Management of Cardiac Arrest in 2005: An Update

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Abstract

Although 40 years have passed since the advent of advanced cardiac life support, out-of-hospital cardiac arrest still carries an ultimate failure rate of 95%. This review reinforces the importance of public education, optimization of the local chain of survival, early bystander access and bystander basic life support, and early defibrillation. It emphasizes the role of simplified basic life support algorithms and demonstrates the low incremental benefit of complex skillful protocols employed in ACLS. The impact of automatic external defibrillators and new medications incorporated into ACLS algorithms is evaluated in the light of contemporary research. The persistent, discouraging, low functional survival rate (less than 5% of out-of-hospital cardiac arrest victims) mandates reassessment of current strategies and guidelines.

IMAJ 2005;7:589–594

Cardiac arrest or sudden cardiac death [1] has an annual incidence of 0.1% (range 0.03–0.015%) in the general population, while the annual incidence of out-of-hospital cardiac arrest approaches 1% [2] in the seventh decade of life. In the United States 300,000 people suffer OHCA every year. It is estimated that a similar number of events occur annually in the USA during hospitalization. The most common etiology of OHCA is coronary artery disease (the precipitating mechanism is poorly understood and is related to ischemic or infarcted myocardium, with superimposed metabolic, electrolyte, autonomic or drug imbalance). Other predisposing conditions are left ventricular hypertrophy, left or right heart failure, diabetes mellitus, precipitating arrhythmias, and other causes of respiratory or circulatory insufficiency.

The rhythms most frequently encountered in OHCA are pulseless ventricular tachycardia and ventricular fibrillation (30–50%), asystole (30–51%), and pulseless electric activity (10–25%). VF degenerates within minutes to asystole, consequently the longer the delay the higher the incidence of asystole. The traditional survival rates to hospital discharge of these three rhythms are 5–34%, <1%, and 1–4% respectively. A recent Central European multicenter study reports hospital admission rates of 44.6%, 24.6% and 32.1% respectively [3]. The same study, however, reported hospital discharge rates of 18.5%, 2.6%

and 7.2% respectively. For the entire cohort, return of spontaneous circulation occurred in 26.3% of patients, hospital admission rate was 33.7% but hospital discharge 9.9% and only half of these patients had acceptable cerebral function. Another contemporary study, the OPALS (Ontario Prehospital Advanced Life Support Study Group) [4] (17 best-performing communities in Ontario, Canada), had even worse results in 4,247 victims of sudden cardiac death: 18% had return of spontaneous circulation, 14.6% were admitted, and 5.1% were discharged from the hospital, of whom 66.8% had good cerebral performance. Sadly, the percent of victims discharged alive and functional is the only acceptable descriptor of a favorable outcome of resuscitation. This figure ranges between 3% and 5%. Unfortunately, only 25% of those admitted to hospital are discharged alive.

Chain of survival

The International Liaison Committee on Resuscitation (ILCOR) symposium in June 2001 (at Utstein Abbey, Norway) [5] suggested that in order to optimize resuscitation results we need to engage in intense education efforts and to further improve the local "chain of survival." The working group emphasized that educational efforts should initially be directed at providing basic life support skills to schoolchildren and to the adult population above 40 years old. They also recommended that BLS education be given to laypersons whose occupational duty is to respond (police, firemen, security guards, aviation crew) and to healthcare providers. The local chain of survival currently has five key components (links): a) early prevention, b) early access to emergency medical services, c) early cardiopulmonary resuscitation, d) early defibrillation, and e) early advanced care.

Early prevention

Early prevention of SCD is a complex issue. At the 2001 meeting of the European Society of Cardiology, Priori et al. [6] presented their guidelines on sudden cardiac death, and an update article by the same group in 2003 [7] stressed the importance of primary and secondary prevention for patients with coronary artery disease and dilated non-ischemic cardiomyopathy. Howev-

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ACLS = advanced cardiac life support
OHCA = out-of-hospital cardiac arrest
VF = ventricular fibrillation

