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Task Force Report

The pre-hospital management of acute heart attacks

Recommendations of a Task Force of the The European Society of Cardiology and The European Resuscitation Council

Introduction

In 1996, the European Society of Cardiology published guidelines on the pre-hospital and in-hospital management of myocardial infarction[1]. These relate primarily to clinical management specifically of this one condition from the onset of symptoms to the phase of secondary prevention and rehabilitation. The problem of acute heart attacks from a community perspective is, however, more complex and many aspects were not within the remit of this earlier document.

Although 'Heart Attack' has no strict medical definition, it is commonly used to indicate a sudden and potentially life threatening abnormality of heart function. We have chosen to use it as a convenient umbrella term to cover the same spectrum of conditions that most frequently elicits its use by lay people. These are chest pain from prolonged myocardial ischaemia, severely symptomatic cardiac arrhythmias, acute breathlessness of cardiac origin, and — most importantly of all — sudden cardiac death and cardiac arrest. There are advantages in considering the underlying conditions as a group for the purposes of the document for three principal reasons: first, they may all have the same underlying cause and one can lead to another; secondly, the strategies to counteract them have much in common; thirdly, in the earliest stage of a cardiac illness during the pre-hospital phase categorization under a specific diagnostic label may be impossible.

The majority of deaths from coronary disease occur in the pre-hospital phase and most victims do not survive long enough to receive medical help. Despite these two challenging facts, inadequate attention and resources have been devoted to emergency systems in most European countries. Thus patterns of care available to heart attack victims in the initial hour or so have changed little in recent decades and speed of response does not usually match the urgency of sudden attacks. Treatment strategies, even for recognized ischaemic syndromes, need some modification to address the special problems of unstable patients often in the evolving phase of the acute attack who face a journey to hospital under circumstances that may be less than ideal. Other diagnoses may be responsible for sudden cardiac death particularly in the younger and older age groups and may require some modification of routine resuscitation procedures. For all these reasons the encouraging reduction in hospital mortality has not been reflected in community mortality. New strategies are needed if any impact is to be made. This report was therefore commissioned by the European Society of Cardiology and the European Resuscitation Council to supplement the existing advice available on the management of myocardial infarction and other forms of acute heart attack, with special reference to the pre-hospital phase.

A decision was made to include the early in-hospital phase — within the emergency department — as part of our remit. The reasons are threefold. Firstly, we wish to emphasize the need for continuity of care as the patient leaves the ambulance and enters the hospital. Secondly, we are aware that failures of communication between these care modalities often delay important treatments. Thirdly, in many centres specialist advice and treatment become available only after patients reach the cardiac (coronary) care unit or investigational areas.

The Task Force was set up by the European Society of Cardiology and the European Resuscitation Council. It was the first to be set up jointly by the two organisations, an appropriate innovation for a logistical challenge that is multidisciplinary, involving ambulance services, general practitioners, emergency physicians, intensivists, anaesthesiologists, internists, and of course cardiologists in all European countries.

Key Words: Acute myocardial infarction, acute coronary syndromes, sudden cardiac death.

Task Force members are listed in the Appendix.

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The epidemiology of acute myocardial infarction and sudden cardiac death

Much of our knowledge of the epidemiology of heart attacks derives from the WHO MONICA Project[2]. In European countries around 40% of all cause mortality

before age 75 years is caused by cardiovascular diseases, independent of the different levels of total mortality[3]. The high variation of ischaemic heart disease mortality is a function of both the incidence of acute heart attacks (probable myocardial infarction) and the case fatality rate (number of fatal cases per 100 total cases). In 29 MONICA populations (age 35–64 years) the mean 28-day case fatality rate from episodes thought to be due to acute myocardial infarction is formidably high, at 49% for men and 51% for women[2], increasing with age.

Despite the international mortality differences, the proportion of case fatalities at different stages during the acute event are very similar in all centres. On average, one third of all cases of myocardial infarction are fatal before hospitalization[2,3], most of them within the first hour after onset of acute symptoms. The *proportion* of deaths occurring out of hospital is very high, particularly in younger people. Norris has recently presented data derived from three British cities[4] in which the ratio of out-of-hospital deaths to in-hospital deaths from acute coronary events (which excludes heart failure) ranged from 15·6: 1 in the youngest cohort aged less than 50 to 2:1 for the oldest group who were aged 70 to 74. A similar trend has been observed in data derived from the population-based MONICA Augsburg Myocardial Infarction Register, although in that city and the two surrounding rural districts the ratios were somewhat less striking (Table 1a and 1b). If this represents a failure of pre-hospital care, the failure is particularly notable in the young and middle aged.

A more detailed account of the sequence of events in the first few hours after acute myocardial infarction is obtained from the MONICA register from Augsburg, one of the MONICA collaborating centres[5] (Fig. 1). This register includes 25–74 year old cases and collects specific information on the pre-hospital phase, additional to the MONICA core design. The Augsburg data highlight the logistical difficulties facing those who seek to improve the prognosis. The 28-day case fatality rate in 3729 cases of acute myocardial infarction of both sexes and all the age groups was 58%. No less than 28% of the total number had died within 1 h of the onset of symptoms, 40% by 4 h, and 51% by 24 h. Sixty percent of all deaths occurred outside hospital, 30% in hospital on day 1, and 10% on days 2–28. Only 10% of pre-hospital deaths were seen alive by a doctor and nearly 60% died unwitnessed.

Patients with acute myocardial infarction who survive long enough to enter hospital undoubtedly benefit from new treatments introduced into routine practice within the last decade or so. These have resulted in a fall in hospital mortality[6], and improved long-term survival[7]. Unfortunately the impact on community mortality rates is influenced only marginally by this success, as a relatively small proportion of potential victims reach hospital to benefit from recent advances. Indeed, no detectable fall in the case fatality rate has been observed in MONICA centres over the years 1985–94 in Augsburg[8] or over the years 1985–91 in Glasgow[9]. A greater investment in hospital treatments

Table 1(a) Ratio of out-of-hospital to in-hospital deaths from acute manifestations of coronary heart disease (deaths from heart failure are not included). (Derived from[4] Norris RM for United Kingdom Heart Attack study collaborative group)

Age (years)	Out-of-hospital fatal events	In-hospital fatal events	Ratio out-of-hospital in-hospital fatal events
<50	78	5	15·6
50–54	67	10	6·7
55–59	115	28	4·1
60–64	202	65	3·1
65–69	313	114	2·7
70–74	397	195	2·0

Table 1(b) Ratio of out-of-hospital to in-hospital deaths from acute manifestations of coronary heart disease (MONICA category 'non-classifiable sudden cardiac deaths' are included). (Derived from population-based MONICA Augsburg Myocardial Infarction Register 1985–1994)

Age (years) (men and women)	Out-of-hospital fatal events	In-hospital fatal events	Ratio out-of-hospital in-hospital fatal events
25–34	22	6	3·7
35–44	109	43	2·5
45–54	386	176	2·2
55–64	942	582	1·6
65–74	1811	1508	1·2
All	3270	2315	1·4

(for example, primary PTCA; or newer, more expensive, and marginally more effective thrombolytic agents) is therefore unlikely to result in any appreciable fall in total mortality. These technological developments should not be discounted. They are valuable to individuals who reach hospital both in terms of early and later case fatality. But improvement in current strategies and the development of new ones are needed to influence the larger number of pre hospital deaths. Improvements in existing services could prevent many of the 12% of deaths that occur between 1 and 4 h from symptom onset and the additional 11% who die between 4 and 24 h[5]. The even larger attrition of the first hour calls for initiatives that are not available in most of Europe at present, but the greatest bar is complacency rather than cost. The mechanism of deaths within 4 h will often be ventricular fibrillation, and in the later ones cardiogenic shock: both could be influenced by the energetic application of prompt defibrillation and reperfusion therapy. Thus epidemiological data suggest that greater deployment of resources for pre hospital care has more potential for reducing the case fatality rate of acute myocardial infarction than has the intensification of treatment in hospital.

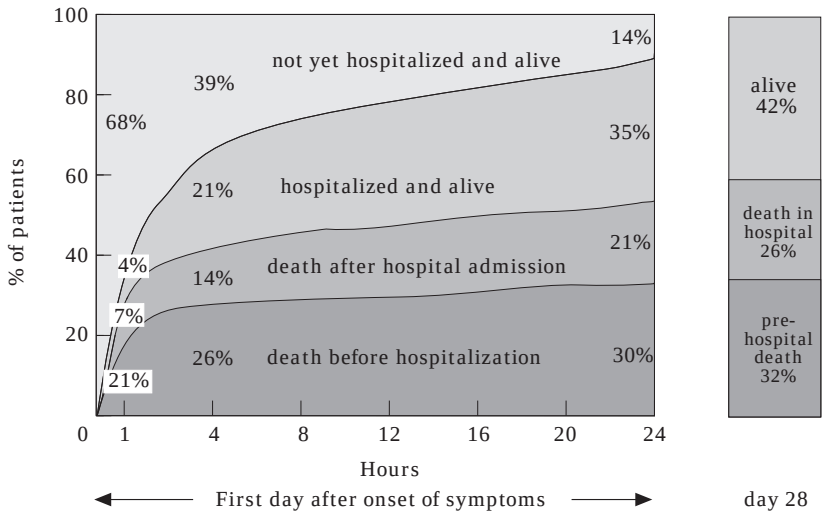

Figure 1 **Survival in the first day after acute myocardial infarction. Updated English version of Fig. 3 from Löwel *et al*.[5].**

Pathophysiology with special reference to the influence of time

Muscle jeopardy and necrosis in acute coronary syndromes

Acute myocardial infarction is caused by a sudden and prolonged reduction of coronary blood flow that in turn is the consequence of the abrupt compromise of a major coronary artery or branch. Thrombus formation over a plaque is the major mechanism causing vessel closure but spasm and embolization of thrombotic material into the intra-myocardial vascular bed may contribute. Angiographic studies show that vessels occlude at the sites of mild to moderate stenosis in about 70% of cases[10]. Angiography has no predictive value for the site of a future occlusion.

Abrupt coronary obstruction leads to transmural ischaemia within the area at risk determined by the coronary anatomy. The jeopardized myocardium develops irreversible changes starting in the subendocardium and progressing outwards. This progression of necrosis has been termed the 'wavefront phenomenon'[11]. In anaesthetized dogs, infarct size increases with duration of coronary occlusion for up to 6 h. After 6 h, reperfusion has no effect on infarct size. The temporal and spatial progression of necrosis across the ventricular wall represents a fundamental pathophysiological phenomenon. The most significant implication of these experimental observations is that the salvage of tissue is a time dependent phenomenon. There is no reason to believe humans differ in regard to time dependency. Indirect evidence suggests that in humans average infarct size without reperfusion therapy is about 20% of the left ventricle. If thrombolytic treatment is started 1 h after onset, 70% of jeopardized myocardium is salvaged, but myocardial salvage is 0% for thrombolysis initiated 5 h after onset[12,13].

