RESUSCITATION: AN OVERVIEW OF THE RECOMMENDED GUIDELINES

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Summary

Cerebral resuscitation is the most important goal of all resuscitation efforts and for resuscitation to be successful there must be an unbroken chain of events starting from the basic and intermediate life support (BLS and ILS) and ending with advanced cardiovascular life support (ACLS). Time is equally critical and the time interval between the collapse of the victim and start of the resuscitation effort by the rescuer decides the outcome of all resuscitation efforts. Every community should implement the chain of survival which includes early cardiopulmonary resuscitation (CPR), Early Defibrillation and Early ACLS. Many of the resuscitation efforts will be unsuccessful; the rescuer should know when to stop and more importantly when not to start. Advanced planning is equally important and every resuscitation attempt has a structure that evolves over time and through various phases. In our community as well as our hospitals the level of awareness about resuscitation protocols is very poor and we hope that by reviewing these guidelines we will be able to formulate strategies for resuscitation in our community and hopefully implement these strategies in the near future.

Keywords: Basic life support (BLS), Advanced Cardiovascular life support (ACLS), A B C D Survey, Defibrillation.

Introduction

Resuscitation from cardiopulmonary arrest is one of the most cost effective interventions in medicine. Ventricular fibrillation remains the commonest rhythm at the time of arrest and majority of arrests take place out of hospital. Cardio pulmonary resuscitation (CPR) at home or in a public place helps to start the heart and restore breathing only in 50% of the time even in most successful communities. Even after heart restarts, only one half of the ventricular fibrillation (VF) cardiac arrest victims admitted in hospital survive and go home i.e. 3 out of 4 CPR attempts will be unsuccessful. Successful return of circulation in, out of hospital cardiac arrests in countries such as USA varies from 3-33%; only 20% of such patients survive to be discharged by the hospital and only 50% of such cases have acceptable neurological outcome. There is a growing consensus about the fact that return of spontaneous circulation is not the end of resuscitation and many interventions are needed to improve the long term survival and neurological outcome.

Cerebral resuscitation is the most important goal of CPR and some prefer the term cardio pulmonary cerebral resuscitation (CPCR). Resuscitation starts with basic life support, continues as intermediate life support and ends with advanced cardiovascular life support (ACLS). The intermediate life support, a term coined recently bridges the gap between BLS and ACLS; it includes the use of automatic external defibrillators (AED’S) and some of the advanced airway devices by trained rescue personnel. ACLS includes use of advanced equipment and techniques for establishing and maintaining ventilation as well as circulation, maintenance of intravenous access, therapies for patients with cardiac and respiratory arrest besides treatment of patients with acute coronary syndromes (ACS) and eligible stroke patients.

Despite decades of research in ACLS, survival after cardiac arrest even in the western countries remains poor. Due to ethical constraints much of the research work has been carried in mammalian models and some of the well established results have been tested in human volunteers in well designed and carefully constructed study protocols. Resuscitation by first responders and bystanders has become the target in the developed countries and use of automatic external defibrillators (which can analyze rhythm and recommend appropriate action) by lay or trained rescuers is being encouraged. Biphasic and perhaps triphasic defibrillation is replacing the conventional monophasic defibrillation as a procedure that has unequivocally proven to be of same or better efficacy and less damaging to the myocardium. Time is critical; a short interval of time from collapse to care decides all patient outcomes. Every community should strive to implement the chain of survival and provide high quality ACLS components. Unfortunately in our community...
as well as our hospitals there is a low level of resuscitation protocols. Our aim is to review the resuscitation guidelines so that we can develop effective strategies to implement these guidelines in near future. The new recommendations are based on evidence review and consensus opinion and include guidelines from ACLS conference in year 2000; Emphasis has been laid on classifying various interventions on the basis of their proven clinical effects in well thought of clinical trials.