BLS = basic life support
SCD = sudden cardiac death

er, any preventive efforts have to tackle two major hurdles. Firstly, SCD is unpredictable: >80% of SCD victims are at low risk, and for 30–50% of these patients SCD will be the first manifestation of coronary artery disease. Since most SCD victims belong to a broad-based population at low risk, and risk is related to age, high cost preventive measures are inappropriate. Secondly, 95% of those with out-of-hospital SCD will never leave the hospital and hence will never become candidates for secondary prevention (very high risk). Since implementation of risk stratification and prophylactic measures were considered not feasible, the recent European Society of Cardiology update [7] emphasized medical therapy for both primary and secondary prevention of SCD [Table 1]. Risk assessment of individuals with various cardiac disorders is beyond the scope of this manuscript. Implantable cardioverter-defibrillator therapy for secondary prevention in the presence of coronary artery disease and heart failure is well established. However, the role of this device for primary prevention in arrhythmia-free heart failure patients remains somewhat elusive and controversial, and, if accepted, will impose a tremendous medical and economic burden. Since most SCD victims are at very low risk, it is unlikely that implantable defibrillators will make a significant impact on the epidemiology of SCD. Using heart rate variability, QRS duration, functional class, left ventricular ejection fraction, T-wave alternans, and gender may further discriminate between those heart failure patients who would benefit from primary prevention with an implantable cardioverter defibrillator and those who would not.

The ESC guidelines stress that some medications are clearly associated with increased incidence of death or SCD, while others reduce that risk [Table 2]. Other aspects of prevention are related to public education and include healthy lifestyle, prevention of atherosclerosis and cardiovascular disease, and recognizing and responding to pre-arrest conditions.

Early access to EMS (witnessed SCD) and early bystander CPR

Out-of-hospital cardiac arrest occurs most frequently at home (56–80% of cases) and less commonly in the public domain (15–21%) where the outcome is somewhat more favorable [8]. Nursing home outcome is extremely poor (2–8%). Those who suffer SCD at home are older, more often women, less frequently found in ventricular fibrillation, and are less often witnessed or exposed to bystander or emergency medical service cardiopulmonary resuscitation and early defibrillation. In one study of 24,630 SCD victims, the 30 day survival of home SCD and non-home SCD was 1.7% and 6.2% respectively (P = 0.0001) [9]. According to a U.S. report [10], the survival until hospital discharge of nursing home SCD stands at 0%.

Fewer than 10% of cardiac arrests are witnessed by the emergency medical services, whereas 40% [2] to 78.2% [3] are witnessed by bystanders. Bystander basic life support was attempted in <10% of cases [4] in the OPALS study. Bystander CPR was attempted in 14–46% of the cases reported from other areas of Canada, and 18.4% of cases in a recent Central Europe study

ESC = European Society of Cardiology

Table 1. Modified ESC Guidelines on SCD for primary prevention

	Class I	Class IIa	Class IIb
Post-myocardial infarction	Beta-blocker, ACEI, aspirin, Statins	PUFA Amiodarone	
Myocardial infarction & LVEF⇓	β-blocker, ACEI, Aldosterone blocker	Amiodarone ICD (EF<30%)	
Hemodynamic tolerated VT		Amiodarone β-blocker	ICD Ablation surgery
EF<40% & spontaneous VT (ns/s) inducible at programmed stimulation	ICD		
Dilated cardiomyopathy Primary prevention	ACEI β-blocker	Aldosterone blocker ICD (EF<36%)	
Secondary prevention, dilated & ischemic cardiomyopathy	ACEI, β-blocker ICD	Aldosterone blocker	Amiodarone

Table 2. SCD prevention by drugs after MI (modified from ESC guidelines)

Drug	Relative risk Death/SCD	Class recommendation/ Level of evidence	P
Amiodarone	0.87 / **0.71**	IIA/A	0.03 / **0.0003**
D-sotalol	1.65 / **1.77**	III/B	<0.006 / **0.008**
Dofetolide	0.95	III/A	NS
I-a drugs	1.19	III/B	0.07
I-c drugs	1.31 / **3.6**	III/B	0.1 / **0.0006**
Calcium channel blockers	1.04	III/B	<0.41
Beta-blocker	0.83 / 0.80	I/A	<0.001 / **< 0.001**

[1]. In both the OPALS trial [4] and the Canadian report, it was noted that the most important predictors of survival to discharge of out-of-hospital SCD victims are: cardiac arrest witnessed by a bystander (early access; odds ratio 4.4, 95% confidence interval 3.1–6.4), and early bystander CPR (odds ratio 3.7, 95% confidence interval 2.5–5.7). These data [Figure 1] are consistent with previous reports from the same region [11]. Swedish CPR data for the past two decades demonstrate survival of 2%, 13% and 22% for unwitnessed, bystander-witnessed and emergency medical services-witnessed SCD, respectively [12]. Ventricular fibrillation was observed in 65% of witnessed SCD [13].