In man, several circumstances can change the time course of myocardial necrosis, including preexisting collaterals, ischaemic preconditioning, and age. Preexisting collateral circulation which may limit infarct size[14] can be visualized at angiography in about one third of the cases of acute myocardial infarction[15]. Collaterals developing after infarction do not affect infarct size but may mitigate left ventricular remodelling and reduce the possibility of cardiac failure. Preexisting collaterals may also extend the benefit of reperfusion to patients treated after 6 h[16]. In animal models, brief periods of coronary occlusion increase tissue tolerance to subsequent prolonged vessel closure — the so-called 'preconditioning' phenomenon. ln man, patients with a history of preinfarction angina tend to have less myocardial damage, less cardiac failure, and therefore lower mortality with better left ventricular function at follow-up[17]. Advanced age seems to be associated with increased susceptibility to myocardial injury[18]. Elderly patients may have appreciable damage in the affected zone with commensurate mortality despite sustained arterial patency, possibly because of a greater susceptibility to calcium mediated or oxidative damage. This increased risk may be seen from the decade 65 to 74 years and is most pronounced in those older than 75 years.

Chest pain is variable and subjective and its onset may not coincide with coronary occlusion. Limited benefit following myocardial reperfusion in acute myocardial infarction may result from an underestimation of occlusion time. On the other hand, coronary occlusion may be intermittent despite the presence of continuous pain. Intermittent spontaneous reperfusion may prevent or limit myocardial damage and benefit may then follow from relatively late therapeutic interventions[19].

The following summarizes the available data on the progression of myocardial necrosis that have practical implications for clinical management:

- Although there is variation between species, animal experiments have shown that after 6 h of persistent coronary occlusion only 10–15% of ischaemic myocardium is still viable. Reperfusion beyond 3 to 4 h is then unlikely to result in the salvage of any significant amount of myocardial muscle.
- In humans, a very similar time course usually exists for true salvage. Benefits from reperfusion after this time window must be attributed to different mechanisms.
- Several factors, including thrombus dimensions and structure, can substantially influence the time-window for effective reperfusion in an individual patient. The time window is shortest in previously healthy individuals in whom an artery is abruptly occluded. Previous exertional angina through induction of collateral growth or recruitment of preexisting collaterals, may limit infarct size, and in some cases ischaemic preconditioning may also be protective.
- Intermittent anginal pain at rest as a prodromal symptom characterizes a patient with intermittent occlusion and intramyocardial emboli of activated platelets[19]. Such patients develop infarction by coalescing small focal areas of necrosis of differing ages and the time window for intervention is extended.

Malignant arrhythmias in acute coronary syndromes

Despite the reduction in mortality from ischaemic heart disease in most Western European countries, sudden cardiac death remains a major medical and social problem. More than half the patients with known ischaemic heart disease die suddenly[20,21]. Of those who come to the attention of clinicians, sudden death is the initial presenting event in nearly one third and is associated in the majority of cases with malignant ventricular arrhythmias, usually ventricular tachycardia or ventricular fibrillation.

The incidence of primary ventricular fibrillation (i.e. in the absence of severe haemodynamic compromise) is highest during the very early stages of acute ischaemia, and even with established infarction is rare after the first 4 h[22]. Ventricular tachycardia usually occurs later than 4 h after symptom onset[23] but is not a stable rhythm. Degeneration of ventricular tachycardia is a frequent cause of ventricular fibrillation that appears more than one day after the onset of infarction. Asystole may be a primary arrhythmia or the end result of ventricular fibrillation that has degenerated to an imperceptible amplitude.

Primary sudden death

Up to 20% of patients who suffer sudden cardiac death have no detectable heart disease[24]: the mechanism often remains unknown. Recurrence rate is high after aborted sudden death that was not associated with myocardial infarction[25]. Thus the long term prognosis after primary sudden death is worse than for survivors of cardiac arrest secondary to ischaemic heart disease.

Access to care

From a practical viewpoint, the two most important ways in which heart attacks may present are chest pain due to myocardial infarction and cardiac arrest due to ventricular fibrillation. Appropriate treatments are, respectively, coronary reperfusion and early defibrillation. Both require rapid access to the Emergency Medical System (EMS) but with different priorities. For a patient with cardiac arrest, the need is for a witness only to recognize that an emergency has occurred that requires immediate attention, and for the availability of appropriate first aid followed by rapid defibrillation: speed and simplicity rather than precision and complexity! For acute myocardial infarction, an appropriate response requires greater public knowledge to understand the implications of cardiac pain, with the early availability of a medically competent team to offer accurate diagnosis and reperfusion therapy if indicated. An optimal system must therefore achieve the twin aims of rapid response for cardiac arrest and precision in diagnosis of acute myocardial infarction.

The current situation for meeting these needs in Europe varies widely between countries and within countries, as well as between urban and rural areas. Recommendations for improvements must be made on the basis of what is desirable, yet must also be pragmatic. Where optimal systems cannot be achieved in the foreseeable future, progress can always be made towards the ultimate goal. Many obstacles to progress can be corrected at little or no cost by better organization and by modifications to outmoded laws and practices. Recommendations will therefore be classed as 'basic' which is regarded as the least that is acceptable as an interim standard, and 'optimal' which should be attainable as soon as possible (see section on Principal recommendations).

Delays in providing treatment for cardiac emergencies

Patient decision time

The interval from the onset of symptoms until medical assistance is sought varies widely. Despite widespread public education, reports on patient delays have demonstrated only small trends to shorter time intervals[26]. Decision time is not closely related to knowledge of heart symptoms. Symptoms are often interpreted incorrectly[27] because of psychological defence mechanisms such as denial[28] or displacement and rationalization[29];

but responses are influenced by severity of pain[30], the emotional reactions to it[31], and the degree of left ventricular dysfunction[32].

Doctor decision time
Although call-to-needle times can be very short when general practitioners give thrombolytics pre-hospital[33], many studies have shown that the involvement of the majority who do not themselves give thrombolytic therapy in the management of myocardial infarction results in substantial delay in definitive treatment given after arrival in hospital[34,35]. Calling a general practitioner alone in response to a cardiac arrest may be even less appropriate in countries where few are equipped for defibrillation. Little information is available on the potential value of general practitioners playing a supporting role in coordination with the emergency services but there must be many situations in which this can be of value.

Dispatching
The nature of any response to the request for help in the event of chest pain requires clear guidelines. It will be influenced by the training and qualifications of the dispatcher, the way the message is presented, knowledge of any previous medical history, the cost of sending help when a response is not warranted, and the medical and legal consequences of refusing help when in retrospect it may have been justified. A medical background allows some discretion in the interpretation of calls that would not be appropriate for a non medical dispatcher. The dispatcher has four decisions to make: first, whether or not to send an ambulance; secondly, if an ambulance is to be sent the type of ambulance to be deployed; thirdly how much urgency is needed; fourthly whether advice should be given to the caller on actions to be taken meanwhile. The first of these is the most difficult even for experienced medical dispatchers, because the quality of information is frequently too poor for any safe decision *not* to send an ambulance[36]. For this reason and to avert possible legal consequences, dispatchers tend to send vehicles in all but the most obvious trivial circumstances: 'better safe than sorry' is a valid principle. In consequence, many ambulances are dispatched to patients whose complaints turn out not to have been urgent[37]. Many ambulance control centres send vehicles in response to all requests for help whilst others use algorithms to assess the urgency and priority of calls. These have been introduced in several places in Europe and the U.S.A., but evaluation so far has been limited[38].

Ambulance response interval
The ambulance response interval (which measures the duration from call to arrival at the patient's side) of the first or only tier is in general the shortest of all the delays. In some countries, a time limit is set whereby 95% of all ambulance journeys must be completed within 15 min and 80% within 10 min. In others, strict time criteria are being set for selected cases based on the information received and using systems of prioritized despatch. Both of these approaches are in line with the concepts of early defibrillation for cardiac arrest and early reperfusion for acute myocardial infarction. When ambulance provision includes defibrillation and drug administration (especially thrombolysis for selected patients), then the coronary care unit is effectively brought to the patient within the community: the delay to treatment ends at that point.

The chain of survival for cardiac arrest

In no medical emergency is time such a decisive determinant of outcome as in circulatory arrest. The 'chain of survival' concept clearly describes the important links involved[39,40]. The chain is usually regarded as having four links.

Early access
Immediate access to an ambulance dispatch centre is a primary requirement because any delay in calling the ambulance service inevitably decreases the prospects of survival. The initial contact should not be with a physician, unless he/she has the role of first tier in the EMS and has a defibrillator. In most European countries, access to the EMS is achieved by means of a single dedicated telephone number. The European Council has agreed that a uniform number '112' should be used throughout Europe by 1997, but this has not been widely implemented nor promoted[41]. The caller's description of the problem should influence the degree of priority that is accorded preferably by the use of one of the evaluated algorithm systems: the dispatcher should be alerted by any suggestion of impaired consciousness and should not be reassured by the statement that the victim is breathing, as gasping may continue for minutes after circulatory arrest. Convulsion and vasovagal collapse may cause confusion.

Early cardiopulmonary resuscitation (CPR)
Investigators in Europe and the United States have demonstrated that bystander CPR extends the period for successful resuscitation, and provides a bridge to first defibrillation. It has been estimated that at any point in time between collapse and first defibrillation, bystander CPR at least doubles the chance of survival[42,43], with the possible exception of the first few minutes[43]. Unfortunately, in most European countries bystander CPR is carried out in only a minority of cases.

Early defibrillation 1
In most instances ventricular fibrillation is the initial rhythm associated with circulatory arrest. As time passes, the waveform of ventricular fibrillation loses amplitude and frequency until no deflections can be detected. Electrical defibrillation is the only effective therapy for ventricular fibrillation, and the interval between the onset of the arrhythmia and the delivery of the first defibrillating shock is the main determinant of

successful defibrillation and survival. The possibility of successful defibrillation decreases by more than 5% per minute from the time of collapse. To achieve early defibrillation, it is mandatory that people other than doctors be permitted to defibrillate. In particular, all first tier ambulances should be equipped with defibrillators, and ambulance personnel should be proficient in their use[44]. Non-medical ambulance personnel can be trained in defibrillation in as little as 8–10 h, provided they have good training in basic life support. The important goal of facilitating early defibrillation, with all emergency personnel responding to cardiac arrest being trained, equipped, and permitted to use the modest skill, is still to be widely implemented in most European countries.

Early defibrillation 2. Automated external defibrillation

The automated external defibrillator (AED) can be employed by persons with a limited training targeted to use of the equipment, but without sufficient knowledge for a reliable diagnosis of ventricular fibrillation[45]. This makes it possible to bring the defibrillator to locations with large crowds such as stadiums, airports, shopping malls, and railway stations, where trained first aid personnel can employ them rapidly and in locations where EMS intervention is almost impossible such as airplanes or cruise ships.

Early defibrillation 3. Immediate defibrillation by first responders

Because a considerable time may elapse between the onset of ventricular fibrillation (VF) and the arrival of the emergency medical services, immediate defibrillation by individuals who can be classed as 'first responders' may implement the ideal of early defibrillation[46]. A first responder may be defined as a trained individual acting independently but within a physician-controlled system. The availability of AEDs makes first responder defibrillation a practicable option. Target groups to deliver immediate defibrillation with an AED could include firefighters[46], police and security personnel[47], lifeguards, and flight attendants[48]. Every working day these personnel encounter many members of the public at risk from heart attacks. Although immediate defibrillation by first responders is the logical step after implementation of defibrillation by ambulance personnel, at present no conclusive evidence can show that bystander defibrillation significantly increases survival rates. A few cases of successful defibrillation in-flight and in a railway station have been recorded, and it has been demonstrated that even small differences in call-to-shock time achieved by equipping policemen with defibrillators are critical determinants in the restoration of spontaneous circulation and discharge alive from the hospital[47]. Implementation of programmes for first responder defibrillation should be carefully planned and critically evaluated before wide ranging recommendations can be made. Nevertheless, a recent advisory statement of the International Liaison Committee on Resuscitation (ILCOR) clearly advocates this approach[49].

