

## Fibrillation and defibrillation of the heart

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### Fibrillation is chaos<sup>38 47 83 88 107</sup>

Fibrillation of the atrium or ventricle of the heart is a result of chaotic electrical activity of the heart chambers, resulting in loss of coordinated myocardial contraction. Fibrillation of the atrium is characterized by loss of coordinated atrial contraction and by irregular conduction of this chaotic electrical activity through the A-V conduction system to the ventricles, leading to an irregular pumping pattern of the ventricles. Fibrillation of the ventricles is characterized by loss of coordinated ventricular contraction, leading to an immediate loss of pump function of the heart and causing cessation of blood flow and oxygen supply to the vital organs of the body. Loss of oxygen supply to the brain causes brain damage and eventually brain death after only a few minutes. This represents the pathophysiological substrate of sudden cardiac death (SCD).

The mechanism of initiation of ventricular fibrillation (VF) consists of the simultaneous presence of an arrhythmogenic substrate and a trigger. The substrate is usually an area of ischaemic myocardium or an ischaemic border zone surrounding an infarcted area. The substrate becomes increasingly vulnerable (decreased fibrillation threshold) by increased concentrations of circulating catecholamines, sympathetic imbalance, metabolic abnormalities, proarrhythmogenicity of drugs, and hyper- and hypothermia. The final trigger is usually an early ventricular premature ectopic beat (VPB). Coexistence of abnormal initiation of ventricular beats (increased excitability of the substrate, latent pacemaker activity) and abnormal propagation of the impulse (bradycardia, intraventricular conduction blocks, re-entry phenomena) generate chaotic electric activity in the presence of a lowered fibrillation threshold. An increase or marked slowing of heart rate are predisposing factors in the initiation of VF. When this chaos is present in a critical mass of ventricular myocardium, fibrillation invades the whole ventricle causing VF.

In the initiation of VF, a so-called “R-on-T”

extrasystole is an important factor; late cycle ectopic ventricular tachycardia and idioventricular rhythm initiate VF less frequently.

### Aetiology and epidemiology of VF

*Primary VF* (in the absence of shock or cardiac failure) occurs in the first 5–10 min after the onset of coronary occlusion and is usually induced by an early R-on-T VPB and only rarely by degeneration of ventricular tachycardia. The success rate for resuscitation of patients in the first minute of primary VF is probably 90–95%.<sup>2 21 75</sup>

Data on Holter monitoring have demonstrated that VF waves initially have a large amplitude of >0.2 mV and a median frequency of  $\pm 200$ –300 bpm. Progressively, rate and amplitude decrease and after approximately 12–15 min, fibrillation waves fade to become a flat isoelectric line.<sup>13 53 64</sup>

*Secondary VF* occurs in the presence of shock or cardiac failure and is caused by a variety of mechanisms, including dilatation, stretching, metabolic disorders, sympathetic overactivity and proarrhythmogenicity of drugs. Secondary VF has a far lower prognosis for successful resuscitation, with less than 30% success rate.

Ischaemic heart disease (IHD) is by far the principal cause of VF. More than 30% of all patients with acute myocardial infarction die from VF within the first 2 h after onset of symptoms. The majority of patients presenting with SCD caused by VF are those at risk of IHD (i.e. middle-aged men with other signs and symptoms of IHD). However, SCD caused by VF is not infrequently the first, and lethal, symptom of IHD.

Ischaemia and reperfusion are associated with life-threatening arrhythmias, but only 20% of SCD victims have an acute myocardial infarction. Therefore, transient ischaemia and reperfusion rather than infarction have major roles in triggering the arrhythmias leading to SCD.<sup>39 91</sup>

Other causes of SCD induced by VF include diseases of the heart (reperfusion, heart failure, congenital heart disease, cardiomyopathy, valvular, infiltrative and infectious disorders), transient autonomic and neurohumeral factors, toxic and proarrhythmic effects of drugs, hypothermia, electrolyte and metabolic disorders, hypoxia, hypotension and electric shock. In young subjects

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#### Key words

Heart, resuscitation. Heart, arrhythmia, fibrillation. Equipment, defibrillators. Complications, cardiac arrest.

(age < 40 yr) cardiomyopathy and toxic substances are more important aetiologies than IHD.

Epidemiological investigation (European Monica registry) showed that the incidence of SCD caused by VF was approximately 1 per 1000 inhabitants per year. The incidence is higher in Northern countries and lower in Mediterranean countries. This lower incidence of IHD is believed to correlate with a different nutritional profile (the "French paradox").<sup>20 97</sup>

In common with other symptoms of IHD, the time of onset of VF has a circadian distribution pattern with a relative high risk during the day and the highest peak in the first 3 h after awakening. This time variation is believed to correlate with the degree of sympathetic instability and related biochemical variations.<sup>7 71 96</sup>

### VF and the chain of survival

Survival after pre-hospital cardiac arrest is more likely when activation of the emergency medical services (EMS) system, basic cardiopulmonary resuscitation (CPR), defibrillation and advanced care occur as rapidly as possible. Implementation of these various elements of CPR in the treatment of cardiac arrest can be described best by the concept of "chain of survival". This concept illustrates that failure or weakness of any one of these links is associated with poor results. The concept of chain of survival describes each link in the EMS continuum that is needed for survival in cardiac arrest.<sup>5 30 72</sup>

- The first link in the chain of survival, "early access", is essential to bring trained persons and appropriate equipment quickly to the patient, includes recognition of the collapse, decision to call, calling and dispatch, and is strengthened by public education and availability of an efficient emergency communication system (including a unique simple telephone number for medical emergencies).

- The importance of the second link, "early CPR", by the first person witnessing the arrest (lay person or bystander health care worker) has been well documented. Bystander CPR can maintain the heart in VF for approximately 10–12 min longer. Basic CPR can sustain life until arrival of trained personnel with equipment and is therefore a bridge to defibrillation.

- The most crucial link is "early defibrillation". Initially, pre-hospital defibrillation was performed only by medical and paramedical staff, but recently newer and reliable technology has allowed the defibrillator to be used by first-line technician ambulance personnel. This strategy is of greatest value as long as the preceding links of the chain of survival do not fail. In systems where access time is excessively long, the results of an early defibrillation programme may be disappointing.

- The fourth link, "early advanced life support", implies early intervention of a well-trained and equipped team, working with specially equipped ambulances or rapid intervention vehicles. These teams consist of paramedics (in the USA, UK and Scandinavia) or trained ambulancemen, doctors and/or nurses (in most European countries).

### VF and defibrillation

In the event of VF, electrical defibrillation is the only effective method of terminating the rhythm disturbance and restoring a perfusing cardiac rhythm. Landmark investigators include Zoll and colleagues,<sup>112</sup> Dahl and colleagues<sup>34</sup> and Pantridge, Adgey and Webb.<sup>86</sup> The success of electrical defibrillation is dependent on time and the metabolic state of the myocardium. The chances of successful defibrillation decrease rapidly over time, at a rate of more than 5% per minute. After a few minutes, the amplitude and frequency of the VF waveform decreases and after approximately 15 min degenerates into asystole, probably because of depletion of myocardial high energy phosphate reserves. The presence of asystole usually indicates that the time since collapse has been prolonged.<sup>16 17 22 25 60 93 102</sup>

#### MECHANISM OF DEFIBRILLATION

External defibrillation of the heart involves delivering an adequate electrical current flow through the heart, via electrodes applied to the chest wall, causing simultaneous depolarization of all myocardial cells that are at that moment fully refractory. Defibrillators consist of a power source (battery, AC source), voltage selector, AC–DC converter, capacitor and electrodes.<sup>11 15 35 46</sup> The output energy of the defibrillator is usually expressed in energy, where:

$$\text{energy (J)} = \text{power (W)} \times \text{duration (s)}$$

$$\text{power (W)} = \text{potential (V)} \times \text{current (A)}$$

$$\text{current (A)} = \frac{\text{potential (V)}}{\text{resistance or impedance } (\Omega)}$$

The output waveform of most conventional external defibrillators is half sinusoidal (damped Edmark waveform). The optimum duration of the power wave is 4–12 ms.