**Classification of recommendations for therapeutic interventions**

<table>
<thead>
<tr>
<th>Class</th>
<th>Supporting evidence</th>
<th>Clinical intervention</th>
</tr>
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<tbody>
<tr>
<td>1</td>
<td>At least one randomized clinical trial</td>
<td>Always useful</td>
</tr>
<tr>
<td>2a</td>
<td>Multiple studies with positive results</td>
<td>Useful and safe</td>
</tr>
<tr>
<td>2b</td>
<td>Evidence is generally but not always positive</td>
<td>Within standard of clinical care</td>
</tr>
<tr>
<td>Indeterminate</td>
<td>Inconsistent</td>
<td>Inconclusive</td>
</tr>
<tr>
<td>3</td>
<td>Studies confirm harm</td>
<td>Harmful</td>
</tr>
</tbody>
</table>

**Adult basic and intermediate life support (BLS and ILS)**

**Primary ABCD survey**

It is the first and the most important component of every resuscitation effort. The first step is to ensure that the victim as well as the rescuer is secured from various environmental factors e.g. at the site of a road traffic accident. The rescuer should then assess responsiveness of the victim; the rescuer should always assume that the victim is in cardiac or respiratory arrest or both unless proven otherwise. In trauma one should consider the possibility of cervical spine injury and ensure that the neck is stabilized so that the injury is not aggravated; the rescuer should position himself, kneel besides the victim at the level of his shoulders.

By convention basic life support makes use of mouth to mouth breathing along with chest compressions; however the former in addition to being technically difficult is not always aesthetically acceptable to a bystander attending to a cardiac arrest victim. Numerous animal studies have shown that even though rescue breathing in combination with chest compressions improve arterial oxygen saturation and pH as compared to chest compressions alone, there is no overall improvement in survival. Overall consensus is that in case of out of hospital cardiac arrest, chest compressions alone by lay rescuers are as effective as its combination with mouth to mouth breathing.

In the hospital setting, new protocols for cardio pulmonary resuscitation have been developed to improve perfusion to the heart and brain while circulation is arrested. These include interposed abdominal compression (IAC-CPR), phased thoracic-abdominal compression decompression (PTACD-CPR) or the life stick CPR and active compression–decompression (ACD-CPR). These techniques require advanced training and equipment and there is evidence of better resuscitation out comes with these techniques. Open cardiac massage may be advantageous in hospital setting if performed immediately after cardiac arrest; however it requires highly trained personnel both at the time of arrest as well as after return of circulation. In pericardial tamponade, pulmonary embolism and penetrating chest injuries, open cardiac massage has the potential to save lives. Emergency cardiac pulmonary bypass through femoral vessels and minimally invasive direct cardiac massage through a 2 cm incision and a wand like instrument have shown promise in certain clinical situations.

Of all the resuscitative interventions to improve survival from VF/ pulseless VT, defibrillation is the most useful intervention. Defibrillators that use a monophasic waveform (current moves in positive direction only) are in use in many parts of the world since the last 40 years. Biphasic defibrillation (current polarity is reversed midway through the discharge) have been studied since the early 1980’s and its use is increasing rapidly as it requires less energy, is less damaging to the myocardium and is associated with a higher rate of successful defibrillation in out of hospital cardiac arrests. Survival from VF cardiac arrest declines by 7-10% for each minute without defibrillation. At 4 minute, irreversible organ damage develops, and at 12 minutes survival rate decreases to 2-5%. Therefore hospitals need to establish programs to achieve early defibrillation (class 1). Automatic external defibrillators are recommended for public sites with a high probability of at least one arrest in five years (class 2b). VF/pulse less VT can be defibrillated by mono phasic shocks (200j-300j-360j) or by biphasic shocks (150j-150j-150j); recent work has shown that it is important to restart CPR after defibrillation if defibrillation produces a pulseless rhythm. There is also a growing consensus that VF victims who do not receive defibrillation even after 5 minutes of the event should receive perfusion therapy(CPR) prior to defibrillation.
ALGORITHM FOR ADULT BLS AND ILS($)

**Breathing**: It has now been made mandatory to confirm the proper tube position in the trachea by 5 point auscultation as well as by secondary techniques including end tidal CO$_2$ indicator and esophageal detector devices (class 2a). After confirmation of the proper placement of the tube the rescuer should secure the tube carefully with a tie, tape or tube holder (class 2b).$^{2,10}$