Early advanced care

In many instances, CPR and defibrillation alone do not achieve or sustain resuscitation, and advanced cardiac life support is necessary further to improve the prospect of survival. In some systems, endotracheal intubation and intravenous medication are not provided out of hospital, while in others advanced life support is available from the first tier of the ambulance service, or more commonly by a second tier. Transportation to the hospital intensive care unit should not be allowed to interrupt appropriate advanced care.

Emergency medical systems in Europe

There is a wide variety of emergency medical systems:

- 1-tier systems delivering only basic life support (BLS) by an emergency medical technician (EMT)
- 1-tier systems delivering BLS and defibrillation by an emergency medical technician–defibrillation (EMT–D)
- 1-tier systems delivering BLS and advanced life support (ALS) by paramedics, doctors and/or nurses
- 2-tier systems delivering BLS followed by ALS by doctors, paramedics and/or nurses
- 2-tier systems delivering BLS and defibrillation, followed by ALS by doctors, paramedics and/or nurses

The structure and organization of the emergency medical systems in European countries is summarized in Table 2. In the majority of European countries, doctors have an active role in pre-hospital emergency medical care as part of the first or of the second tier. In England and Wales all emergency ambulances have at least one paramedic, whilst in parts of Scandinavia paramedics serve as members of the second tier. An experienced nurse is part of the crew of every ambulance in the Netherlands. The availability of physicians in the field may be the reason for the legislation delaying the implementation of defibrillation by ambulance personnel in too many countries, yet this practice may still improve the prospects of early defibrillation. The wisdom of such legislation must therefore be questioned.

Some of the wide variety of approaches and organizations are better suited than others for responding to circulatory arrest (Table 2). When a two-tiered system exists, the training level of the first responding ambulance personnel may not permit recognition of ventricular fibrillation and subsequent defibrillation. In this situation the time taken for arrival of the second tier causes an unacceptable delay. Two solutions should be considered: either improve the training of first responders to enable them to diagnose and treat ventricular fibrillation and carry out defibrillation[44], or introduce automated external defibrillators (AED) within the first tier. The latter solution has been evaluated and proved successful[50] and with recent models easier to use and safe[46]. The situation is least favourable if a single-tier system exists without the possibility of pre-hospital defibrillation: this must be a priority for change. Best performance, in terms of survival, has been achieved by

Table 2 EMS system in European countries

Country	1st tier	2nd tier	Emergency phone	Who is allowed to defibrillate
Austria	emt	md	144	md, emt(*)
Belgium	emt-(d)	md	100	md, rn, emt-d
Bulgaria	md	md	150	members of resuscitation team
Croatia	md	—	94	md
Czechia	emt	md	155	md, pm
Denmark	emt-(d)	(pm)	112	dr, rn, emt-d
Finland	emt-(d)	md	112	everybody trained
France	emt	md	15	md, rn, emt-d
Germany	emt-paramedic	md	112	md, pm
Greece	emt	—	166	md
Hungary	emt-(d)	rn/md	104	md, pm, emt-d
Iceland	emt-(d)	md	0112	md, emt(*)
Ireland	emt	—	999	md, rn, pm
Italy	emt	(md)	118	md, rn
Netherlands	rn	—	06-11	md, rn
Norway	emt-d	md/pm	113	md + assistant in function
Poland	md	md	999	md, emt(*)
Portugal	md	—	115	md, emt(*)
Romania	rn/md	md	06	md, rn
Russia	md	—	03	md/representative
Slovakia	md/pm/rn	md	155	md, rn, emt-d, pm
Slovenia	md	—	94	md, rn, emt-d
Spain	emt/rn/md	—	061	md, rn no law
Sweden	emt-d	pm	112	md, rn, emt-d
Switzerland	emt	(md)	114	md, rn, pm
Turkey	md	—	118	md
U.K.	emt-(d)	(pm)	999	no law
Yugoslavia	md	md	94	md, rn, emt

md = medical doctor; rn = nurse; pm = paramedic; emt = emergency medical team; emt-d = emergency medical technician qualified for use of AED; Abbreviations between brackets indicate local variations in the country; (*) in presence of a doctor. For credential to defibrillate, the minimum training level is mentioned.

two-tiered systems with AED availability in the first tier, and well trained paramedics or emergency physicians in the second tier[51].

Legislation relating to basic life support and defibrillation

For historical, organizational, and political reasons, legislation relating to resuscitation and defibrillation varies greatly within European countries. Information about legal regulations was collected from 28 European countries. Throughout most of Europe, providing CPR when indicated is an intrinsic part of the duties of all who respond to cardiac arrests as members of the emergency system. In virtually all European countries, every health care provider and everyone who has been trained in CPR has a moral and sometimes a legal obligation to offer help, according to the legislation relating to 'non-assistance to endangered persons'. The Belgian law is cited as an example, but other countries have similar legislation[52]. In 21 of the 28 countries that were surveyed, anyone (or anyone who has been instructed) is permitted or at least not forbidden to initiate CPR.

In the majority of European countries, defibrillation is considered to be a medical procedure. This is a reflection of the historical and continuing involvement of doctors in out-of-hospital emergencies and disasters. Delegation to non-medically qualified personnel of acts that are usually performed by doctors is, however, legally possible in many European countries if a doctor is not immediately available. In countries where historically only ambulancemen and paramedics are present in the field, the implementation of early defibrillation by ambulancemen has been readily accepted. In countries where a medical presence is common in the second or even the first tier, the introduction of defibrillation by first attending ambulancemen has progressed slowly. In two of 28 countries the law restricts the act of defibrillation exclusively to doctors. In four countries defibrillation can be delegated only to nurses. In another four countries, ambulance personnel are allowed to defibrillate only in the presence of a doctor. In 16 of 28 countries, defibrillation can legally be delegated to nurses, paramedics, or qualified health care professionals. Two countries have no legal restrictions relating to defibrillation. Thus in at least 10 European countries the law is an obstacle for nationwide implementation of AED programmes by non-physicians.

Other approaches for improving access and decreasing delay

Priority access for high risk patients
Besides improving existing facilities, some new approaches have been adopted to improve access and early treatment for patients with acute cardiac symptoms or circulatory arrest. Rapid access to the system for selected high risk patients (mostly with previous myocardial infarction) has been reported[53]. Not only may immediate access be ensured, but also arrhythmia analysis can be performed and treatment advised if appropriate. Specialized centres can offer direct access to dispatchers who can draw on computerised histories of their patients and can also compare electrocardiograms transmitted by patients with reference electrocardiograms on file. Results suggest a reduction of the median interval between onset of symptoms and arrival in hospital to 1 h for patients in the system compared with 3 h for the general population and also an improvement in 1-year post infarction mortality. Controlled scientific evaluation of this concept is not yet available, however.

Telephone CPR
The outcome of resuscitation is consistently better if basic life support is started by bystanders. Currently this is not performed in the majority of cases of circulatory arrest, partly because of ignorance and lack of confidence and training. Telephone guided CPR by people who have had no previous training has proved feasible[54], and evidence of its efficacy is suggestive though not yet convincingly established[55]. The technique requires intensive training of dispatchers who must use strict protocols.

Pre-hospital triage of patients with acute coronary syndromes and arrangements for care

The recognition that recent chest pain is likely to have a cardiac origin always has therapeutic implications, but this is of special importance with evolving myocardial infarction which requires immediate assessment. The diagnosis of acute myocardial infarction becomes certain only with the passage of time, depending on the patient's developing history, the evolution of abnormalities on the ECG, and a characteristic rise and fall of biochemical markers of myocardial damage. Pre-hospital prediction of the final diagnosis is based only on a snapshot of the clinical history and a single ECG recording, but can be reasonably accurate. With clinical assessment alone, the diagnostic accuracy of experienced clinicians is about 75%[56]. With the addition of the ECG, accuracy may be increased to 90-95%[57,58].

Pre-hospital triage for reperfusion therapy in evolving myocardial infarction

The decision to start thrombolysis or refer a patient for primary angioplasty is made by an integrated evaluation of the history, physical examination, an ECG, and a careful consideration of the risks and benefits of treatment. Making such a decision takes time, so for the majority of cases without special diagnostic difficulty clinical assessment must be carried out only once and by the person making the therapeutic decision. In the most efficient systems this role is undertaken by an appropriately trained physician who arrives with the ambulance. Where patients are seen before hospital admission by medical personnel who are unable to make such decisions, much time may be lost by carrying out a full clinical assessment which is repeated later. Rapid triage requiring urgent treatment rather than precise diagnosis should be the aim under these circumstances, including a decision — where facilities permit — on whether the patient should be taken directly to a specialised cardiac unit or to the emergency department. The most important guide is a 12-lead ECG.

The electrocardiogram (ECG) for pre-hospital triage and treatment

At present, many ambulance systems cannot record, interpret, or transmit a 12-lead ECG, but the importance of these facilities should not be overlooked. Several different methods may be used.

Telephonic ECG transmission
Ideally an ECG will be recorded and interpreted on site shortly after the first contact with the patient. In the absence of a system for immediate ECG interpretation, the tracing may be transmitted to a hospital for interpretation by a physician[59]. This must be accomplished with speed and without loss of quality. High quality transfer may be possible with standard telephone lines or digitised networks for computerized communication. Mobile phones have been used but the results with analogue systems may not be reliable[60]. It should be noted that digital mobile telephone networks use compression algorithms that may significantly distort the ECG signal. The reliability of this system has not yet been fully established. Telephone transmission is unlikely to be appropriate in urban areas because some delay is almost inevitable.

Computerized ECG interpretation
Most of the computerized interpretation algorithms have been developed for standard 12-lead ECGs in non acute settings: the sensitivity of the algorithms may be too high for pre-hospital use. The purpose of pre-hospital ECG interpretation is to identify relatively obvious infarction. In the pre-hospital setting diagnostic

algorithms should have lower sensitivity and good specificity to reduce the risk of inappropriate thrombolysis.

Personnel providing pre-hospital thrombolysis

Ideally, thrombolytic treatment should be given at the first opportunity, by the first qualified person to see the patient, whether this be before or after hospital admission. Personnel providing pre-hospital thrombolysis should be trained in all aspects of the diagnosis and treatment of myocardial infarction. Physicians giving thrombolysis may be cardiologists, internists, emergency physicians, intensivists from a hospital base, or community based general practitioners. In countries that do not have doctors on ambulances, non-physician personnel giving thrombolysis pre-hospital may include paramedics and nurses trained in coronary care, but only if appreciable delay will be averted thereby, and then only for cases in whom indications are unequivocal. It is axiomatic — yet still needs to be stressed — that the final responsibility for vicarious judgements on thrombolysis must remain with the physicians responsible for ambulance care, and that all implications be carefully considered in the light of local needs, practice, and sentiment.

According to the extent of their experience and training, qualified physicians do routinely exercise clinical judgement in the many cases of suspected acute myocardial infarction where the diagnosis is uncertain or relative contraindications are present. The existence of conventional protocols for thrombolytic therapy should not necessarily override a physician's decision in these difficult cases. Due allowance must always be made for clinical skills; indeed survival has been shown to be related to the experience of the physician in charge[61]. Non-medically qualified personnel on the other hand should not carry this responsibility; for them protocols must be rigid enough effectively to replace clinical judgement.