After defibrillation, myocardial contraction is re-established within minutes, but initially cardiac output may be extremely low, especially after prolonged VF, and continues to increase over minutes or even hours. This phenomenon has been described as the post-countershock pulseless rhythm or post-countershock myocardial depression.

Inspired by the work of Russian investigators and by the experience of implanted automatic defibrillators, the use of biphasic or triphasic waveforms has been advocated. Indeed, post-countershock dysfunction of the myocardium appears to depend on shock waveform. The defibrillation threshold seems to be lower using biphasic waveforms and causes less myocardial dysfunction with implantable defibrillators. Others have suggested that the biphasic waveform may reduce the defibrillation threshold by prolonging refractoriness which may protect ventricular cells from re-fibrillation wavefronts. The clinical relevance of biphasic waveform defibrillation at different energy levels is being investigated.<sup>12 49 109</sup>

## TRANSTHORACIC IMPEDANCE: THRESHOLD AND ENERGY REQUIREMENTS

Not all cases of VF can be defibrillated by an external shock. The minimum shock strength capable of abolishing fibrillation is defined as the defibrillation threshold. The defibrillation threshold is influenced by anatomical variations in heart size, ventricular filling and lung volumes, and electrical variables such as defibrillation waveform and metabolic state, and biochemical and physiological differences in temperature, tissue pH and  $PO_2$ , extracellular potassium concentration, ischaemia and drugs. The energy requirement for defibrillation increases with the duration of fibrillation. The sum of these variables can be responsible for a five-fold difference in threshold as time elapses.<sup>37 59 77</sup>

In VF/tachycardia, defibrillation is accomplished by passage of sufficient electrical current through the heart to depolarize a critical mass of myocardium. The amount of current flowing through the heart depends on the energy of the shock and transthoracic impedance. During transthoracic defibrillation in humans, as little as 4% of the delivered transthoracic current traverses the heart because parallel pathways (thoracic cage, lungs) divert the current away from the heart.<sup>33 74</sup>

Transthoracic impedance depends on many factors: time to defibrillation; electrode size, contact, pressure, and distance from the heart; previous shocks; and ventilatory phase. The success of defibrillation depends not only on threshold but also on probability.

Defibrillation at too high energy levels can be proarrhythmic and cause functional and morphological damage to the myocardium. Shocks below the defibrillation threshold are ineffective and harmful because they may re-initiate VF by stimulating parts of the myocardium during a vulnerable period.<sup>82 95 108</sup>

Prospective clinical investigations have demonstrated that the success of defibrillation and subsequent hospital discharge rates were almost identical in patients receiving an initial shock of 175 or 320 J. Therefore, the energy selected for the first shock is a compromise between probability of success and risk of damage.<sup>58 66 101</sup> Based on this scientific evidence, the following guideline has been accepted widely for defibrillating an adult presenting with VF: first shock—200 J; second shock—200–300 J; subsequent shocks  $\leq$ 360 J.

In children, a weight-related initial energy of  $2 \text{ J kg}^{-1}$  is recommended.<sup>50</sup>

The average human transthoracic impedance is 70–80  $\Omega$ , but can occasionally be as high as 150  $\Omega$  and as low as 15  $\Omega$ . The energy for the first shock can be predicted and adjusted based on automatic measurement of impedance, and inappropriate low energies can be avoided in patients with high impedances.<sup>67</sup> This facility is already available in modern equipment.

## INFLUENCE OF DRUGS ON FIBRILLATION AND DEFIBRILLATION THRESHOLDS

Intervention studies have demonstrated that some routinely used drugs are proarrhythmic, serving

Table 1 Pharmacological profiles of antiarrhythmic drugs

Drug	Fibrillation threshold	Defibrillation threshold	Proarrhythmogenicity
Quinidine	++	+++	+
Procainamide	++	0	+
Encainide	++	+++	+
Flecainide	++	+++	+
Lignocaine	++	+	+
Bretylium	++	–	+
Amiodarone	++	+	0/+
Verapamil	+	++	0
Diltiazem	+	++	0
Nifedipine	+	0	0
Adrenergic agents	–	0	+
Beta-blockers	++	+	0
Sotalol	+	–	+

only to aggravate the situation.<sup>8 41 45 51 52 54 61 65 68 70 73 81 85 89 90 98 100</sup>

The pharmacological profiles of bretylium, amiodarone and beta-blocking drugs are probably superior to other commonly used antiarrhythmics in the treatment of VF refractory to countershock (table 1). Bretylium and lignocaine increase the fibrillation threshold significantly. However, lignocaine also increases the defibrillation threshold whereas bretylium does not. Lignocaine in association with acidosis produces a further increase in the energy required; respiratory alkalosis causes a reduction in energy required to defibrillate successfully. Administration of lignocaine during myocardial reperfusion in CABG allows defibrillation with fewer DC shocks of lower energy and current. Bretylium lowers the defibrillation threshold in dogs and facilitates conversion of hypothermia-induced VF. However, deleterious effects of bretylium on haemodynamic recovery from VF have occasionally been reported.

The effect of other antiarrhythmic drugs is less clear (table 1): procainamide has no effect on defibrillation energy requirements. Flecainide significantly increases the defibrillation threshold and has adverse proarrhythmic and haemodynamic effects in dogs. Propafenone reduces energy requirements for defibrillation in pigs.

The calcium channel blocking drugs, diltiazem and verapamil, increase the defibrillation threshold, whereas nifedipine and nisoldipine do not.

Adrenaline has no effect and propranolol increases the defibrillation threshold. Beta stimulation decreased the defibrillation threshold significantly in dogs, an effect that was blocked by propranolol. Sotalol (DL and D) decreased defibrillation energy requirements.

Excessive administration of magnesium may increase the defibrillation threshold.

## PRACTICAL CONSIDERATIONS

*Paddle size, shape and position*

Electrode pad size is an important determinant of transthoracic current flow during external countershock. Larger self-adhesive electrocardiogram/defibrillator pads are associated with a lower transthoracic impedance and improved defibrillation

success rates with low energy shocks. The optimum electrode size is  $\pm 13$  cm in diameter in adults, 8–10 cm in children and 4.5–5 cm in infants.<sup>10,36</sup> There has been no attempt to standardize the physical characteristics of adhesive pads for automated external defibrillators.

Paddle position influences current flow through the myocardium. One electrode should be placed below the outer half of the right clavicle and the other just outside the usual position of the cardiac apex (V4–5 position). Anterior–posterior (one paddle over the precordium and the other on the back just behind the heart) electrode placement may be recommended in patients with implanted defibrillators or pacemakers. The influence of polarity on the defibrillation threshold is negligible.<sup>103</sup>

Disposable coupling pads are as effective as electrode paste.<sup>11</sup> Jelly is probably less satisfactory as it tends to be spread across the chest wall during resuscitative efforts: the shock then arcs across the chest wall, failing to reach the heart in adequate strength.