Early access and early bystander CPR are the weakest and the most modifiable and influential links in the chain of survival. Regrettably, the dramatic improvement in mobile communications features and availability, as well as advances in trans-telephone monitoring and emergency or alert devices have not produced an inexpensive, widely applicable technology, that is both sensitive and specific, to detect and automatically alert the emergency medical services regarding an impending or occurring SCD.
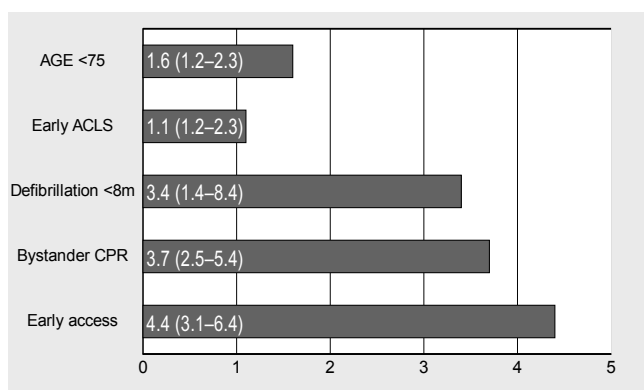
CPR = cardiopulmonary resuscitation

Figure 1. Factors affecting survival to hospital discharge (odds ratio and 95% CI) after sudden death in the OPALS trial

Early defibrillation

For the last two decades this parameter has been known to affect the outcome of SCD. Defibrillation in the initial 3 minutes of VF or pulseless VT carries a survival rate of over 50% [14]. Time to defibrillation is clearly related to survival. (Survival with defibrillation at 9 minutes is 4.6%, 8 minutes 5.9%, 7 minutes 7.5%, 6 minutes 9.5%, and 5 minutes 12.0% [15]). In the OPALS study, defibrillation prior to 8 minutes was associated with an odds ratio of 3.4 (95% CI 1.4–8.4) for survival.

Shock-resistant VF remains a challenge. Some believe that therapy for this condition requires CPR for 90–180 seconds using high voltage biphasic defibrillators [16], and conventional drugs like epinephrine (class IIA recommendation), vasopressin, amiodarone and lidocaine (class IIB recommendation).

Early advanced care

This link was investigated in the recent OPALS study [4]. The purpose of the study was to test the incremental benefit for survival of out-of-hospital cardiac arrest victims, by adding advanced life support to the program of rapid defibrillation and basic life support in the emergency medical services of 17 communities in Ontario. The addition of endotracheal intubation (attempted 90.6%, succeeded in 93.7%), intravenous line (88.7% attempted, 89% successful), use of intravenous medications – epinephrine (95.6%), atropine (87.3%), lidocaine (23.6%) – and fluid bolus (42.4%), yielded a superior rate of ROSC (18% vs. 12.9%, $P < 0.001$) and higher hospital admission rate (14.6% vs. 10.9%, $P < 0.001$). However, survival to hospital discharge (5.1% vs. 5%, $P = 0.83$) and cerebral performance did not improve. The authors concluded that the addition of advanced life support interventions did not improve survival of OHCA. They suggested that healthcare planners make CPR by laypeople and rapid defibrillation the educational and training priorities of the EMS.

How can we execute early defibrillation?

Results of several studies have led to the unequivocal (class I, ESC guidelines) [5] recommendation that the automatic external defibrillator be used in the public domain by EMS personnel [17], police [18], commercial aircraft crew [19], and security guards [14]. Hence, OHCA standard of care dictates early defibrillation by the first responding professional rescuer. In the 2000 international guidelines for AED use by EMS providers, it was recommended that all ambulances responding to SCD carry an AED or defibrillator, with personnel trained and permitted to use these devices. It has been suggested that defibrillation be instituted as a core competency of doctors, nurses, and other healthcare professionals, and that defibrillators be placed on general hospital wards. However, the concept of AED at home remains unproven. In a recent U.S. registry of over 14,720 patients undergoing adult CPR, only 1.4% were defibrillated by AED [20]. Since 80% of SCD occur at home the population does not seem to benefit dramatically from AED installation in the public domain. Moreover, in a recent study by the Public Access Defibrillator Trial Investigators [21], community volunteer units trained in CPR were compared with units equipped with CPR and AED skills and equipment. Benefit for the patients treated by the latter group (survival 14% vs. 23.4%, $P = 0.03$) was demonstrated but this benefit was limited only to the public units. In the residential units the survival rate remained <3.3% and was not affected by AED training and feasibility. Stolz et al. [22] reported a worse outcome (survival to hospital discharge dropping from 23.7% to 14.1%, $P = 0.112$) after introduction of early paramedic defibrillation (performed 5.7 ± 2.4 minutes after SCD), but these findings are not in agreement with other reports [17–19].