Transporting patients with acute coronary syndromes to hospital

Although little documentation exist on the subject, we make the following recommendations for the transport of patients suffering from heart attacks.

Mode of transportation
All patients with chest pain due to a possible heart attack should be treated as stretcher cases. The position on the stretcher should be determined by what is most comfortable for the patient, but we recommend 40° elevation of the head end of the stretcher as a starting point. Peripheral intravenous access should be achieved at the outset.

Speed of transportation
These patients should be transported to hospital as rapidly as prudence permits but haste must not add to discomfort or anxiety levels. All patients given pre-hospital thrombolysis should be handled very carefully, and it is especially important to protect the head.

Emergency equipment
In addition to appropriate ECG equipment, all emergency ambulances used for transporting patients with acute heart attacks should have a defibrillator (manual or AED) and other conventional resuscitation equipment which must be available and ready for use at all times. The personnel staffing the ambulances should be competent in its use. Monitoring of the cardiac rhythm is mandatory but must not replace continuous clinical assessment. Pulse oximetry may give valuable information. Automatic monitoring of blood pressure may also be useful. All emergency ambulances must be equipped with oxygen delivering systems.

Choice of hospitals for heart attack victims
Patients suspected of having a myocardial infarction should be taken to a hospital that is adequately equipped and staffed for diagnosis, monitoring, and reperfusion therapy: it will not necessarily be the nearest. Some consideration has to be given to distance, however, because the time taken before definitive treatment is given should generally not exceed 60 min from the time the ambulance is alerted. If this time is expected to be exceeded, pre-hospital thrombolysis should be considered (see section on Reperfusion therapy).

Report from the ambulance to the receiving hospital
Hospitals should be alerted to the impending arrival of patients with suspected myocardial infarction[63] because of the need to shorten door to needle time if thrombolysis has not been given in the pre-hospital phase or if preparations have to be made for primary angioplasty. Ambulance crews should state the expected time of arrival and also give accurate information on patients' condition including severity of pain, haemodynamic status, cardiac rhythm, and ECG findings.

Staffing of ambulances
All emergency ambulances should be manned by at least two and ideally three persons qualified to carry out the treatment recommendations.

The hospital interface for acute coronary syndromes

The hospital interface must ensure continuity between the pre- and in-hospital management of patients with acute coronary syndromes. The in-hospital facilities for patients will usually dictate the nature and site of the interface. By whatever means patients are admitted, all diagnostic information obtained before presentation must be available to the receiving team to avoid unnecessary duplication of investigations and the inherent delay in therapeutic approaches.

Delays in hospital[62]
Door delays. Registration procedures should not impede triage of the patient with suspected acute myocardial

infarction. It may be expedited by prior notification of the patient's arrival. Door-to-needle time may be shortened if intravenous (i.v.) cannulation and recording the ECG have already been carried out, and ECG monitoring electrodes have been attached before the patient reaches hospital, but no advantage is gained if door-to-needle time is reduced at a cost of a commensurate increase in patient-to-door time.

Data delays. A standing order should ensure that if an initial or follow up ECG is required, it can be recorded without individual permission being sought. An electrocardiograph and a competent operator should be available at all times. The result should be drawn to the attention of the physician in charge of the case immediately.

Decision delays. Protracted delays in reaching a therapeutic decision may occur if the patient's history is atypical, or if the ECG shows non-specific abnormalities, bundle branch block, or evidence of previous myocardial infarction. Much depends on the experience of the physician. Seeking a second opinion from a cardiologist may cause further delay. Some doubts can be resolved more rapidly by serial ECG recordings. Expert opinion is needed, however, if a choice is to be make between thrombolysis and primary angioplasty.

Drug delays. Thrombolytic therapy should be stored, prepared, and when appropriate initiated in the emergency department (see below).

The admission of all patients with chest pain directly to a cardiac (coronary) care unit (CCU) or intensive care unit (ICU) for evaluation is the preferred option. It is, however, beyond the practical capability of many units. Some patients are assessed in specific chest pain assessment areas, but most hospitals initially receive patients in an Emergency Department (ED) and subsequently arrange admission and transfer if appropriate. The time difference in achieving reperfusion therapy comparing admission to an ED with direct admission to a CCU/ICU may be as long as 45 min[63], but this is unnecessary and inexcusable. Patients should be moved from the ED to a dedicated cardiac care area within 20 min of arrival unless initiation of thrombolysis or other urgent treatment will be delayed thereby. The diagnostic and therapeutic resources of a CCU or ICU must always be immediately available to ensure smooth and rapid access to whatever procedures are needed and to avoid administrative delays.

All areas receiving patients with acute heart attacks must have a dedicated resuscitation room immediately available, with medical and nursing staff skilled in BLS and ALS. Availability of full resuscitation equipment and immediate defibrillation is mandatory. Appropriate treatment for pain, serious acute complications such as acute heart failure, and life-threatening arrhythmias must be at hand and ready for immediate use. External pacing should be available but the limitations of the technique must be recognized by all with access to it[64].

The potential role of an ED in 'protecting' CCU/ICU and in-patient facilities by screening and triaging patients with undiagnosed chest pain must be acknowledged. Over half of all patients presenting to an ED with chest pain may be discharged appropriately[65]. Many inner-city EDs see very large numbers of patients every day with a wide variety of emergency conditions. As a consequence patients requiring specific treatment such as thrombolysis may experience delay[66]. These delays are principally related to the time for triage, for obtaining and correctly interpreting the 12-lead ECG, and for obtaining cardiological or specialist involvement when this is required[34,67]. Where these problems exist, they must be recognized and steps taken to counter the delays as effectively as possible. Methods must be in place for audit of performance and regular rehearsal sessions.

For patients who do not have the advantage of pre-hospital thrombolysis, hospital delays may be of crucial importance especially if the interval from onset of symptoms to hospital presentation is short. 'Fast tracking' systems can be effective in reducing 'door-to-needle time' for patients who can be identified rapidly and unequivocally as being suitable for thrombolysis. These patients will have a clear cut clinical history and ECG changes diagnostic of acute infarction[68]. Selection of suitable patients without contraindications can be made reliably and with acceptable safety without specialist involvement or knowledge using a small set of prepared questions[69]. If immediate transfer to the CCU/ICU is not possible and PTCA is not an option, then thrombolysis can be initiated in the ED by specialist teams or in the absence of contraindications by ED staff working to agreed protocols. Bolus administration of thrombolytic agents can simplify procedures in these situations, but irrespective of the system used, a 'door-to-needle time' of less than 30 min is a realistic target. If the average time is longer for patients without important contraindications the system should be examined and improved.

Unfortunately, not all patients with infarction fall into a fast-track category that gives no diagnostic difficulty. Clinical algorithms to improve diagnostic accuracy in doubtful cases have, however, proven unreliable and unwieldy[70–72]. The single most useful screening investigation remains a good quality 12-lead ECG, but its limitations must be recognized. Whilst about 80% of patients with acute infarction have an ECG at presentation that is clearly abnormal[73], in a proportion the changes are subtle and non diagnostic. Confirmation of the diagnosis may require time, but where suspicion is high, repeat ECGs should be taken at intervals of no more than 10 min for the first 30 min. For the minority of cases in which junior medical staff experience difficulty in the interpretation of an ECG, direct transmission by electronic means for specialist interpretation can help. Fax machines may be useful for this purpose in the absence of a dedicated system[74].

Such measures are appropriate when there is a fear of clinical error; but measures to seek other opinions take time and must be avoided as far as possible.

Treatment of acute coronary syndromes in the pre-hospital phase

General measures for patients without overt complications

Pain relief

Pain should be relieved as quickly as possible. This is a priority because pain will increase anxiety and the resulting sympathetic stimulation will aggravate myocardial ischaemia. Pain should therefore be controlled adequately as soon as possible. Opioids such as morphine (or diamorphine where its use is permitted) should be administered intravenously and titrated until pain is adequately relieved. Subcutaneous and intramuscular injections should be avoided. Nitrates and intravenous beta blockers that may be given for other reasons can contribute to pain relief by improving the underlying ischaemia. Anxiolytics — in particular benzodiazepines — may be given if anxiety is perceived as a major component of the patient's distress, although in most cases the euphoriant effect of an opioid will make this unnecessary.

Treatment of early nausea, vomiting, hypotension, and bradycardia

These common features of the initial phase of acute heart attacks may be due to excess vagal tone and/or the side effects of analgesics, nitrates, and beta-blockers. Antiemetic drugs such as metoclopramide may be used to counter nausea and vomiting. Bradycardia (with or without hypotension) despite the relief of pain and nausea may be improved by the administration of atropine. Persisting hypotension is likely to reflect severe myocardial damage (see section on Cardiogenic shock).

Aspirin administration

Aspirin significantly improves the prognosis of patients with suspected acute myocardial infarction or unstable angina[75]. The efficacy of aspirin in reducing cardiovascular death seems to be similar in patients treated early and late[76]. Thus aspirin (150 to 300 mg, preferably) should be given to all patients with acute coronary syndromes in the absence of clear contraindications irrespective of the delay between presumed onset of symptoms and first evaluation. Since antiplatelet activity may be obtained within 30 min[77] antithrombotic protection should not be delayed until arrival in hospital. Aspirin is simple to administer, it does not require specific monitoring, and as a single dose it is well tolerated. The additive effect of aspirin and fibrinolytics on cardiovascular mortality and the preventive effect of aspirin on the 'excess' of recurrence of myocardial infarction with thrombolysis was observed when aspirin was given immediately before the infusion of fibrinolytic agents[76]. If fibrinolytic therapy is given in the pre-hospital phase aspirin should be administered concomitantly to help prevent early reocclusions.

Heparin administration

Before the widespread use of fibrinolytics and aspirin, heparin was the reference anti-thrombotic treatment for the acute phase of myocardial infarction. A meta-analysis[78] reviewed the results of studies comparing heparin with control. In the absence of aspirin, results in favour of heparin were observed with respect to mortality, stroke, pulmonary embolism, and reinfarction. But the review of those trials in which heparin was evaluated in the presence of aspirin showed at best a modest effect for these endpoints with no benefit on stroke Moreover, the risk of major bleeding was significantly increased by 50%. Heparin as an adjunctive treatment to streptokinase and aspirin has not been shown to improve mortality in two large trials but it did increase the risk of bleeding[79,80]. Urokinase, tPA, and rPA are more effective in the presence of heparin, which is usually recommended as an adjuvant for these agents, but at present no convincing evidence exists for starting heparin in the pre-hospital phase even when fibrinolytics are prescribed, and caution is advised unless or until any added risk of intracerebral bleeding has been quantified.

Pre-hospital beta-blockade

The efficacy of beta-blocking agents in preventing death and reinfarction after myocardial infarction is well established. Many trials and meta-analyses[81–84] have assessed the value of starting intravenous beta-blockade early after the onset of symptoms. A meta-analysis of the trials available to early 1985[84] showed a 13% reduction in total short term mortality ($P<0{\cdot}02$), a 20% reduction in reinfarction ($P<0{\cdot}02$), and a 15% reduction in ventricular fibrillation or cardiac arrest ($P<0{\cdot}05$) and the two subsequent large trials[81,82] were consistent with this evidence. In addition, intravenous beta-blockade reduces ischaemic pain and tachyarrhythmias. Despite these results, experience of beta-blockade in the early phase of myocardial infarction is limited. No evidence of a mortality benefit from early beta-blockade as compared with delayed beta-blockade was seen in one randomized trial[85] but the study was not powered for showing differences in mortality. A significant reduction in reinfarction was observed, however. In general, use of the drugs with thrombolytics seems to be safe, and it is also feasible in the pre-hospital context[86]. They may be considered for tachyarrhythmia or hypertension and as adjunctive therapy for pain relief. For routine use, however, the balance between potential benefit and possible side effects such as hypotension and bradycardia in patients who are also receiving fibrinolytics and/or nitrates should be considered very carefully. The task force consider there is no strong indication for systematic use of beta-blockade before hospital admission.