Before performing external defibrillation, all nitro patches and ointments should be removed from the chest wall.<sup>110</sup>

#### *External defibrillation and pacemakers*

Pacemaker problems reported during external defibrillation include transient malfunctions of pacing, capture and sensing. Although modern pacemakers are less likely to be disturbed by external defibrillation, an increase in pacemaker threshold has been described. With implanted pacemakers, anterior–posterior electrode or paddle placement maintaining a distance of 12–15 cm from the pacemaker module is recommended. Pacing functions should be checked immediately in patients who have been defibrillated.<sup>69,80,113</sup>

#### *Technical problems*

In a review of more than 2100 technical reports related to defibrillator problems, some failures were attributable to component malfunctions, but errors in operator use and defibrillator care and maintenance accounted for the majority of defibrillator failures. Where defibrillators are used relatively frequently, such as in ambulances, emergency departments, intensive and coronary care units, operating theatres and catheterization laboratories, it is recommended that the operational state of the equipment is assessed every day using appropriate checklists.<sup>29</sup> Inadequate training and re-training increase the chances of operator errors at the critical moment.

### **Resuscitation algorithm for VF**

Based on the available scientific knowledge, a universal algorithm for the advanced life support (ALS) management of VF patients has been proposed by the International Liaison Committee on Resuscitation (ILCOR) (fig. 1). This committee includes representatives from the European Resuscitation Council, American Heart Association,

Canadian Heart and Stroke Foundation, Australian Resuscitation Council and Southern African Resuscitation Council.<sup>1,3,4,44,94</sup>

After confirmation of cardiac arrest, a precordial thump should be given immediately and the defibrillator pads applied. The diagnosis of VF should be followed immediately by three electric shocks of 200, 200–360 J, without interposed CPR. After the initial three shocks, the airway should be secured and i.v. access achieved, if these manoeuvres have not already been performed. At this stage adrenaline 1 mg i.v. should be given (2.5 mg if given by tracheal tube), and 10 sequences of five chest compressions: one ventilation should be given.

The next shocks should be given as quickly as possible and the interval between the third and next (fourth) shock should not exceed 2 min: the more rapid a coordinated rhythm is restored, the better the prospects of long-term success. In patients whose trachea is intubated, the next shock should be given immediately after the 10 CPR cycles have been completed and adrenaline was given, and tracheal intubation should be aborted temporarily if unsuccessful after approximately 30 s.

The fourth shock forms the beginning of a new sequence of three shocks. Additional doses of adrenaline 1 mg may be given at less than 2-min intervals (fig. 1).

### **Automated external defibrillator (AED)**

The most frequent initial rhythm is SCD is VF. The most effective treatment for VF is electrical defibrillation; the probability of successful defibrillation diminishes rapidly over time and VF tends to convert to asystole within a few minutes. The rationale of AED programmes is to reduce the time to first defibrillation.

Originally, defibrillation could only be performed by emergency care providers who were trained in ALS. Today it is often performed by lesser trained BLS personnel. AED eliminates the need for training in rhythm recognition and makes early defibrillation by untrained personnel practical and achievable.

The first automated implantable defibrillators were developed by Mirowski in 1970. In the past 40 yr, external defibrillation has become the most important tool for termination of lethal arrhythmias. The first successful defibrillation of ventricular fibrillation outside hospital was achieved by the Belfast Mobile Coronary Care Unit in 1966 and the first automated defibrillators appeared in Brighton in the late 1970s.<sup>56,57</sup>

#### SOME TECHNICAL NOTES ABOUT AED

Automated defibrillators are now available for use primarily outside hospital. The terms automated and shock-advisory defibrillators refer to external defibrillators that incorporate a rhythm analysis system. AED are attached to the patient by two adhesive pads with connecting cables to analyse the rhythm and deliver the shock. The information is given by voice or a visual display, or both, and the

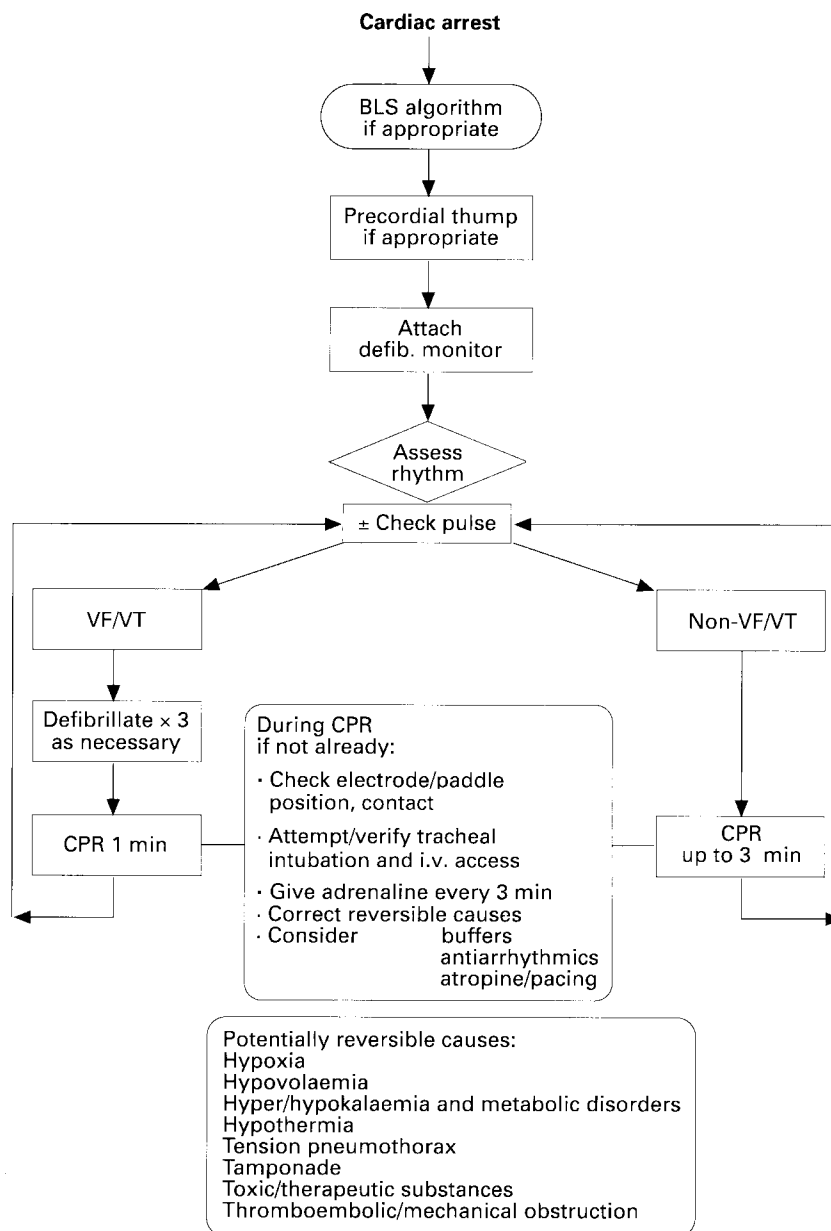


Figure 1 Advanced life support (ALS) algorithm for the management of cardiac arrest in adults. Note that each successive step is based on the assumption that the one before has been unsuccessful.<sup>56</sup>

actual delivery of the shock is triggered manually.

These systems have great potential for use by basic ambulance men who may not be trained in ECG interpretation. Although specificity for VF is approximately 100%, sensitivity for coarse VF is approximately 90–92%, for fine VF even lower and for VT only 50%.<sup>23 32 87</sup>

Moreover, in patients with an implanted automatic defibrillator, failure of the diagnostic algorithm has been documented when the patient also has an implanted pacemaker.<sup>80</sup>

The present equipment is relatively expensive, and performance is dependent on good maintenance.<sup>4</sup> A new generation of AED are now appearing on the market using new battery technology and some also have new waveform technology. They are small, simple, reliable and less expensive. Some of the new devices have the facility for ECG display. These new generation AED will stimulate widespread

availability of defibrillators in ambulances, doctor's cars, places of work, public places, aeroplanes and cruise ships, etc.