Should we always defibrillate first?

Wik and co-workers [23] investigated whether defibrillations should always be executed first or does the patient need chest compressions for 3 minutes prior to defibrillation to enhance the outcome of defibrillation. The study randomized 200 patients with out-of-hospital VF to standard care (immediate defibrillation) versus chest compressions and CPR for 3 minutes prior to defibrillation attempts [Figure 2]. ROSC was similar in both groups, however for patients with ambulance response time >5 minutes (pre-defined subgroup) ROSC was 38% in the defibrillation first arm versus 58% in the CPR first cohort ($P = 0.04$). This translated into a survival benefit at 1 year of 4% versus 20% ($P = 0.01$). These data are supported by another human study, conducted by Cobb and team [24], where 90 seconds CPR prior to defibrillation resulted in a higher survival rate when EMS response time exceeded 4 minutes. In a separate report, Eftestol and associates [25] assessed the effects of CPR on five VF parameters as predictors of ROSC. The favorable effects of CPR on VF were noted after CPR sequences of >3 minutes duration. The "chest compressions first" concept

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CI = confidence interval
ROSC = return of spontaneous circulation

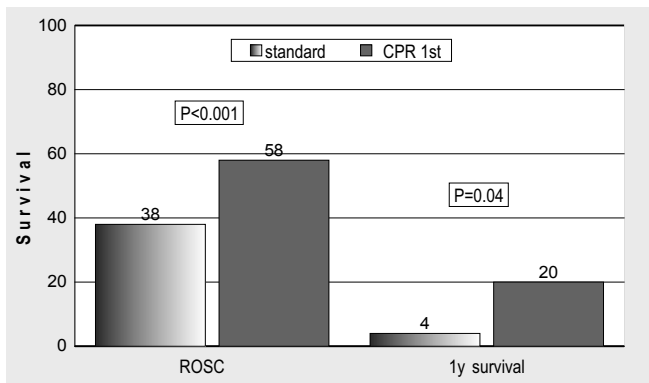AED = automatic external defibrillator

Figure 2. Standard protocol (defibrillation first) versus 3 minutes CPR first in resuscitated VF > 5 minutes duration (per ambulance response time)

is supported by a "VF swine model" reported by Berg et al. [26]. Thirty pigs were in untreated VF for 8 minutes and were assigned to either a standard protocol of defibrillation first (n=15), or chest compressions for 90 seconds and then standard protocol. In the defibrillations-first arm no pig had ROSC and 10 pigs degenerated into pulseless electric activity during the initial defibrillation attempts. In the CPR-first group 13 pigs (87%) had ROSC with the initial defibrillation attempts. It was noted that mean VF frequency of >12 was a predictor of successful defibrillation and ROSC. Mean VF frequency dramatically improved after 90 seconds CPR from 8.8 to 15.1 Hz. Menegazzi et al. [27] found (in the VF swine model) that if VF of moderate duration had a scaling exponent >1.3, immediate defibrillation was ineffective and even delayed ROSC.

It is concluded that prolonged VF (which can be characterized by electrocardiograph parameters) exceeding 5–8 minutes mandates 90–180 seconds of CPR prior to defibrillation attempts. Moreover, after a failed set of defibrillations, CPR time should be extended to 3 minutes (and not 1 minute as dictated by ILCOR protocols [28]) prior to additional defibrillation attempts. Interruption of chest compressions results in adverse hemodynamic effects [29]. Moreover, interruptions of <20 seconds duration prior to defibrillation result in a dramatic reduction of successful defibrillation and ROSC [30].

Simplified BLS

The ILCOR basic life support guidelines (2001) omitted the requirement for removal of a foreign body using blind finger sweep by lay rescuers under the assumption that choking occurs in <1% of adults with SCD. Research [31] suggests a ratio of 15:2 of chest compressions to ventilations, and that for one and two rescuers CPR is optimal and results in better oxygenation [32] than chest compressions alone [33]. However, in a randomized clinical trial (n=520) comparing conventional CPR to chest compression alone, the former showed no advantage (survival to hospital discharge) over the latter [34]. Moreover, in conventional or traditional CPR, half of the time is dedicated to breathing, with an average of 16 seconds interference time for 2 breaths of a single rescuer. This interruption results in hemody-

namic drawbacks and yields a lower rate of successful defibrillation and ROSC. "Chest compressions-only" BLS is far preferable to doing nothing and is actually as good as chest compressions combined with ventilation. However, if ventilation can be performed without interference of chest compressions it will yield much better arterial and tissue oxygenation.