Prophylactic use of oral or intravenous nitrates

More than 80 000 patients with acute myocardial infarction have been involved in 22 studies comparing early intravenous or oral nitrates with control groups. Two large studies, GISSI-3[87] and ISIS 4[88], contributed most of the patients and reported no mortality benefit. A meta-analysis[88] showed only a 5·5% reduction of mortality ($P = 0·03$). This translates into a saving of 3·8 deaths per 1000 treated. Whether this benefit is sufficient to justify routine use of nitrates is debatable, particularly with the added uncertainties of the pre-hospital phase. Nitrates may be deleterious in cases of right ventricular ischaemia or infarction which may complicate inferior left ventricular changes[89]. Persistent pain or the presence of heart failure may of course be valid indications for their use for patients with these specific conditions, but they are not at present recommended for routine administration.

Prophylactic use of ACE inhibitors

Long term use of ACE inhibitors started a few days after myocardial infarction has been established as an effective treatment to reduce mortality and reinfarction in patients with clinical signs of heart failure or with an impaired ejection fraction[90]. Early treatment with ACE inhibitors is considered relatively safe[91], although it increases the risk of hypotension, cardiogenic shock, and renal dysfunction[88]. Because of these side effects and of the lack of information on the early pre-hospital phase, the Task Force members cannot recommend the prophylactic pre-hospital use of ACE inhibitors.

Prophylactic use of antiarrhythmic therapy

Lidocaine has been advocated to prevent ventricular fibrillation in patients with acute myocardial infarction. Several studies have been performed to test the efficacy of prophylactic lidocaine for this indication. Meta-analyses[92–94] have shown a reduction of approximately 35% in the incidence of ventricular fibrillation but also a non-significant trend to an increase in mortality. Studies restricted to the pre-hospital phase have included data on 7386 patients, but these have not provided any evidence for a reduction in mortality as a result of prophylactic antiarrhythmic therapy. One important point must be made: under the conditions of the trials, defibrillation was immediately available so that the reduction in the incidence of ventricular fibrillation would not have been expected to have been translated into a mortality benefit. Ventricular fibrillation that was not associated with lethal haemodynamic compromise should have been promptly reversed. No advantage could therefore be gained to balance any deleterious drug effects. This would not always be so in the pre-hospital phase: we have, however, no direct evidence of the benefit of prophylactic lidocaine when defibrillation is not an immediate therapeutic option. With current knowledge routine use of lidocaine or other prophylactic antiarrhythmics in the pre-hospital phase cannot be recommended.

Reperfusion therapy

Thrombolysis

Thrombolytic therapy is beneficial by restoring patency of the infarct-related artery and improving the remodelling process, but the clinical benefit depends largely on how quickly and completely reperfusion is achieved.

Hospital trials. The Fibrinolytic Therapy Trialists' (FTT) Collaborative Group has reported an overview of nine randomized trials each of at least 1000 patients with suspected acute myocardial infarction in which fibrinolytic therapy has been compared with control or placebo[95]. For patients presenting with ST elevation or bundle branch block the mortality benefit was 30/1000 for those randomized 0–6 h, and 20/1000 for those randomized 7–12 h from onset. This treatment has been widely assimilated into hospital practice and its use over the past decade has been associated with a fall in hospital mortality[6]. The FTT overview, while confirming that earlier treatment is associated with greater benefit, suggested that the benefit of reducing delays to thrombolysis is relatively modest. The relationship between absolute mortality reduction and time of randomization was represented by a straight line with a negative slope of 1·6/1000/h and an intercept of 35/1000. The complex relationship between mortality reduction and time of administration of thrombolytic therapy may not, however, be represented accurately by a straight line[96]. A meta-analysis of 22 randomized trials of thrombolytic therapy with more than 100 patients has shown that a nonlinear benefit/time regression line provides the best fit to the data (Fig. 2)[13]. The beginning of the benefit/time regression line is very steep, but there is a marked inflection at about 2 h. Both of these results were drawn from a post hoc analysis of non-randomized groups with different characteristics (for example age and severity of symptoms) and a reliable estimate of benefit as a function of delay cannot be established by this means. But there is no doubt that to reap the full benefit of thrombolytic therapy it has to be given as early as possible i.e. at the first opportunity in the community.

Pre-hospital trials. Retrospective analyses of placebo controlled trials of thrombolytic therapy given in hospital cannot tell us by how much the mortality rate would have been reduced had the same patients been given the treatment earlier. Neither can the additional benefit of pre-hospital thrombolysis be inferred from the gradient of the graph shown in Fig. 2. The benefit of earlier thrombolysis can be quantified only with a trial design in which patients are randomly allocated to receive thrombolytic therapy either on presentation in the community or alternatively after admission to hospital. The three largest randomized trials comparing pre-hospital with hospital thrombolysis are EMIP[57] (n = 5469), MITI[97] (n = 360), and GREAT[56] (n = 311). It should be noted that EMIP was terminated prematurely because of lack of funds, and neither MITI nor

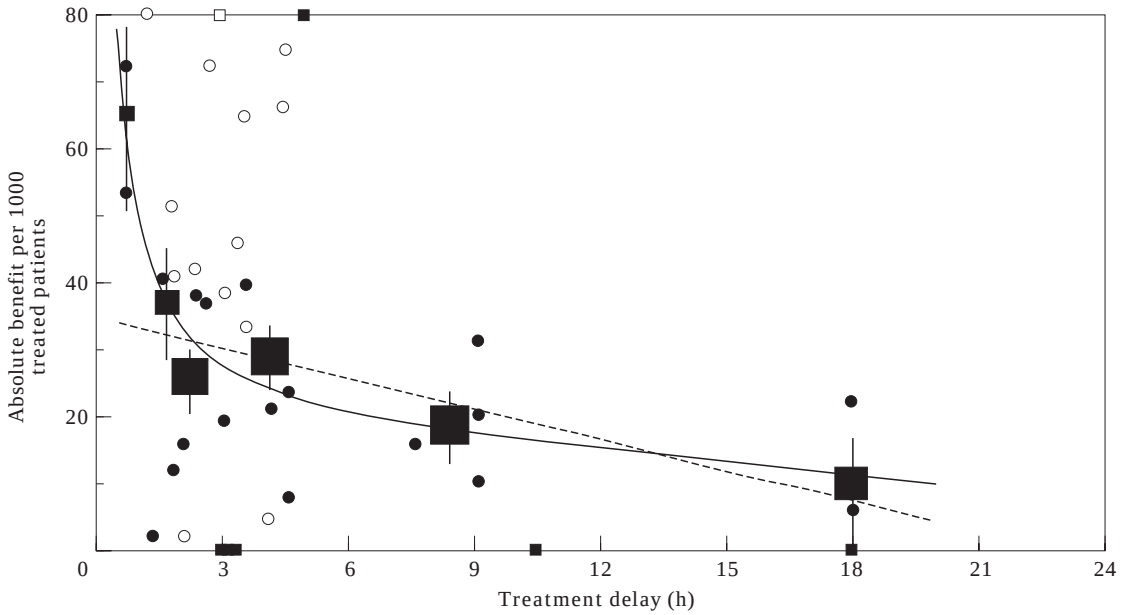

Figure 2 **Absolute 35 day mortality reduction versus treatment delay. Regression equation reproduced from Boersma *et al*.[13]. The linear regression line (broken) and non-linear (continuous) regression line are fitted to the data. The non-linear line provides the best fit. Small closed dots: information from trials included in FTT analysis. Open dots: information from additional trials. Small squares: data beyond scale of x/y cross. Black squares: average effects in six time-to-treatment groups.**

GREAT were designed as trials with a mortality endpoint. None of the trials of pre-hospital thrombolysis has individually shown a statistically significant mortality difference at one month by intention-to-treat analysis, but as well as being small there are practical and ethical constraints on the design and conduct of such trials which reduce their ability to achieve a statistically significant result. A meta-analysis of the three major pre-hospital trials with additional data from five smaller ones has, however, shown a significant mortality reduction with pre-hospital compared with hospital thrombolysis ($P = 0{\cdot}002$), with a benefit/time gradient at 35 days of 21 ($\pm$ SE 6) per thousand per hour[13]. This is the best available estimate we have of the magnitude of the benefit of earlier thrombolysis, being derived from intention-to-treat analyses of appropriately designed trials. Early mortality is not the only consideration. Myocardial salvage and an important effect on remodelling may also reduce the tendency to subsequent heart failure. Follow-up of GREAT showed substantial deferred mortality benefit additional to that seen within the first month[98] whereas in MITI[99] pre-hospital thrombolysis showed no further influence on long-term mortality. With or without late benefit, the evidence from randomized clinical trials of pre-hospital thrombolysis is fully consistent with the large body of theoretical, experimental, and clinical evidence in favour of early thrombolysis.

Implications of the benefit/time gradient of thrombolysis. In terms of its potential for saving life, initiating thrombolytic therapy is as urgent as the treatment of cardiac arrest[100]. Although time is more critical in the latter situation, similar mortality benefits may be expected if both strategies were optimized. As a general policy, treatment should be initiated on site if practicable, and by the first qualified person to see the patient. Thus, thrombolytic treatment should be given ideally in the pre-hospital phase. Where ambulance staffing arrangements have made this policy difficult to implement (for example in countries that do not regularly have doctors in ambulances) strategies should be sought urgently that will allow pre-hospital thrombolysis if the combined journey time and in-hospital delay is more than 60 min, or if the journey time is 30 min or more. In the latter case the overall time saving will usually be in excess of an hour because in-hospital delay, seldom less than 30 min, is also obviated. If thrombolytic therapy is not given pre-hospital, the goal should be to reperfuse the occluded artery as quickly as possible in hospital. In the absence of contraindications, any delay to the start of definitive therapy of more than 30 min calls for a critical examination of the system.

Choice of thrombolytic agent for use pre-hospital. Where pre-hospital thrombolysis is provided by hospital physicians travelling into the community in a mobile coronary care unit, the same drugs may be used as are given in hospital. The medical staff will have daily familiarity with their doses and administration. But where domiciliary thrombolysis is to be provided by general practitioners, each using this treatment only 3–4 times a year, convenience of administration and storage are important. For most general practitioners, the need

for thrombolytic agents or heparin to be administered by slow infusion precludes their use in the community, and one of the agents that can be given by bolus injection deserve consideration.

Primary angioplasty

Coronary flow is restored only after a delay following the administration of thrombolytic therapy, and in a substantial minority of patients flow may not be restored at all. Delays in achieving coronary reperfusion following drug therapy may be circumvented by the use of primary angioplasty, which may also be used for patients in whom thrombolytic therapy is contraindicated. Primary angioplasty yields higher coronary patency rates than thrombolytic therapy, and full patency is achieved immediately the angioplasty balloon is deflated following successful dilatation. But primary angioplasty is clearly a hospital procedure, and there is an unavoidable preliminary 'door-to-balloon' time. Clinical trials comparing primary angioplasty with hospital thrombolysis are encouraging[101], but the full benefits of angioplasty may not be well sustained[102]. To date, the only available evidence comparing pre-hospital thrombolysis with primary angioplasty has not shown any advantage with the interventionalist strategy[103] and there are no randomized trials. Early 'rescue' angioplasty has a role where reperfusion by thrombolysis has failed,[104] but is often unsuccessful[105]. In localities where both pre- hospital thrombolysis and angioplasty are available, local policies for the early management of patients with acute myocardial infarction should be followed.