#### EARLY DEFIBRILLATION BY FIRST-RESPONDING AMBULANCE PERSONNEL

Electrical defibrillation is the only effective therapy for VF. The chances of successful defibrillation decrease rapidly over time by more than 5% per minute. It has been demonstrated in Belgium and Sweden that bystander CPR can maintain the heart in VF longer, for approximately 10–15 min, and is therefore a bridge to first defibrillation.

In two-tiered EMS systems, the first ambulance generally arrives several minutes before the second ambulance, which usually has the defibrillator and medically qualified personnel on board. The Belgian CPR registry showed that 69% of cardiac arrest

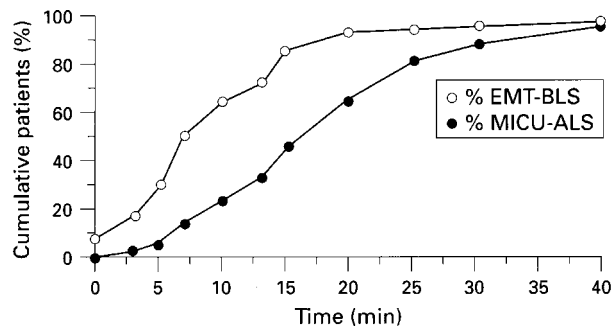


Figure 2 Cumulative inclusion of cardiac arrest patients by the first-tier ambulance (EMT-BLS) and by the second-tier ambulance (MICU-ALS), illustrating that the first-tier arrives significantly faster than the second tier.<sup>19</sup>

patients were first attended by the basic ambulance manned by two EMT; the median time interval between collapse and start of BLS resuscitation by the EMT was 8 min; the median time interval between collapse and defibrillation by the second-tier medical ALS team was 16 min. It was concluded that EMT can be expected to reach a substantial number of VF victims within a few minutes after collapse and many minutes before arrival of the medical ALS team (fig. 2).<sup>19</sup>

Therefore, it seemed logical to equip first-tiered ambulances with defibrillators and to train first-line ambulancemen in the use of AED. The expected result is that EMS systems, based on early defibrillation by first-line ambulancemen, will provide not only shorter times to initial countershock but also, by having delegated initial defibrillation to first responders, they will produce significantly shorter times to i.v. access, tracheal intubation and initial adrenergic drug therapy.<sup>55</sup>

#### AED training

Experiences in Scandinavia, the UK, Germany, Belgium and the USA have demonstrated that a training course of less than 8–10 h followed by 6-monthly refresher courses is sufficient to instruct an ambulanceman in the correct use of an AED according to a standard procedure. Knowledge of basic CPR is a prerequisite for successfully attending an AED course.

The AED training course and subsequent implementation of an AED programme, should be placed under full medical supervision. The course should include background information, knowledge of the equipment and its maintenance, and knowledge of the procedure, and should be tested by an examination. Formal 6-monthly refresher courses are needed, with feedback to discuss each intervention.

To achieve the goal of early defibrillation, ideally all emergency personnel responding to cardiac arrest should be trained, equipped and permitted to defibrillate.

Following the early pre-hospital experience in Belfast and Brighton, defibrillator technology has been developed further and systems are now implemented in many countries in a community wide basis. Early defibrillation with AED by ambulance personnel is now available widely in the

UK and Scandinavia, and in parts of Germany and Belgium. Pilot studies are emerging in many European countries: France, Spain, Italy, Austria, Switzerland. In the Netherlands and Spain, all ambulances are manned by experienced nurses or doctors, or both, and are equipped with manual defibrillators.

As a result of these experiences it has been recognized that the success of an early defibrillation programme depends on the following factors:

- the programme should be placed under medical control
- the majority of arrests should be witnessed
- the time interval between cardiac arrest and first CPR should be <4 min
- the time interval between cardiac arrest and defibrillation should be <12 min
- there should be a minimum number of interventions
- there should be a programme of training and retraining
- there should be a programme for monitoring the performance of the programme.

#### SLOW IMPLEMENTATION OF AED BY FIRST-RESPONDING AMBULANCE PERSONNEL IN EUROPE

AED programmes are only partially implemented in continental Europe. Important reasons for this slow implementation include: lack of motivation and awareness; organization of EMS systems; and legal issues. Moreover, in the USA, widespread implementation of AED programmes has also been delayed by regulatory authorities.<sup>31</sup>

#### Awareness

In Europe, cardiovascular disease accounts for more than 45% of mortality. However, public awareness is most influenced by information provided by the media. In a recent survey in a sample of the adult Belgian population, only 8% of respondents answered that cardiovascular disease was their major concern, whereas the values were 18% for traffic accidents and 17% for AIDS.<sup>9</sup>

#### Organization

In all European countries access to the EMS system is by a specific telephone number. The European Union has recommended the use of 112 as the uniform European emergency telephone number.<sup>14 24 42 106</sup>

There is a wide variety of European EMS systems: one-tiered systems delivering BLS by an EMT (basic ambulance person); one-tiered systems delivering BLS and defibrillation by an EMT-d; one-tiered systems delivering BLS and ALS by paramedics, doctors and/or nurses; two-tiered systems delivering BLS followed by ALS; and two-tiered systems delivering BLS and defibrillation, followed by ALS (table 2). Best performance, in terms of survival, is obtained with systems with highly qualified staff provided they are available quickly.

The structure and organization of the EMS

**Table 2** EMS systems in European countries. Local variations in the country are indicated between brackets. md = medical doctor; rn = nurse; pm = paramedic; EMT = emergency medical technician; EMT-d = emergency medical technician qualified for use of AED. (\*) In the presence of a doctor. For permission to defibrillate, the minimum training level is mentioned

County	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	md, rn, EMT-d
Croatia	md	—	94	md
Czechia	EMT	md	155	md, pm
Denmark	EMT-(d)	(pm)	112	md, rn, EMT-d
Finland	EMT-(d)	md	112	Everybody trained
France	EMT	md	15	md, rn, EMT-d
Germany	EMT-(d)	md	112	md, pm, EMT-d
Greece	EMT	—	166	md
Hungary	EMT-(d)	rn/md	04	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, EMT-d
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/m	—	061	md, rn no law
Sweden	EMT-d	pm	90 000	md, rn, EMT-d
Switzerland	EMT	(md)	114	md, rn, pm
Turkey	md	—	118	md
UK	EMT-(d)	(pm)	999	No law
Yugoslavia	md	md	94	md, rn, emt

systems in European countries, and the legal background for defibrillation practice are summarized in table 2

In the majority of continental European countries, doctors have an active role in pre-hospital emergency medical care, as part of the first or second tier. In most parts of Scandinavia and the UK, paramedics serve as members of the first or second tier. This medical presence on the field could be a reason for slow involvement of ambulance personnel in defibrillation.

### Legislation

Because of historical, organizational, political and religious reasons, legislation relating to resuscitation and defibrillation is different in European countries. In countries where historically only ambulancemen and paramedics were present in the field, implementation of early defibrillation by ambulancemen was easy. However, in countries where a medical presence was prominent in the second or even in the first tier, introduction of defibrillation by first-line ambulancemen was much slower.

Information on legal regulations has been collected from 28 European countries:

**Providing help.** In all European countries (except the UK, where there is no legislation), anyone is legally obliged to provide help to endangered persons.

**Providing CPR.** In 20 of 28 countries, anyone (or anyone who has been instructed) is legally allowed

(or not forbidden) to start CPR. In five countries there is no specific legislation and in three countries CPR is restricted to health care professionals. In seven of 28 countries anyone who has been instructed, in 12 of 28 any health care professional (whether or not in an official capacity), and in four of 28 countries only doctors are legally obliged to start CPR. In five countries there is no specific legislation.

