 0·5 mg) myocardial contractility is significantly improved without producing myocardial irritability.

It need hardly be stressed that digoxin should never be given intravenously to a patient already on oral therapy. Smaller doses are required in the presence of poor renal function, old age, shock, hypoxaemia and also if potassium-depleting diuretics are used concurrently.

2. Furosemide: The dosage of this diuretic is dependent on renal state, degree of cardiac failure and pulmonary oedema. Twenty to forty mg intravenously usually suffices, but larger doses can be given safely. The loss of potassium associated with the action of furosemide tends to provoke arrhythmias, especially if the patient is on digitalis. Adequate potassium replacement is important whenever this invaluable diuretic is used in the patient with acute myocardial infarction.

3. Aminophylline: This drug is given in the dosage of 0·5 g very slowly by the intravenous route.

4. Morphia: This should be administered as described earlier.

5. Venesection: Medical or surgical venesection can be attempted in resistant cases of pulmonary oedema.

6. Oxygen.

7. Hospitalization: This is always indicated. Cardiac failure nearly always indicates severe cardiac infarction, a bad prognosis and the need for prolonged and careful intensive coronary care.

THE TREATMENT OF SHOCK

Shock resulting from a severely damaged myocardium must not be confused with hypotension resulting from reflex neurogenic shock, peripheral circulatory failure from morphia overdosage and dehydration.[2] True cardiogenic shock, where the patient is hypotensive, confused, has cold extremities, and is sweating profusely, has an appalling mortality even with the best facilities in an ICCU.

Treatment can be initiated by lying the patient flat, elevating the foot of the bed and by giving various drugs such as atropine, morphia, digoxin and oxygen. Drugs should not be given intramuscularly or subcutaneously because they form depots from which they will subsequently be absorbed and distributed at uncertain times.[7] Without drip equipment and help, it is virtually impossible to institute an intravenous isoprenaline infusion with a minidropper, or other intravenous infusions such as 4·2% sodium bicarbonate, hydrocortisone in big doses as well as volume-expanding fluids.

Pentecost[25] states that with the existing unsatisfactory methods for treating shock, the patient's main hope lies in the fact that it is claimed that early diagnosis of infarction coupled with arrhythmia control, may dramatically reduce the incidence of shock.

DISCUSSION

There can be little doubt that all patients with suspected myocardial infarction should be seen as soon as possible and have continuous ECG monitoring. This would facilitate early and accurate diagnosis of the warning dysrhythmias. This ideal is not attainable at present although highly successful efforts have been made in Belfast.[22,23]

Thus until the ideal situation comes about, the responsibility of early management rests with the doctor first contacted. The GP should appreciate his key role and be fully acquainted with the new knowledge and objectives in the immediate management of his patients.

By applying the following simple rules (see Table I) which are based on well-established observations made in ICCU the GP may well save many lives and lessen the extent of myocardial damage.

TABLE I. INDICATIONS FOR USE OF LIGNOCAINE AND ATROPINE IN THE PROPHYLAXIS AND TREATMENT OF MINOR ARRHYTHMIAS

Indication	Atropine	Lignocaine
1. Ectopic beats (no associated bradycardia)		X
2. As a routine with pulse rate above 60/min		X
3. Pulse rate below 60/min	X	
4. Ectopic beats with pulse below 60/min	X	
5. Ectopic beats after sinus tachycardia is achieved		X
6. Administration of morphia	X	

1. Lignocaine should be administered, without delay, whenever ectopic beats are felt provided that there is no associated bradycardia.
2. There is a strong case to be made for the routine administration of lignocaine, in the absence of bradycardia, to all patients whether ectopic beats are noted or not.
3. Atropine should be given whenever the pulse rate is below 60 per minute. Atropine injections should be repeated if the bradycardia returns.
4. If ectopic beats are present with a bradycardia, atropine is the drug of choice. Once a sinus tachycardia has been achieved the ectopic beats usually disappear.
5. If ectopic beats persist after an atropine-induced sinus tachycardia, lignocaine should then be administered.

6. The routine use of atropine whenever morphia is administered is strongly recommended.

There should be continuous close surveillance by the GP until the patient has been admitted to an ICCU. This implies frequent observations of the pulse and blood pressure, and watchful awareness for the signs of pump failure, shock and the recurrence of arrhythmias when the effects of the drugs wear off. The GP should appreciate that it is his responsibility to give the above treatment and not defer it until a consultant is called or leave it to the hospital staff if the patient is to be hospitalized. Where an ICCU or hospital bed is not immediately available, the GP will have to manage his patient with the abovementioned principles in mind. The risk of sudden death decreases rapidly after the third day; thereafter less frequent visits are needed. In small hospitals, especially in the rural areas, without any ICCU facilities, it is important to train sisters in the principles of modern intensive coronary care, particularly the importance of noting arrhythmias and the use of lignocaine and atropine. Well-trained nurses can play an extremely valuable role. This has been appreciated in the ICCU where the trained sisters occupy a key position.

When the patient has to be managed at home, it is suggested that 2 - 4-hourly visits be made by the practitioner in view of the fact that both lignocaine and atropine, in the dosages suggested, maintain their effect for 2 - 4 hours respectively. This would apply for the first few days and depend on whether rhythm disturbances and other complications had occurred or not.

I wish to thank Dr L. Vogelpoel, part-time Senior Consultant to the Cardiac Clinic of Groote Schuur Hospital, for his invaluable criticism and suggestions in the preparation of this article.

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