Management of complications of acute coronary syndromes

Arrhythmias

Sustained arrhythmias occurring in the context of a myocardial infarction may have immediate or longer term prognostic implications. Not only should immediate treatment be considered, but adequate documentation should be achieved whenever it is possible to do so. Therefore a full 12-lead ECG should be recorded if facilities are available and if delay caused by registration will not compromise the safety of the patient. The European Resuscitation Council, after consultation with the European Society of Cardiology, has produced guidelines for the treatment of periarrest arrhythmias[106,107] that are potentially malignant but have not caused clinical circulatory arrest. The guidelines are presented as three algorithms: for bradyarrhythmias, for broad complex tachycardias which can be equated under emergency conditions to ventricular tachycardia unless there is good evidence to the contrary, and to narrow complex tachycardia which can be equated with supraventricular tachycardia including atrial fibrillation (Fig. 3). These guidelines are not intended to override expert assessment of situations that may be complex, but provide advice applicable for most situations.

Bradyarrhythmias. Generally the same principles apply for sinus bradycardia and for atrioventricular block. If a recognizable prelude to asystole is present (such as Mobitz II atrioventricular (AV) block, complete heart block with a wide QRS complex, or pauses of longer than 3 s) transvenous pacing is indicated. This is unlikely to be available in the pre-hospital phase but the perceived need may hasten hospital admission and lead meanwhile to the consideration of the use of atropine, chronotropic catecholamines, or external pacing if this is available. In the absence of any immediate threat of asystole, a heart rate that is unacceptably slow in absolute terms or too slow for the haemodynamic state of the patient (which may occur either as a complication of the infarction or as a side effect of drug treatment) will usually respond to atropine in a dose of 500 μg to 3 mg. Sinus bradycardia or AV nodal block complicating inferior myocardial infarction may best be left untreated if well tolerated, and may even be advantageous in terms of tolerance to myocardial ischaemia. Bradycardia in the acute phase of chest pain may also respond to effective analgesia which can counteract excess vagal tone.

Broad complex tachycardias. Single premature ventricular beats generally require no treatment. Some prolonged or complex arrhythmias such as couplets, or runs of ventricular beats at a relatively slow rate are usually well tolerated and likewise do not require treatment. But if arrhythmias are severe enough to cause or exacerbate pain, hypotension, or heart failure, or are judged to be a possible prelude to ventricular fibrillation, they should be treated initially with lidocaine using an intravenous dose of 50 mg over 2 min repeated to a total dose of 200 mg. One important point should be made: complex ventricular arrhythmias complicating bradycardia should be treated by measures, such as atropine, designed to increase the basic rate, and not by antiarrhythmics. Suitable second line antiarrhythmics of class I or III depend in part on local availability and custom. Repeated administration of one or several antiarrhythmics should however be avoided as far as possible to avoid uncontrollable (and unforeseeable) unwanted effects such as depression of the myocardium or conducting system. Where several doses are needed, drugs with a short half life such as lidocaine or ajmaline may involve less hazard. Cardioversion in the absence of circulatory arrest is rarely indicated in the pre-hospital phase but severe haemodynamic compromise from rapid ventricular tachycardia should be treated by prompt electrical cardioversion after appropriate sedation.

Narrow complex tachycardia. Patients with well tolerated sinus tachycardia and normal or high blood pressure may not require specific treatment in the pre-hospital phase although beta-blockers should be considered (see section on Pre-hospital beta-blockade). Continuing pain or early heart failure must be excluded as a cause of sinus tachycardia. The most common narrow complex tachyarrhythmia after infarction is

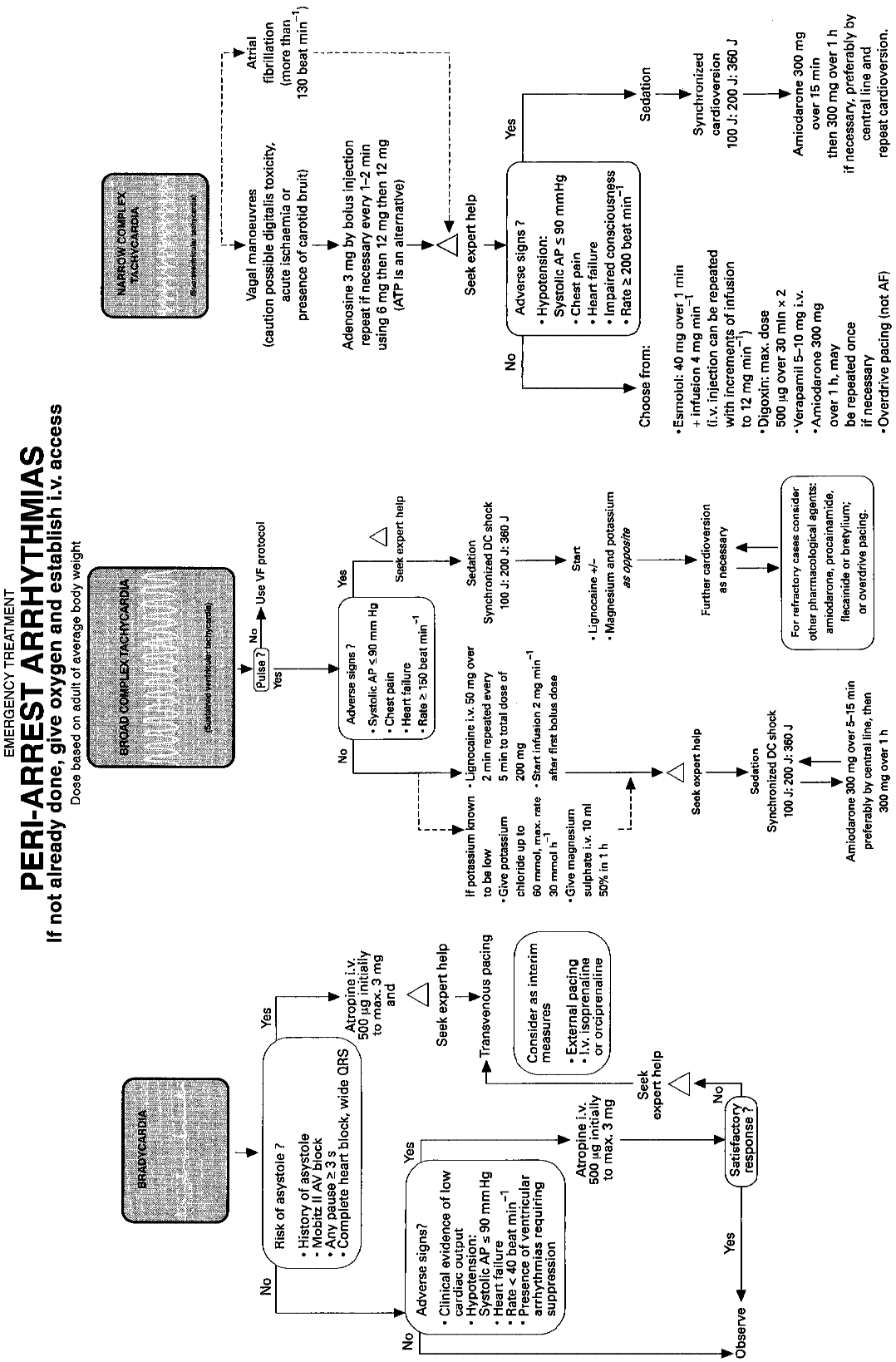

Figure 3 **The ERC algorithm for the treatment of peri-arrest arrhythmias updated in 1998[106,107].**

atrial fibrillation. Urgent cardioversion is indicated In the presence of severe haemodynamic compromise and a heart rate exceeding about 130 per minute. In other cases, intravenous beta blockade (preferably with a short acting agent such as esmolol) may be useful. Diltiazem or amiodarone also have a role, but caution is needed in the presence of hypotension. Intravenous digoxin is likely to act too slowly to be appropriate in the pre-hospital phase. Paroxysmal supraventricular tachycardia is unusual as a complication of acute myocardial ischaemia, but if necessary adenosine as a bolus dose of 6 to 12 mg may be tried. Verapamil, diltiazem, or beta blockers are second line options.

Acute heart failure

All patients with left ventricular failure should receive oxygen by mask or intranasally. In these patients the following treatments can be considered either separately or in combination depending on the response of symptoms: diuretics such as intravenous furosemide, intravenous glyceryl trinitrate or isosorbide dinitrate[108], or oral nitrates at doses sufficiently high to produce a vasodilating effect. These treatments may induce or potentiate hypotension and should be titrated accordingly. This is especially true for patients with higher degree AV block and/or right ventricular infarction. In refractory pulmonary oedema intubation and respirator treatment with positive end-expiratory airway pressure may be life-saving.

Cardiogenic shock

True cardiogenic shock should not be diagnosed until any important brady- or tachyarrhythmias or hypovolaemia that might be contributing to hypotension have been treated effectively. In patients with cardiogenic shock due to right ventricular infarction, volume augmentation is indicated using a test infusion limited to 200 ml of colloid. For other causes of cardiogenic shock, dopamine ($2{\cdot}5–5\ \mu g\ .\ kg^{-1}\ .\ min^{-1}$) or dobutamine ($4–20\ \mu g\ .\ kg^{-1}\ .\ min^{-1}$) may be used alone or in combination with norepinephrine/noradrenaline ($0{\cdot}5–20\ \mu g\ .\ min^{-1}$) or epinephrine/adrenaline ($0{\cdot}5–20\ \mu g\ .\ min^{-1}$). (Note that the doses of epinephrine and norepinephrine are not quoted in relation to body weight.) Dopamine in higher doses transiently increases blood pressure at the cost of a rise in left atrial pressure and a fall in cardiac output[109].

Cardiac arrest

Most cardiac arrests occur in the home with a relative within sight or sound. Rehabilitation programmes, sports groups for coronary patients, and self-help groups can help to train the families of heart attack victims who are at risk of further episodes, but motivation is often lacking. The partners of patients affected are often elderly, a group which is least likely to accept training[110]. Younger individuals, athletes, students, and members of organized groups are much more willing to learn BLS techniques. The focus for offering training should therefore be directed to schools and other learning facilities, sports clubs, companies, or specific occupational groups, such as policemen, railway personnel, and public service drivers. Transport workers have a higher probability than most of witnessing a cardiac emergency. A 'cascade' principle will help those with most aptitude become trainers and instruct an appreciable proportion of the population. In some countries a first-aid course with BLS training is required for obtaining a driver's license, a practice strongly recommended.

Guidelines for BLS have been published by the major resuscitation councils including the ERC[111]. A central problem in the performance of BLS is the aesthetic acceptability of mouth-to-mouth ventilation. The perception of risk of infection poses another barrier to acceptability. Until recently, the only other option was the mouth to nose method, which has similar aesthetic problems. BLS courses participants should be informed that the cardiac arrests they are most likely to witness will be in a close relative within the home. Mouth-to-mouth ventilation would almost always be acceptable in such cases.