**Providing defibrillation.** In most European countries, defibrillation was considered until recently to be solely a medical intervention, which reflects the historical involvement of doctors in out-of-hospital emergencies and major incidents. In four of 28 countries the law restricts the act of defibrillation exclusively to doctors, and in five countries only nurses can be delegated to defibrillate. In six countries, ambulance personnel are allowed to defibrillate only in the presence of a doctor. In 11 of 28 countries defibrillation can legally be performed by nurses, paramedics or qualified health care professionals. In the UK and Spain there is no legal restriction on providing defibrillation. This means that in at least 10 European countries the law is an obstacle for nationwide implementation of AED programmes by non-physicians. In countries where nurses may be allowed to defibrillate, there is a potential problem for ambulances that are manned by EMT.

The cost of hardware is decreasing for new generation devices. Training and re-training are very demanding and require substantial effort and logistic solutions. Other major obstacles are public and political awareness, tradition and structure of the EMS systems, and motivation of the medical community. Increasing public and political awareness and increasing motivation in the medical profession can help to overcome these obstacles.

### RESULTS OF AED PROGRAMMES OF EARLY DEFIBRILLATION BY FIRST-LINE AMBULANCE PERSONNEL

Reported data on survival after cardiac arrest caused by VF by non-medically qualified personnel using an AED, range from 4 to 31%.<sup>6 26 43 48 63 78 79 92 99</sup> These variations may be related to: differences in characteristics of the treated population; differences in methodology and quality of registration; and differences in the performance of the AED programme.

One of the key elements that determines success of an AED programme is the percentage of cardiac arrest patients found in VF, which is a reflection of the speed of intervention of the EMS system. Reported incidences of VF in different cities and countries around the world range from 62% to less than 10%. As expected, there is a significant correlation between the incidence of VF and subsequent survival. As a consequence, it is unclear if the reported differences in survival rates are a result of differences in system organization, treatment procedures or in the skills of the personnel.

The "Utstein style" presents a glossary of terms and reporting guidelines for description of cardiac arrest, resuscitation, the EMS system and outcome,

and was published by representatives of the American Heart Association, the European Resuscitation Council, the Heart and Stroke Foundation of Canada, the Southern African Resuscitation Council and the Australian Resuscitation Council. The recommendations were published simultaneously in *Circulation*, *Resuscitation*, *Annals of Emergency Medicine* and other major journals. The in-hospital "Utstein style" has been published in 1997.<sup>18 27 28</sup>

#### IMMEDIATE DEFIBRILLATION BY FIRST-LINE TRAINED INDIVIDUALS

As considerable time may elapse between the onset of VF and arrival of the emergency medical services, immediate defibrillation by first-line trained bystanders may be the long-term realization of the concept of early defibrillation. A first responder is defined as a trained individual, acting independently within a medically controlled system.

Although immediate first-responder defibrillation is the logical theoretical next step after implementation of defibrillation by ambulance personnel, there is at present no evidence that bystander defibrillation increase survival rates. There have been occasional cases of defibrillation in-flight and in a railway station.

Programmes should be developed to test the principle that first responders assigned to the management of cardiac arrest in the community should be trained and permitted to defibrillate. This could include police, security officers, life guards, voluntary aiders and those assigned to provide first aid at their place of work. Every programme designed to implement first-line defibrillation should be evaluated critically to assess if first-line defibrillation increases survival.<sup>40 62 76 84 104 105</sup>

#### Conclusions

Cardiorespiratory arrest caused by sudden arrhythmia as a consequence of myocardial ischaemia or infarction continues to be a leading cause of unexpected death in the community. Increased public education in the recognition of early symptoms and signs of a heart attack is likely to achieve a significant reduction in the present mortality and morbidity of out-of-hospital heart attack.

Electrical defibrillation is the single most important therapy for the treatment of VF. The time interval between onset of VF and delivery of the first defibrillation shock is the main determinant of survival. This should be as short as possible. Early defibrillation has a crucial role in the chain of survival. To achieve the goal of early defibrillation, it is mandatory to allow individuals other than doctors to defibrillate. At present, however, early defibrillation by ambulance personnel using automated external defibrillation (AED) is not yet widely implemented.

Obstacles to implementation of AED by ambulance personnel are: legislation, structure of EMS system, economic priorities, awareness,

motivation and tradition. It is also recognized that success of an early defibrillation programme is dependent on the following factors: the programme is placed under medical control; the majority of arrests are witnessed; the time interval between cardiac arrest and first CPR is usually <4 min; the time interval between cardiac arrest and defibrillation is usually <12 min; there is a minimum number of interventions; there is a programme of training and re-training; and there is a programme for monitoring the performance of the programme.

#### Recommendations

- The medical profession is urged to increase awareness of the public and of those responsible for emergency medical services and regulatory powers to permit changes in practice and legislation where necessary.
- It is essential to integrate the concept of early defibrillation into an effective emergency cardiac care system. This is best characterized by the "chain of survival" concept, which includes early access to the emergency medical services (EMS) system, early cardiopulmonary resuscitation when needed, early defibrillation when indicated and early advanced cardiac care.
- All *emergency personnel* should be trained and permitted to operate an appropriately maintained defibrillator if their professional activities require that they respond to persons experiencing cardiac arrest. This includes all first-line emergency personnel working in an organized EMS system, both inside and outside hospital (e.g. emergency medical technicians (EMT), non-EMT first responders, fire fighters, volunteer emergency personnel, physicians, nurses and paramedics).
- All emergency ambulances and other emergency vehicles that respond to or transport cardiac patients should be equipped with a defibrillator.
- Defibrillation should be a core competence of professional nurses, and defibrillators should be available widely on general hospital wards.
- All defibrillator programmes must operate within medical controls by qualified and experienced physicians. They should ensure that every link of the chain of survival is in place and should have access to all information required to permit system audit.
- To monitor the programme, there must be appropriate registration of the interventions according to the Utstein style.

#### References

1. ACC/AHA guidelines for the management of patients with acute myocardial infarction. A report of the ACC/AHA Task Force on Practice Guidelines (Committee on Management of Acute Myocardial Infarction). *Journal of the American College of Cardiology* 1996; **28**: 1328-1428.
2. Adgey A. Initiation of ventricular fibrillation outside the hospital. In: Adgey A, ed. *Acute Phase of Ischemic Heart Disease and Myocardial Infarction*. Nyhoff: Martinus, 1982; 67-76.