Simplicity is essential for any method that is taught widely but rarely applied by professionals and laypersons alike. Since none of the more elaborate methods of circulatory support (plunger, mechanical piston or vest CPR, interposed abdominal compressions and direct cardiac massage – all ILCOR class IIb recommendations) results in better clinical outcome, it is probably best to adhere to rapid (>100/min), intense (>4 cm deep), uninterrupted chest compressions as the mainstay of CPR.

Medications for ACLS

The 2001 ILCOR recommendations (class IIb) mentioned an intravenous bolus of vasopressin 40 units as an alternative to adrenalin for shock-refractory VF after a study by Lindner's group [35] demonstrated vasopressin's superiority to adrenaline in the treatment of out-of hospital VF. In another study however, Stiell et al. [4] were not able to show any survival advantage of vasopressin over adrenalin at 1 hour and upon hospital discharge. To further complicate the picture, a meta-analysis [36] of animal experiments did reflect vasopressin superiority over both adrenalin and placebo. In 2004 Wenzel and co-workers [3] reported a multicenter, randomized trial comparing vasopressin (589 patients who received a repeated i.v. bolus of 40 units 3 minutes apart) to epinephrine (597 patients received repeated 1 mg i.v. bolus 3 minutes apart). Patients who failed ROSC after the study medications (373 in the vasopressin arm and 359 in the epinephrine arm) were allowed to receive additional epinephrine. Vasopressin did not offer any advantage over epinephrine in patients with VF or pulseless electric activity. However, in the asystole cohort (n=528, accounting for 45% of the study population), vasopressin was superior to adrenalin as stand-alone drug (first analysis): 29 versus 20.3 patients were admitted to hospital ($P = 0.02$), and 4.7% versus 1.5% ($P = 0.04$) were discharged. Half of the patients discharged in both arms were neurologically impaired. For the "initial non-responders" who required additional epinephrine in addition to the study medication (second analysis) there was again superiority of vasopressin over adrenalin in the asystole cohort: 22.5% vs. 13.3% hospital admission rate ($P = 0.02$), and 3.8% vs. 0% discharge rate ($P = 0.008$). The authors concluded that the superiority of vasopressin over epinephrine was limited to patients with asystole or patients with refractory cardiac arrest.

The role of amiodarone is less well defined. The study by Kudenchuck and collaborators [37] showed a benefit in survival to hospital admission in patients receiving amiodarone as compared to placebo (odds ratio 1.6, 95%CI 1.1–2.4, $P = 0.02$). This advantage was most pronounced in those who had ROSC prior to amiodarone administration and received amiodarone not later than 16 minutes from dispatch. Amiodarone therapy did not offer any advantage in survival to hospital discharge,

and more bradycardia and hypotension were noted with amiodarone therapy. Another study, by Dorian et al. [38], comparing amiodarone to lidocaine showed increased survival to hospital admission (22.8% vs. 12%, $P = 0.009$), but no significant difference in hospital discharge (5% vs. 3%). Hence, amiodarone like lidocaine remains an acceptable treatment (class IIb recommendation) for shock-refractory VF.

Conclusion

The occurrence of SCD remains mostly unpredictable. The most effective measures for dealing with this catastrophic event are public education and improving the local chain of survival. Modifiable survival-enhancing elements of the chain of survival are probably early access to EMS by bystanders, early bystander CPR, and early defibrillation. Chest compressions and defibrillation are the two key elements of basic life support and should be executed in a skillful and timely manner. If the patient has been in VF for more than 5 minutes it is probably preferable to perform CPR for 90–180 seconds prior to defibrillation attempts. Early advanced life support and medications play a minor role in the outcome of CPR. However, in patients with asystole, vasopressin may be superior to epinephrine.

Sadly, over the last decade even upscale EMS systems were able to only modestly improve overall survival to hospital discharge from 3.5% [7] to 5% [2]. This is related to the fact that OHCA is usually unpredictable, occurs mostly at home, is frequently unwitnessed, and, if witnessed, rarely receives adequate bystander CPR. Since only 2–5% of SCD victims (25% of those admitted) leave the hospital, it is crucial that we substantiate our knowledge regarding anoxic encephalopathy and post-SCD brain resuscitation. Methods of early detection, quantification, and prognosis of anoxic encephalopathy are required in order to selectively engage in hypothermic therapy and to develop brain salvage algorithms [39].

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