Mouth to mouth ventilation is 'unphysiological' since the victim is ventilated with a hypoxic/hypercarbic gas mixture[112]. In addition it is often performed badly[113] but even then it may not be fruitless[43]. Sudden cardiac arrest is initiated by malignant ventricular arrhythmias in 80 to 90% of patients. In such cases there will be residual oxygen in the lungs and arterial system: a circulation may then help to support life for several critical minutes. Thus chest compression alone may increase the chance of survival even if ventilation is *not* performed. Animal experiments support this concept[114]. Without losing sight of the goal of optimum resuscitation[115], it should be made clear in BLS teaching that even incomplete measures can contribute to the patient's survival chances[115]. This may be of special importance in communities where there is reluctance to use mouth to mouth ventilation because of the risk of infection[116,117].

In some cases, the patient requires not only BLS and early defibrillation but also additional measures summarized under the term advanced life support (ALS). Standard guidelines on ALS have been set up and published by the European Resuscitation Council (ERC) the American Heart Association and other resuscitation councils (Fig. 4)[118]. There are four major components as follows.

Defibrillation

Defibrillation is the single most important intervention producing a successful outcome from sudden cardiac arrest. There are few studies on the optimal energies or waveform to use[119,120]. Current recommendations are for direct current (DC) shocks with a conventional damped sinusoidal waveform to be given with energies of 200 J for the first two shocks, and further ones at 360 J[118]. Emphasis on correct technique during defibrillation is crucial to maximize the chances of success[121].

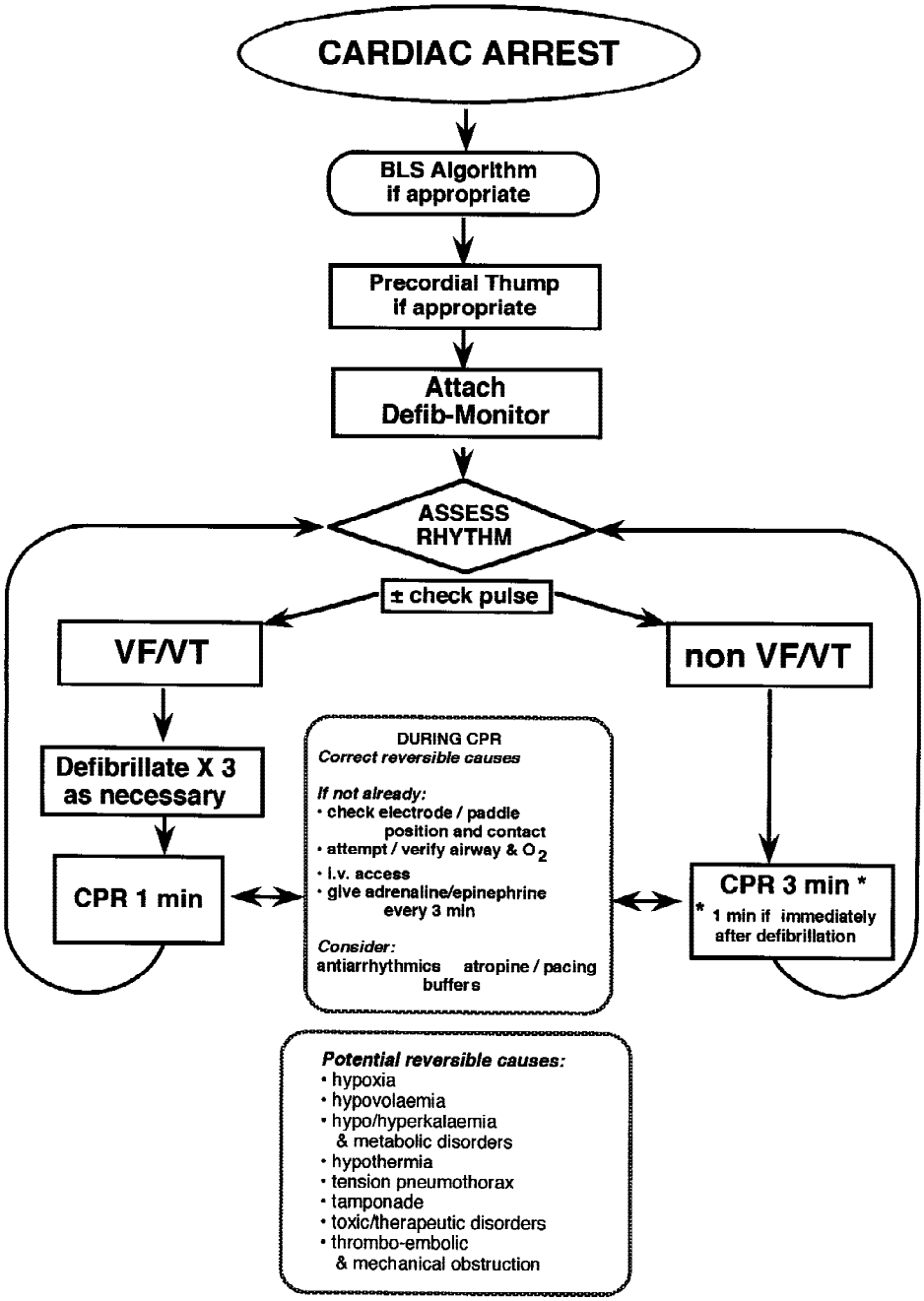

Figure 4 **The 1998 ERC algorithm for treatment of cardiac arrest[118]).**

Preliminary investigations with other waveforms such as biphasic rectangular and sinusoidal shocks have shown similar results with lower energies[122]. They offer potential advantages including reducing the myocardial injury produced by a defibrillating shock and the development of smaller, lighter, and less expensive defibrillators[123], and will be acceptable for routine use if shown to be of equal or greater efficacy and safety compared with conventional shocks. Automated external defibrillators (AEDs) enable less qualified persons in multi-tiered rescue systems to perform defibrillation[48,124] (see section on Ethics of pre-hospital resuscitation).

Airway management

The primary function of simple adjuncts in mouth-to-mouth ventilation is as a hygienic barrier. Complete protection from infection cannot be achieved with cloths and filters. Ventilation masks provide more effective protection but may require a second helper and therefore have only limited applicability. The basic ALS

adjunct is a ventilation mask connected to a self-inflating bag with an external oxygen source. It does not protect against aspiration and makes the use of positive end-expiratory pressure (PEEP) impossible. A cuffed tracheal tube is the 'gold standard' for airway protection, but requires considerable expertise and regular practice in the technique[125]. The laryngeal mask, oesophageal obturator airway, and the 'Combitube' are second-line alternatives[125].

Drug therapy and delivery

The optimal route of drug delivery in cardiac arrest is pervenous. Central venous access provides the most efficient and rapid access to the circulation, but the technique is time consuming and necessitates considerable expertise and has potentially fatal hazards in relation to puncture of a non-compressible artery (a particular problem if thrombolysis may be needed subsequently)[126]. The peripheral route is usually easier, but drug delivery to the central circulation is slow; the tracheal route is a second line choice, because of impaired absorption and unpredictable pharmacodynamics[126,127]. The drugs which can be administered by this route are limited to lidocaine, epinephrine/adrenaline, and atropine; they should be diluted in 10 ml of saline or Ringer's solution to hasten absorption.

Epinephrine/adrenaline remains the most commonly used drug in CPR. It is given in a dose of 1 mg i.v. for asystole or electromechanical dissociation (EMD), and after three DC shocks have been unsuccessful in terminating ventricular fibrillation (VF). Further doses are given at up to 3 min intervals. High dose adrenaline has not been shown to improve overall survival[128]. Recent studies suggest that other vasopressors such as vasopressin might have comparable or even more beneficial effects[129,130] but so far there is inadequate information on clinical effects and outcome.

Buffer therapy is no longer a primary component of drug therapy in resuscitation. The most widely used agent is sodium bicarbonate, and it is suggested that its use in judicious amounts (50 ml of 8·4% solution) is limited to situations of severe acidosis (arterial pH $<7{\cdot}1$ and base deficit > -10)[131]. Further doses should be administered under guidance of repeated arterial blood gas analysis.

Atropine is recommended for asystole, using a single intravenous 3 mg dose, although clear evidence of efficacy is limited. For VF persisting after 6–12 shocks, an antiarrhythmic agent may be considered: the ERC guidelines suggest an i.v. bolus of 100 mg lidocaine[118]. Patients developing VF during acute myocardial infarction may have low plasma concentrations of potassium and magnesium. It has been assumed that magnesium supplements may be useful in the treatment and prophylaxis of resistant VF, but this has not been confirmed[88]. Magnesium is, however, highly effective in treating torsades de pointes[132]. A long QT syndrome or an iatrogenic effect of antiarrhythmic drugs is often the underlying cause of this specific arrhythmic morphology. Beta blockers have been recommended in therapy-resistant malignant arrhythmias[133]. Their efficacy in the acute and convalescent phases after infarction is clear, but their effectiveness in cardiopulmonary resuscitation has not been confirmed.

The brain is the organ most sensitive to cardiac arrest and deterioration of cerebral blood flow and oxygenation. Even after restoration of circulation the processes of vascular dysfunction and inadequate regional blood flow may lead to continued damage, with leucocytes playing a prominent role in the 'no-reflow' phenomenon[134]. Calcium flux changes are also believed to be important in the process of neuronal damage: they induce a cascade of deleterious biochemical processes[135]. Neither calcium antagonists, nor any other agents, have been shown to exhibit beneficial effects on outcomes[136]. These aspects reinforce the need to optimize the basic aspects of post resuscitation care with the maintenance of normal cerebral and myocardial perfusion and oxygenation and blood chemistry.

Mechanical resuscitation measures

Within limits, higher frequencies of chest compression increase cardiac output and coronary blood flow. Disadvantages are that the rescuer becomes exhausted more rapidly and more risk of injury to the patient[137]. The recommended chest compression frequency is 100/min[111]. Other techniques such as interposed abdominal compression, active compression/decompression (ACD), a combination of both techniques (Lifestick), and vest CPR have been shown to improve various haemodynamic aspects of CPR, but no study has shown evidence for an improved eventual outcome. Among monitoring aids, end-tidal CO_2 measuring devices are the most valuable. End-tidal CO_2 measurement during CPR provides a measure of cardiac output and is useful in quantifying the efficacy of mechanical resuscitation measures and providing a prognostic role[138].

Ethics of pre-hospital resuscitation from cardiac arrest

The moral aspects of any medical intervention can be defined according to the following principles[139,140].

- The principle of beneficence — to do net good.
- The principle of non-maleficence — to do no harm.
- The principle of respect for the patient's autonomy.
- The principle of justice.

The principles of beneficence and non-maleficence can be considered together in this instance. CPR should in general be used only if it has a some chance of producing net benefit for the patient or victim. CPR is not harmless — it can be a violent, damaging, painful, alarming, and an undignified intervention. In situations where CPR is deemed to be futile, or if a patient has expressed an informed wish not to have CPR resuscitation, it should not be attempted. This information is, however, only exceptionally available outside the hospital.

The basis for do not attempt resuscitate (DNAR) orders fall into three categories[141,142]:

- CPR cannot be successful.
- The quality of life after CPR is likely to be poor.
- The patients informed and expressed wishes.