3. Advanced Cardiac Life Support Committee of the European Resuscitation Council. Management of periarrest arrhythmias. A statement for the Advanced Cardiac Life Support Committee of the European Resuscitation Council. *Resuscitation* 1994; **28**: 151–159 and *Resuscitation* 1996; **32**: 281.
4. Advanced Life Support Working Group of the ERC. Guidelines for advanced life support. *Resuscitation* 1992; **24**: 111–132.
5. Ahnefeld FW. Die Wiederbelebung bei Kreislaufstillstand. *Verhandlungen Deutsche Gesellschaft für Innere Medizin* 1968; **74**: 279–287.
6. Arntz HR, Oeff M, Willich SN, Storch WH, Schroder R. Establishment and results of an EMT-D program in a two-tiered physician-escorted rescue system. The experience in Berlin, Germany. *Resuscitation* 1993; **26**: 39–46.
7. Arntz HR, Willich SN, Stern R, Linderer T, Bruggemann T, Kelinski K, Schroder R. Circadian variation of cardiopulmonary disease onset in the general population: an emergency care system perspective from Berlin. *Annals of Emergency Medicine* 1994; **23**: 281–285.
8. Arredondo MT, Guillen SG, Quinteiro RA. Effect of amiodarone on ventricular fibrillation and defibrillation thresholds in the canine heart under normal and ischaemic conditions. *European Journal of Pharmacology* 1986; **125**: 23–28.
9. Anonymous. Ziekten waar Belgen bang voor zijn. *Artsenkrant* April; 1996: 1–3.
10. Atkins D, Sirna S, Kieso R, Charbonnier F, Kerber R. Pediatric defibrillation: importance of paddle size in determining transthoracic impedance. *Pediatrics* 1988; **82**: 914–918.
11. Aylward PE, Kieso R, Hite P, Charbonnier F, Kerber RE. Defibrillator electrode-chest wall coupling agents: influence on transthoracic impedance and shock success. *Journal of the American College of Cardiology* 1985; **6**: 682–686.
12. Bardy GH, Gliner BE, Kudenchuk PJ, Poole JE, Dolack GL, Jones GK, Anderson J, Troutman C, Johnson G. Truncated biphasic pulses for transthoracic defibrillation. *Circulation* 1995; **91**: 1768–1774.
13. Bayes de Luna A, Coumel P, Leclercq JF. Ambulatory sudden death: mechanism of production of fatal arrhythmias on the basis of data from 157 cases. *American Heart Journal* 1989; **117**: 151–159.
14. Bossaert L. The complexity of comparing different EMS-systems: A survey of EMS-systems in Europe. *Annals of Emergency Medicine* 1993; **22**: 99–102.
15. Bossaert L, Koster R. Defibrillation: methods and strategies. *Resuscitation* 1992; **24**: 211–226.
16. Brown CG, Dzwonczyk R, Werman HE, Hamlin RL. Estimation of the duration of ventricular fibrillation. *Annals of Emergency Medicine* 1989; **18**: 1181–1185.
17. Brown CG, Griffith RF, Van Ligten P, Hoekstra J, Nejman G, Mitchel L, Dzwonczyk R. Median frequency—a new parameter for predicting defibrillation success rate. *Annals of Emergency Medicine* 1991; **20**: 787–789.
18. Callahan M, Madsen CD. Relationship of timeliness of paramedic advanced life support interventions to outcome in out-of-hospital cardiac arrest treated by first responders with defibrillators. *Annals of Emergency Medicine* 1996; **27**: 638–648.
19. Calle P, Van Acker P, Buylaert W, Quets A, Corne L, Delooy H, Bossaert L, Martens P, Mullie A. Should semi-automatic defibrillators be used by emergency medical technicians in Belgium? *Acta Clinica Belgica* 1992; **47**: 6–14.
20. Cambou JP, Arveiler D, Amouyel P, Ruidavets JB, Haas B, Montayé M, Bingham A, Richard JL. Coronary disease in France: data from the MONICA registers (1985–1991). *Revue d'Epidemiologie et de Sante Publique* 1996; **44**: S46–S52.
21. Campbell R, Murray A, Julian D. Ventricular arrhythmias in the first 12 of acute myocardial infarction. Natural history study. *British Heart Journal* 1981; **46**: 351–357.
22. Carlisle E, Allen JD, Kernohan WG, Anderson J, Adgey AAJ. Fourier analysis of ventricular fibrillation of varied aetiology. *European Heart Journal* 1990; **11**: 173–181.
23. Cobbe S, Redmond M, Watson J, Hollingworth J, Carrington D. "Heartstart Scotland"—initial experience of a national scheme for out of hospital defibrillation. *British Medical Journal* 1991; **302**: 1517–1520.
24. Conseil de l'Europe. Etude comparative sur l'organisation et le fonctionnement des services d'aide médicale urgente. *Comparative Study of the Organisation and Functioning of Emergency Medical Assistance Services*. Strasbourg: Conseil de l'Europe, 1990.
25. Cox-SV, Woodhouse SP, Weber M, Boyd-P, Case C. Rhythm changes during resuscitation from ventricular fibrillation. *Resuscitation* 1993; **26**: 53–61.
26. Cummins R. From concept to standard-of-care? Review of the clinical experience with automated external defibrillators. *Annals of Emergency Medicine* 1989; **18**: 1269–1276.
27. Cummins R, Chamberlain D, Abramson N, Allen M, Baskett P, Becker L, Bossaert L, Delooy H, Dick W, Eisenberg M, Evans T, Holmberg S, Kerber R, Mullie A, Ornato J, Sandoe E, Skulberg A, Tunstall-Pedoe H, Swanson R, Thies W. Special report: Recommended guidelines for uniform reporting of data from out-of-hospital cardiac arrest. *Circulation* 1991; **84**: 960–975.
28. Cummins R, Chamberlain D, Hazinski MF, Nadkarni V, Kloeck W, Kramer E, Becker L, Robertson C, Koster R, Zaritsky A, Bossaert L, Ornato J, Callanan V, Allen M, Steen P, Conolly B, Sanders A, Idris A, Cobbe S. Recommended guidelines for reviewing, reporting and conducting research on in-hospital resuscitation, the "in-hospital Utstein style". *Resuscitation* 1997; **34**: 151–185 and *Circulation* 1997; **95**: 2213–2239.
29. Cummins R, Chesmore K, White R. Defibrillator failures, causes of problems and recommendations for improvement. *Journal of the American Medical Association* 1990; **264**: 1019–1025.
30. Cummins R, Ornato J, Thies W, Pepe PE. Improving survival from sudden cardiac arrest: the "chain of survival" concept. *Circulation* 1991; **83**: 1832–1847.
31. Cummins RO, Dean R, Pepe PE. Ventricular fibrillation, automatic external defibrillators, and the United States Food and Drug Administration: Confrontation without comprehension. *Annals of Emergency Medicine* 1995; **26**: 621–631.
32. Cummins RO, Stults KR, Haggart B, Kerber RE, Schaeffer S, Brown DD. A new rhythm library for testing automatic external defibrillators: performance of three devices. *Journal of the American College of Cardiology* 1988; **3**: 597–602.
33. Dahl C, Ewy G, Thomas E. Transthoracic impedance to direct current discharge: effect of repeated countershocks. *Medical Instruments* 1976; **10**: 151–154.
34. Dahl C, Ewy G, Warner E, Thomas ED. Myocardial necrosis from direct current countershock. *Circulation* 1974; **50**: 956–961.
35. Dalzell G, Adgey A. Determinants of successful transthoracic defibrillation and outcome in ventricular fibrillation. *British Heart Journal* 1991; **65**: 311.
36. Dalzell G, Cunningham S, Anderson J, Adgey A. Electrode pad size, transthoracic impedance and success of external ventricular defibrillation. *American Journal of Cardiology* 1989; **64**: 741–744.
37. Dalzell GW, Adgey AA. Determinants of successful transthoracic defibrillation and outcome in ventricular fibrillation. *British Heart Journal* 1991; **65**: 311–316.
38. Davies MJ. Anatomic features in victims of sudden coronary death: Coronary artery pathology. *Circulation* 1992; **85**: I-19–I-24.
39. d'Avila A, Wellens F, Andries E, Brugada P. At what time are implantable defibrillator shocks delivered? Evidence for individual circadian variance in sudden cardiac death. *European Heart Journal* 1995; **16**: 1231–1233.
40. Destro A, Marzaloni M, Sermasi S, Rossi F. Automatic external defibrillators in the hospital as well? *Resuscitation* 1996; **31**: 39–43.
41. Echt DS, Black JN, Barbey JT, Cox DR, Cato E. Evaluation of antiarrhythmic drugs on defibrillation energy requirements in dogs. *Circulation* 1989; **79**: 1106–1117.
42. Eisenberg M, Horwood B, Cummins R, Reynolds-Haertle R, Hearne T. Cardiac arrest and resuscitation: a tale of 29 cities. *Annals of Emergency Medicine* 1990; **19**: 238–243.
43. Ekstrom L, Herlitz J, Wennerblom B, Axelsson A, Bang A, Holmberg S. Survival after cardiac arrest outside hospital over a 12-year period in Gothenburg. *Resuscitation* 1994; **27**: 181–187.