**Circulation**: The next step is to obtain IV access, determine rhythm and give appropriate agents.

**Differential diagnosis**: The rescuer should search for, find and treat reversible causes.$^{2,10}$

After successful return of circulation it is important to adopt organ protective strategies because all organs especially the brain and the heart are sensitive to oxygen and nutrient deprivation that occurs during and after the circulatory arrest and as a result of reperfusion injury. Deaths are common due to cardiac or neurological failure and most of the deaths occur in the first 48 hours after resuscitation.$^{27}$ The vital organ protective strategies that could improve the outcome of resuscitation include use of low energy defibrillators, use of vasopressors that do not increase myocardial oxygen consumption, hypothermia,$^{17,28}$ use of ionotropic (Dobutamine), metabolic (Glucose-Insulin-potassium), and mechanical (Intra-aortic balloon pumps, cardio pulmonary bypass, minimally invasive cardiac massage), circulatory support; use of K channel openers, sodium/hydrogen exchange blockers, calpain inhibitors (Cariporide), antioxidants and regulation of gene expression is also under evaluation.

There have been atleast two published randomized trials in humans that have compared two groups of post VF cardiac arrest patients who responded to resuscitation with return of spontaneous circulation;$^{29,30}$ the two groups were randomized to receive normothermia and mild hypothermia (32-34°C). Outcome was judged as favorable neurological recovery to permit discharge the patient. The group receiving mild hypothermia was found to have better out comes as compared to those receiving normothermia.

**Overview of acls rhytms**$^2$

**A. Lethal rhythms**
1. VF/pulse less VT
2. Asystole
3. Pulseless electrical activity (PEA)

* Do not shock Asystole.
B. Potentially lethal rhythms
1. Unstable tachycardias**
2. Unstable bradycardias.

**Unstable rhythms are the ones which lead to haemodynamic compromise (failure, decreased level of consciousness, persistent chest pain or continued VPC’S in the setting of a possible acute MI hypotension), congestive heart failure.

C. Non lethal rhythms
1. Stable bradycardia.
2. Stable atrial flutter/fibrillation.
4. Stable broad complex tachycardia; unknown type.
5. Stable VT.

The lethal rhythms²

The lethal rhythms need immediate recognition and appropriate action by the rescuer e.g. if a patient has ventricular fibrillation or pulseless ventricular tachycardia urgent defibrillation is the treatment of choice

while as the same treatment is hazardous and potentially lethal in cases of pulseless electrical activity (presence of electrical depolarization in the absence of a pulse) or asystole (the underlying cause needs to be corrected in the latter situation).

Since the 60’s epinephrine was being used as the drug of choice for shock refractory VF;epinephrine acts as an alpha agonist causing systemic vasoconstriction which maintains peripheral vasoconstriction; which maintains peripheral vascular tone and prevents vascular collapse besides increasing coronary perfusion.²,¹⁰

Vasopressin, a potent vasoconstrictor which acts by increasing cyclic adenosine monophosphate may be superior to epinephrine for treatment of shock refractory VF/pulseless VT; there is no evidence to support it role in PEA or asystole where epinephrine continues to be the initial drug of choice. As compared to epinephrine which has a half life of 3-4 min, vasopressin has a half life of 10-20 min and is given in a single dose of 40 IU IV.¹⁰ Vasopressin is also associated with less side effects, improved haemodynamics, and improved survival compared with epinephrine.

ALGORITHM FOR LETHAL RYTHMS²

primary abcd survey

no pulse

CPR continues /Assess Rhythm

ventricular fibrillation /tachycardia
attempt defibrillation up to 3 shocks
vasopressin 40 u iv single dose
immediate emergent pacing (2b) I brady systolic cardiac arrest
atropine 1mg iv if PEA is slow, repeat every 3-5 min, if needed (total dose 0.04 mg/kg)

asystole/pulseless electrical activity

secondary abcd survey

epinephrine 1 mg iv ,repeat 3-5 min

no response

consider antiarrythmic agents

look for 5H’And 5T,s

hypovolemia tablets (drug overdose)
hypoxia tamponade(cardiac))
h+ ion (Acidosis) tension Pneumo thorax
hyper/hypokalemia thrombosis coronary
hypothermia thrombosis pulmonay
The tachycardias

The rescuer should find out clinically whether the patient is stable or not; it is also important to classify whether it is a broad or narrow complex tachycardia because it guides the choice of drug therapy.