Where resuscitation is not appropriate, calls to the emergency services may not be made. But in any case, decisions should ideally take into consideration advice from relatives or friends: this may be possible even in the pre-hospital phase, because the majority of collapses occur in the patients home with relatives or carers close-by. If the patient was known to have expressed a view before his or her collapse with respect to resuscitation, this should weigh heavily on the doctor's decision although such advance directives are not always made in the light of real knowledge. In the absence of relevant information resuscitation should proceed.

Once resuscitation has been started, it should be discontinued only for well defined reasons[118]. New information may become available on the approximate duration of cardiac arrest or on underlying disease and its prognosis. In the absence of definite indications for cessation, attempt at resuscitation should continue as long as the waveform of ventricular fibrillation is present. But asystole which has lasted for 15 min or more is evidence of futility. Exceptions to this advice relate to the special situations of children, drowning, hypothermia, and drug intoxication.

Psychological aspects of pre-hospital care

The need for psychological support for a victim of a heart attack is clear. The topic, however, has been poorly assessed in guidelines and textbooks, whilst physicians and nurses who are overloaded by the need for giving clinical care tend to neglect this essential facet. Every individual needs to feel secure. This need is undermined by any illness that is perceived as posing a major threat. Although the chest pain of a coronary heart attack may not be severe, it is usually identified correctly as being of cardiac origin[143] whether or not the victim is prepared to accept this recognition to the extend of calling for aid. Once help is available the patient is moved into an unfamiliar vehicle, then into an often overcrowded and fraught emergency department, followed by a unit characterized by intimidating high technology. Many procedures follow, executed by a succession of strangers working in an atmosphere of extreme urgency. All of this poses a considerable psychological challenge and leaves little time for adequate communication[144–146]. Relatives or other bystanders also need support, particularly if they were involved in a resuscitation attempt, whether or not this was successful.

Consequences of psychological stress. The uncertainty, fear, anxiety, and stress felt by the patient are unpleasant experiences and likely to be an adverse factor in the evolution of myocardial ischaemia. Heightened sympathetic activity may induce changes in heart rate, arterial pressure, and myocardial oxygen consumption that may be equivalent to reasonably strenuous physical exertion. In addition there may be adverse changes in coronary vascular resistance particularly in atherosclerotic segments, increases in platelet aggregation, and anti-fibrinolytic factors that can all interfere with coronary flow. It is axiomatic that the management of anxiety and stress should play an important part in the treatment of acute ischaemic attacks.

Pharmacological approach. Drugs such as opioids and benzodiazepines play an important role in the relief of anxiety. Opioids themselves may suffice, but anxiety can often outlive pain: for some cases therefore, anxiolytic agents may be needed when indications for opioids are no longer present.

Psychological support. This should be provided continually using plain language that can be understood by the patient, given that their mental state may be obtunded by illness and drugs. 'There is perhaps no other situation in medicine inwhich the words of a physician bear as much potential for good or evil as in the management of myocardial infarction'[147]. Psychological support is required throughout the illness and into the convalescent phase, but this should be started at the earliest opportunity. Ideally patients should be told in advance of what is in store for them: a description of a coronary care unit can mitigate the anxiety of the patients first experience.

The rights of the patients. Patients have a right to be kept well informed. They should understand the origins of their illness and what is being done to help. The hospital mortality of acute ischaemic syndromes is now low enough to permit an optimistic appraisal, and clearly emphasis on a good prognosis is more helpful than undue discussion on risk. Whilst truth should never be compromised, the wise physician will couch it in terms that are likely to be acceptable to the patient. Optimism can also be boosted by discussions about the future, including early plans for rehabilitation and return to work.

Consent for standard therapy. Many have suggested the need to discuss with patients the risks of fibrinolytic therapy. Whilst different European countries might vary in convention and legal requirement, the point should be made that full information presented thoughtlessly may well frighten the patient and thereby increase risk: this must be a questionable procedure in terms of good medical practice and ethics. It should be sufficient to explain to a patient (who will be anxious or under the influence of sedation or both) that the treatment to be given will improve the chances of a good recovery. In this particular emergency situation full information may protect the doctor but not the patient. In this case appropriate partial disclosure should be supported universally by informed medical opinion.

Principal recommendations

Large differences exist between and also within countries with regard to access to care for victims of acute cardiac emergencies. The arrangements for the pre-hospital management of heart attack victims also varies greatly between countries. The preferred option of doctors on emergency ambulances is not a practical possibility within the foreseeable future in some European countries, but the principles of triage and arrangements for care are similar for all systems. We recommend minimum requirements for the organisation and implementation of emergency care that should exist in all European countries now ('basic') and also for more advanced strategies that should be adopted when it is practicable to do so ('optimal'). We also give a step-by-step guide to pre-hospital management of acute heart attacks.

Access to care

Public education

- *Basic*: Widespread knowledge of symptoms of acute heart attack.
- *Basic*: Access to a central emergency number and free calls to an ambulance dispatching centre.
- *Optimal*: Public media campaigns to teach symptoms of heart attacks, how to respond, and the reasons for community involvement.
- *Optimal*: Community wide knowledge of and training in Basic Life Support.
- *Optimal*: Use of the agreed common European emergency number (112) more widely implemented.

Ambulance dispatch

- *Basic*: Strategic positioning of ambulances in order to minimise ambulance delay.
- *Basic*: Trained dispatchers using priority based systems.
- *Optimal*: Dispatch controlled by physicians.
- *Optimal*: Telephone assisted CPR.

Pre-hospital resuscitation for cardiac arrest

- *Optimal*: Early defibrillation by introduction of semi-automated defibrillators activated by trained disciplined groups, following ERC guidelines.
- *Basic*: All emergency ambulances with defibrillators and ECG monitors and operators competent in their use.
- *Basic*: All emergency ambulances with the means of delivering high concentrations of oxygen.
- *Basic*: Licence for ambulance personnel to perform basic life support and defibrillation according to ERC guidelines.

Pre-hospital triage and arrangements for care for acute coronary syndromes

- *Basic*: Ambulances staffed by at least two and preferably three people qualified to carry out all appropriate recommendations.
- *Optimal*: All emergency ambulances equipped to record an ECG with staff trained in its use. Interpretation immediately available by a physician on the ambulance, by appropriately trained nurses or paramedics in countries without ambulance physicians, by a computerised ECG algorithm, or by use of telephone or radio transmission.
- *Optimal*: Pre-hospital administration of thrombolysis by physicians especially when the time saved is likely to be more than 60 min (strongly recommended).
- *Optimal*: Consideration of pre-hospital initiation of thrombolytic therapy by medically trained and certified non physician personnel if any other strategy leads to considerable delays. Any such system must be under strict medical control, use stringent inclusion and exclusion criteria, and should be subject to continuing medical audit.• *Basic*: Hospitals should be notified of the impending arrival of possible heart attack victims to facilitate immediate continuity of care.
- *Optimal*: Direct admission to CCU or appropriate dedicated area for immediate reperfusion therapy, based on advanced information from ambulance (preferred option for in-hospital treatment).
- *Basic*: Registration procedures for heart attack victims should not impede triage nor delay urgent treatment.

Early in-hospital management

- *Basic*: Triaged admission to Emergency Department if direct transfer to CCU or other dedicated area not possible.
- *Basic*: Electrocardiography must be immediately available in all Emergency Departments with operators skilled in their use.
- *Basic*: Suitability for thrombolysis to be assessed by first receiving physician without routine doctor-to-doctor referral.
- *Basic*: Thrombolysis not delayed by transfer of patients to CCU. Those not treated pre-hospital and not admitted directly to CCU should have treatment initiated in the Emergency Department using a fast-track system for those without contraindications and having immediate advice available for those with uncertain indications (if necessary with use of fax machine, telephone, or other electronic method for ECG transmission).

Pre-hospital treatment of acute coronary syndromes

The management of acute coronary syndromes in hospital is broadly agreed, but circumstances when the victim is first seen in the community demand some differences in the approach. The following steps are offered as a step-by-step guide to management in this different environment, but it is recognized that other traditions or variations in the availability of facilities or drugs must necessitate some regional modifications in practice.

(A) Management of non-complicated chest pain of presumed cardiac origin
1. Take brief relevant history.
2. Make brief assessment of vital signs (including blood pressure and heart rate).
3. Establish ECG monitoring.
4. Ensure resuscitation equipment is available or coming.
5. Give short acting nitrate if pain is still present and systolic blood pressure >90 and no bradycardia.
6. Take 12-lead ECG.
7. Give oxygen: 3 to 5 $l.min^{-1}$ via a face mask (unless this causes undue patient distress).
8. Establish i.v. access.
9. Give aspirin 150 to 300 mg orally (or i.v. if available) unless contraindicated.
10. If no pain relief obtained with a nitrate, give morphine i.v. starting with 5 mg (or equivalent dose if other opioid used) titrated up to a maximum pre-hospital dose of 20 mg for acceptable pain control.
11. Give antiemetic such as metoclopramide 20 mg i.v. if necessary.
12. If patient remains anxious despite opioid give benzodiazepine.
13. If indications are present for thrombolysis (and in the absence of contraindications or arrangements for primary angioplasty) initiate thrombolysis if appropriate in the pre-hospital phase (recommended especially if journey time may be more than 30 min or the delay or call-to-needle time for in-hospital thrombolysis may exceed 60 min).
14. If indications for thrombolysis are not present, but the ECG shows evidence of ischaemia, a bolus of heparin should be given. This will not preclude subsequent thrombolysis or primary PTCA in the hospital.

(B) Management of respiratory distress of presumed cardiac origin, in addition to any relevant measures shown above
1. Give oxygen: nasopharyngeal catheter or face mask 6 to 8 $l.min^{-1}$.
2. Rapidly increase nitrate i.v. up to 150 $\mu g . min^{-1}$ according to blood pressure tolerance. Buccal nitrate is a more convenient preparation for pre-hospital use and can provide rapid and useful nitrate concentrations.
3. Give furosemide (frusemide) 40–80 mg i.v.
4. Give morphine 5 mg i.v. (or equivalent) if not already administered. Titrate using increments of half the original dose until adequate pain relief obtained.
5. In the absence of obvious improvement consider continuous positive airway pressure (CPAP) if available.
6. If the patient's condition remains or becomes critical, immediate oral endotracheal intubation is mandatory, followed by mechanical ventilation with positive airway pressure (PAP) titrated according to blood pressure and oxygenation.
7. If an arrhythmia has contributed to the development of pulmonary oedema it should be treated if possible to do so before hospital admission.

(C) Management of left ventricular failure presenting as cardiogenic shock
1. If there is no clinical pulmonary oedema, try careful volume loading. Test with 100–200 ml of colloid.
2. Give dobutamine 4–20 $\mu g . kg^{-1} . min^{-1}$.
3. Consider vasopressor if patient remains or becomes critical.

(D) Management of symptomatic arrhythmias
For symptomatic sinus tachycardia
1. If sinus tachycardia more than 120 beats $. min^{-1}$ without overt heart failure, give metoprolol or atenolol 5 mg slowly i.v. Can be repeated up to total dose of 15 mg i.v. (three doses with 2 min intervals).
For bradyarrhythmias and tachycardia
1. Ensure pain relief is adequate.
2. Consider need for blood pressure control.
3. Follow algorithm for bradyarrhythmias, broad complex tachycardias, and narrow complex tachycardias shown in Fig. 3.

(E) Management of cardiac arrest
1. Use precordial thump for witnessed event.
2. Administer 100% oxygen and give CPR if defibrillator not available for immediate use. Defibrillator made ready.
3. Follow algorithm for VF/VT and non VF/VT rhythms shown in Fig 4.

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Appendix

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