44. Emergency Cardiac Care Committee, American Heart Association. Guidelines for CPR and emergency cardiac care. *Journal of the American Medical Association* 1992; **268**: 2171–2302.
45. Euler DE, Zeman TW, Wallock ME, Scanlon PJ. Deleterious effects of bretylium on hemodynamic recovery from ventricular fibrillation. *American Heart Journal* 1986; **112**: 25–31.
46. Ewy G. Electrical therapy for cardiovascular emergencies. *Circulation* 1986; **74** (Suppl. IV): 111–116.
47. Farb A, Burke AP, Tang AL, Liang TY, Mannan P, Smialek J, Virmani R. Coronary plaque erosion without rupture into a lipid core. *Circulation* 1996; **93**: 1354–1363.
48. Gallehr JE, Vukov LF. Defining the benefits of rural emergency medical technician-defibrillation. *Annals of Emergency Medicine* 1993; **22**: 108–112.
49. Gurvich NL, Makarychev VA. Defibrillation of the heart with biphasic electrical impulses. *Kardiologiya* 1967; **7**: 109–112.
50. Gutgesell H, Tacker W, Geddes L, Davis J, Lie J, McNamara D. Energy dose for ventricular defibrillation of children. *Pediatrics* 1976; **58**: 898–901.
51. Hecker BR, Lake CL, Kron IL, Mentzer RM, Crosby IK, Nolan SP, Crampton RS. Influence of magnesium ion on human ventricular defibrillation after aortocoronary bypass surgery. *American Journal of Cardiology* 1985; **55**: 61–64.
52. Hernandez R, Mann DE, Breckinridge S, Williams GR, Reiter MJ. Effects of flecainide on defibrillation thresholds in the anesthetized dog. *Journal of the American College of Cardiology* 1989; **14**: 777–781.
53. Hinkle L. The immediate antecedents of sudden death. *Acta Medica Scandinavica* 1981; **210** (Suppl. 651): 207–217.
54. Hite PR, Schroder E, Kieso RA, Fox EK, Kerber RE. Effect of calcium channel blockers on hemodynamic responses to defibrillation. *American Heart Journal* 1989; **117**: 569–576.
55. Hoekstra JW, Banks JR, Martin DR, Cummins RO, Pepe PE, Stueven HA, Jastremski M, Gonzalez E, Brown CG. Effect of first-responder automated defibrillation on time to therapeutic interventions during out-of-hospital cardiac arrest. *Annals of Emergency Medicine* 1993; **22**: 1311–1290.
56. Chamberlain D, Cummins R. Advisory statements of the International liaison committee on resuscitation. *Circulation* 1997; **95**: 2172–2173 and *Resuscitation* 1997; **34**: 99–100.
57. Jaggerao N, Heber M, Grainger R, Vincent R, Chamberlain D. Use of an automated external defibrillator—pacemaker by ambulance staff. *Lancet* 1982; **ii**: 73–75.
58. Jakobsson J, Rehnqvist N, Nyquist O. Energy requirement for early defibrillation. *European Heart Journal* 1989; **10**: 551–554.
59. Jones DL, Irish WD, Klein GJ. Defibrillation efficacy. Comparison of defibrillation threshold versus dose–response curve determination. *Circulation Research* 1991; **69**: 45–51.
60. Jones DL, Klein GJ. Ventricular fibrillation: the importance of being coarse? *Journal of Electrocardiology* 1984; **17**: 393–399.
61. Jung W, Manz M, Pfeiffer D, Tebbenjohanns J, Pizzulli L, Luderitz B. Effects of antiarrhythmic drugs on epicardial defibrillation energy requirements and the rate of defibrillator discharges. *Pacing and Clinical Electrophysiology* 1993; **16**: 198–201.
62. Kaye W, Mancini ME, Richards N. Organizing and implementing a hospital-wide first-responder automated external defibrillation program: strengthening the in-hospital chain of survival. *Resuscitation* 1995; **30**: 151–156.
63. Kellermann AL, Hackman BB, Somes G, Kreth TK, Nail L, Dobyns P. Impact of first-responder defibrillation in an urban emergency medical services system. *Journal of the American Medical Association* 1993; **270**: 1708–1713.
64. Kemp FF, Josephson M. Cardiac arrest recorded on ambulatory electrocardiograms. *American Journal of Cardiology* 1984; **53**: 1577–1582.
65. Kerber R, Pandian N, Jensen S, Constantin L, Kieso R, Melton J, Hunt M. Effect of lidocaine and bretylium on energy requirements for transthoracic defibrillation: experimental studies. *Journal of the American College of Cardiology* 1986; **7**: 397–405.
66. Kerber RE. Energy requirements for defibrillation. *Circulation* 1986; **74** (Suppl. IV): 117–119.
67. Kerber RE, Martins JB, Kienzle MG, Constantin L, Olshansky B, Hopson R, Charbonnier F. Energy, current, and success in defibrillation and cardioversion: clinical studies using an automated impedance-based method of energy adjustment. *Circulation* 1988; **77**: 1038–1046.
68. Kieso RA, Fox EK, Kerber RE. Effect of nisoldipine on hemodynamic responses to defibrillation. *American Heart Journal* 1991; **121**: 834–839.
69. Kim S, Furman S, Waspe L, Brodman R, Fisher JD. Unipolar pacer artifacts induced failure of an automatic implantable cardioverter/defibrillator to detect ventricular fibrillation. *Pacing and Clinical Electrophysiology* 1991; **14**: 7–12.
70. Koo CC, Allen JD, Pantridge JF. Lack of effect of bretylium tosylate on electrical ventricular defibrillation in a controlled study. *Cardiovascular Research* 1984; **18**: 762–767.
71. Krantz DS, Kop WJ, Gabbay FH, Rozanski A, Barnard M, Klein J, Pardo Y, Gottdiener JS. Circadian variation of ambulatory myocardial ischemia. Triggering by daily activities and evidence for an endogenous circadian component. *Circulation* 1996; **93**: 1364–1371.
72. Kuschinsky B, Schmiedel R, Unterkofler M. Erste Hilfe in der Bundesrepublik Deutschland. Untersuchungen zum Rettungswesen. Bericht 18. Bonn: Deutsches Rotes Kreuz, 1986.
73. Lake CL, Kron IL, Mentzer RM, Crampton RS. Lidocaine enhances intraoperative ventricular defibrillation. *Anesthesia and Analgesia* 1986; **65**: 337–340.
74. Lerman BB, Deale OC. Relation between transcardiac and transthoracic current during defibrillation in humans. *Circulation Research* 1990; **67**: 1420–1426.
75. Lown B, Wolf M. Approaches to sudden death from coronary heart disease. *Circulation* 1971; **44**: 130–142.
76. Macdonald JW, Brewster MF, Isles CG. Defibrillation by general practitioners: an audit of resuscitation in a Scottish rural practice. *Scottish Medical Journal* 1993; **38**: 79–80.
77. Martin-D, Garcia J, Valeri CR, Khuri-SF. The effects of normothermic and hypothermic cardiopulmonary bypass on defibrillation energy requirements and transmural impedance. Implications for implantable cardioverter-defibrillator implantation. *Journal of Thoracic and Cardiovascular Surgery* 1995; **109**: 981–988.
78. Mauer D, Schneider T, Diehl P, Dick W, Brehmer F, Juchems R, Kettler D, Kleine-Zander R, Klingler H, Rossi R, Roth H, Schüttler J, Stratmann D, Strohmeier U, Zander J. Erstdefibrillation durch Notarzte oder durch Rettungshelfer? Eine prospektive, vergleichende Multicenterstudie bei ausserklinisch aufgetretenem Kammerflimmern. *Anaesthesist* 1994; **43**: 36–49.
79. Mols P, Beaucarne E, Bruyninx J, Labruyere JP, De-Myttenaere L, Naeije N, Watteuw G, Verset D, Flamand JP. Early defibrillation by EMTs: the Brussels experience. *Resuscitation* 1994; **27**: 129–136.
80. Monsieurs KG, Conraads VM, Goethals MP, Snoeck JP, Bossaert LL. Semi-automatic external defibrillation and implanted cardiac pacemakers: understanding the interactions during resuscitation. *Resuscitation* 1995; **30**: 127–131.
81. Montenero AS, Bombardieri G, Barilaro C, Di Francesco P, Santarelli P, Calvi V, Schiavello R, Alessandrini F, Pisano E, Salsano M. Intravenous propafenone reduces energy requirements for defibrillation in pigs. *Cardiologia* 1990; **35**: 291–294.
82. Morgan JP, Hearne SF, Raizes GS, White RD, Giuliani ER. High-energy versus low-energy defibrillation: experience in patients (excluding those in the intensive care unit) at Mayo Clinic-affiliated hospitals. *Mayo Clinic Proceedings* 1984; **59**: 829–834.
83. Myerburg RJ, Kessler KM, Castellanos A. Sudden cardiac death: Structure, function, and time dependence of risk. *Circulation* 1992; **85**: I-2–I-10.
84. O'Rourke MF, Donaldson E. An airline cardiac arrest programme. *Circulation* 1994; **90**: 1548 (Suppl.).
85. Otto CW, Yakaitis RW, Ewy GA. Effect of epinephrine on defibrillation in ischemic ventricular fibrillation. *American Journal of Emergency Medicine* 1985; **3**: 285–291.
86. Pantridge J, Adgey A, Webb S. Electrical requirements for ventricular defibrillation. *British Medical Journal* 1975; **2**: 313–315.