If the patient is unstable, the rescuer should establish heart rate as the cause of symptoms (almost always HR of more than 150). Next step is to cardiovert the unstable rhythm. Modern defibrillators/cardioverters can deliver either unsynchronized or synchronized shocks; in the former the shock may fall anywhere in the cardiac cycle, while as the latter is delivered in synchrony with the peak of the QRS complex. The synchronized shocks thus avoid delivery of shock during cardiac repolarisation, a period of vulnerability during which a shock can precipitate a VF. The ACLS recommendation is to synchronize for patients with stable tachycardias and patients with unstable tachycardias who are not so unstable that a few moments of delay might lead to a further clinical deterioration. Otherwise, to avoid dangerous delays, very unstable patients, such as those in severe shock or pulse less VT, should receive unsynchronized shock. Should the unsynchronized shock cause VF, defibrillation should be attempted immediately.

For synchronized cardio version the first step is to prepare equipment; oxygen saturation monitor, i.v. line and intubation equipment should be available and if possible the patient should be premedicated with midazolam±fentanyl under the supervision of an anaesthesiologist. The next step is to give synchronized DC shocks in monomorphic VT with a pulse, paroxysmal supraventricular tachycardia (PSVT), atrial Fibrillation (AF), atrial flutter with 100-200-300-360J of monophasic shocks or equivalent biphasic shocks 70-120-150-170J. If the patient is unstable the rescuer should go for urgent cardioversion. If the patient is stable the rescuer should then classify the arrhythmia and go to the individual algorithm.

The stable narrow complex tachycardias are further classified into PSVT, multi focal atrial tachycardia (MAT) and junctional tachycardias. The distinction is important because PSVT is caused by reentry phenomenon while as the others in addition to ectopic atrial tachycardia are usually caused by an automatic or irritable focus and do not respond to cardio version.

In VT, emphasis is laid on whether the patient is stable or not, morphology (monomorphic or Polymorphic), ejection fraction and baseline QT. When QT is normal, VT is due to ischemia or electrolyte imbalance; the rescuer should attempt to treat ischemia, correct electrolytes.

In a victim with prolonged QT baseline (Torsades), the rescuer should correct electrolytes, try magnesium, overdrive pacing, isoprotrenol, phenytoin or lidocaine.

Atrial fibrillation/atrial flutter should be treated in a staged fashion depending on several factors; if the patient is unstable, the rescuer should cardiovert at once, if the patient is stable, control of rate gets priority followed by conversion of rhythm if it is deemed to be necessary and/or appropriate.

**ALGORITHM FOR STABLE WIDE COMPLEX TACHYCARDIA, UNKNOWN**

<table>
<thead>
<tr>
<th>Preserved cardiac function</th>
<th>EF less than 40% CHF</th>
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<tbody>
<tr>
<td>DC cardioversion</td>
<td>DC cardioversion</td>
</tr>
<tr>
<td>Procainamide</td>
<td>Amiodarone</td>
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</table>

*The rescuer should try 50J for PSVT or atrial flutter in the first attempt to cardiovert.

**ATRIAL FIBRILLATION /FLUTTER PROTOCOL**

Is the patient Stable or Unstable?