87. Pantridge J, Geddes J. A mobile intensive care unit in the management of myocardial infarction. *Lancet* 1967; **ii**: 271–273.
88. Pantridge J, Geddes J. Cardiac arrest after myocardial infarction. *Lancet* 1966; **i**: 807–808.
89. Ruffey R, Schechtman K, Monje E. Beta-adrenergic modulation of direct defibrillation energy in anesthetized dog heart. *American Journal of Physiology* 1985; **248**: 674–677.
90. Ruffey R, Schechtman K, Monje E, Sandza J. Adrenergically mediated variations in the energy required to defibrillate the heart: observations in closed-chest, nonanesthetized dogs. *Circulation* 1986; **73**: 374–380.
91. Sedgwick ML, Dalziel K, Watson J, Carrington DJ, Cobbe SM. The causative rhythm in out-of-hospital cardiac arrests witnessed by the emergency medical services in the Heartstart Scotland Project. *Resuscitation* 1994; **27**: 55–59.
92. Sedgwick ML, Dalziel K, Watson J, Carrington DJ, Cobbe SM. Performance of an established system of first responder out-of-hospital defibrillation. The results of the second year of the Heartstart Scotland Project in the 'Utstein Style'. *Resuscitation* 1993; **26**: 75–88.
93. Strohmeier HU, Lindner KH, Lurie KG, Welz A, Georgieff M. Frequency of ventricular fibrillation as a predictor of defibrillation success during cardiac surgery. *Anesthesia and Analgesia* 1994; **79**: 434–438.
94. Task Force on the Management of Acute Myocardial Infarction of the European Society of Cardiology. Acute myocardial infarction: pre-hospital and in-hospital management. *European Heart Journal* 1996; **17**: 43–63.
95. Trouton TG, Allen JD, Yong LK, Rooney JJ, Adgey AA. Metabolic changes and mitochondrial dysfunction early following transthoracic countershock in dogs. *Pacing and Clinical Electrophysiology* 1989; **12**: 1827–1834.
96. Tsivoni D. Circadian variation of total ischaemic burden and ischaemic threshold. *European Heart Journal* 1996; **17** (Suppl. G): 59–63.
97. Tunstall-Pedoe H, Morrison C, Woodward M, Fitzpatrick B, Watt G. Sex differences in myocardial infarction and coronary deaths in the Scottish MONICA population of Glasgow 1985 to 1991. Presentation, diagnosis, treatment, and 28-day case fatality of 3991 events in men and 1551 events in women. *Circulation*. 1996; **93**: 1981–1992.
98. Ujhelyi MR, Schur M, Frede T, Gabel M, Markel ML. Differential effects of lidocaine on defibrillation threshold with monophasic versus biphasic shock waveforms. *Circulation* 1995; **92**: 1644–1650.
99. Walters G, Glucksman E, Evans TR. Training St John Ambulance volunteers to use an automated external defibrillator. *Resuscitation* 1994; **27**: 39–45.
100. Wang M, Dorian P. DL and D sotalol decrease defibrillation energy requirements. *Pacing and Clinical Electrophysiology* 1989; **12**: 1522–1529.
101. Weaver W, Cobb L, Copass M, Hallstrom A. Ventricular defibrillation: a comparative trial using 175-j and 320-j shocks. *New England Journal of Medicine* 1982; **307**: 1101–1106.
102. Weaver WD, Cobb LA, Dennis D, Ray R, Hallstrom AP, Copass MK. Amplitude of ventricular fibrillation waveform and outcome after cardiac arrest. *Annals of Internal Medicine* 1985; **102**: 53–55.
103. Weaver WD, Martin JS, Wirkus MJ, Morud S, Vincent S, Litwin PE, Morgan C. Influence of external defibrillator electrode polarity on cardiac resuscitation. *Pacing and Clinical Electrophysiology* 1993; **16**: 285–290.
104. Weisfeldt ML, Kerber RE, Mc Goldrick RP, Moss AJ, Nichol G, Ornato JP, Palmer DG, Riegel B, Smith SC Jr. Public access defibrillation: A statement for healthcare professionals from the American Heart Association Task Force on Automatic External Defibrillation. *Circulation* 1995; **92**: 2763.
105. White R, Vukov L, Bugliosi T. Early defibrillation by police: initial experience with measurement of critical time intervals and patient outcome. *Annals of Emergency Medicine* 1996; **28**: 480–485.
106. WHO Regional Office for Europe. Monitoring of the strategy for health for all by the year 2000. Eur/RC38/11. Copenhagen: WHO, 1989.
107. Willich SN, Maclure M, Mittleman M, Arntz HR, Muller JE. Sudden cardiac death. Support for a role of triggering in causation. *Circulation* 1993; **87**: 1442–1450.
108. Wilson CM, Allen JD, Bridges JB, Adgey AA. Death and damage caused by multiple direct current shocks: studies in an animal model. *European Heart Journal* 1988; **9**: 1257–1265.
109. Wilson CM, Bailey A, Allen JD, Anderson J, Adgey AA. Transthoracic defibrillation threshold of sine and trapezoidal waveforms in defibrillation. *Journal of Electrocardiology* 1989; **22**: 241–247.
110. Wrenn K. The hazards of defibrillation through nitroglycerin patches. *Annals of Emergency Medicine* 1990; **19**: 1327–1328.
111. Zipes D, Fisher P, King R, Nicoll A, Jolly W. Termination of ventricular fibrillation in dogs by depolarising a critical amount of myocardium. *American Journal of Cardiology* 1975; **36**: 37–44.
112. Zoll P, Linenthal A, Gibson W, Paul M, Norman L. Termination of ventricular fibrillation in man by externally applied electric countershock. *New England Journal of Medicine* 1956; **254**: 727.
113. Zullo MA. Function of ventricular pacemakers during resuscitation. *Pacing and Clinical Electrophysiology* 1990; **13**: 736–744.