- Proceed more urgently for the Unstable patient
- Is cardiac function impaired?
  - If yes (EF less than 40% or CHF) Use drugs limited to digoxin diltiazem & amiodarone Avoid verapamil, betablockers, procainamide/leocainamide
  - Is WPWS present?
    - If yes a void adenosine, beta blockers calcium channel blockers and digoxin
    - Is the onset of AF less than 48 hours
      - If yes avoid cardioversion or drugs that cardiovert unless guided by TEE or after Anticoagulation 4 weeks prior and 3 weeks after cardio version.
      - For early cardio version IV heparin is given after TEE, cardioversion is done within 24 hours and anticoagulation continued for 4 weeks.
Bradycardias
The key clinical question is whether the bradycardia is making the patient ill or something else is producing the bradycardia. We should treat only the symptomatic bradycardias but we should recognize the red flag bradycardias i.e. bradycardias that are likely to deteriorate even if the patient is asymptomatic. These include:

a. Second degree AV block Type 2.
b. Third degree heart block (complete heart block)

It is important to recognize bradycardias produced by acute myocardial infarction, especially the inferior or the RV infarction; the parasympathetic effects cause bradycardia but hypotension, if present is due to hypovolemia and such patients need a fluid challenge with normal saline (250-500 ml over 15-30 min) and this action may be life saving. Atropine is the agent of choice in the initial management of many bradycardias; it acts by blocking the effects of vagal nerve discharges. However areas of heart not innervated by vagus will not respond to atropine; therefore atropine is not indicated in third degree heart blocks or mobitz type 11 heart blocks. Its use may accelerate the atrial rate and produce increased Av nodal block.

Acute coronary syndromes (ACS)
Acute MI has been recognized as a spectrum of disease requiring a more comprehensive name: acute coronary syndromes. Initially patients with ACS were classified on the basis of their initial ECG as Q wave MI, non Q wave MI and unstable angina and patient management was based on this classification; however now a new ischemic chest pain algorithm has been developed to include all the patients presenting with chest pain. The importance of this algorithm is to provide immediate general assessment (10 min) and immediate general treatment to all patients with chest pain and then to stratify the patients into three groups on the basis of 12 lead ECG and ST segment deviation.

All ACLS providers are trained for the emphasis on early reperfusion in ST elevation, avoiding fibrinolytic therapy in ACS with ST depression as it poses a risk of harm.
ALGORITHM FOR ISCHEMIC CHEST PAIN:**31**

**Immediate assessment<10 min.**
- Vital signs, O2 saturation, brief & targeted history exam
- Check eligibility for fibrinolytics, cardiac enzymes
- Electrolytes coagulation studies, chest X-ray (<30min)

**Immediate General Treatment (MONA)**
- Aspirin 160-325mg po
- Nitroglycerin SL/Spray*

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**Assess 12 lead ECG**
- ST Elevation/New onset LBBB
- ST Depression or dynamic T Inversion/High risk unstable angina
- Non Diagnostic ECG

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**Non Reperfusion Delay**
- ACE Inhibitors after 6 hrs
  - Or when stable

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**< 12 HOURS**
- Select a reperfusion Strategy***
  - Fibrinolytic Therapy PCI/CABG
- Assess Time from Onset of Symptoms
  - High Risk Defined by:
    - Persistent Symptoms
    - Recurrent Ischemia
    - Depressed LV Function
    - Global ECG Changes
  - Prior AMI/PCI/CABG
- Perform Cardiac Catheterization
- Primary PCI Selected
  - Experienced Operators (>75/yr)
  - High volume center (>250/yr)
  - Cardiac Surgical Back up
  - Goal door to door in 60-120min

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**> 12 HOURS**
- Unstable/New onset Angina
  - Or YES Troponin Positive

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**ADMIT IN CCU**
- Continue Adjunct Therapy
  - Serial ECG/Cardiac Enzyme
  - Consider Imaging Study
  - (2D Echo/Radio Nuclear)
- EVIDENCE OF ISCHAEMIA
  - YES
  - NO
- DISCHARGE/FOU

**Unstable/New onset Angina**
- Or YES Troponin Positive

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**New recommendations for acute coronary syndromes**:31

**a.** All patients with MI need aspirin and beta blockers in the absence of contraindications.

**b.** Prehospital 12 lead ECG (class 2a) improves prehospital diagnosis and reduces hospital based time to treatment.

**c.** Use of prehospital checklist for indications and contraindications, could shorten the time to reperfusion.

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**A. ST Elevation Acute MI:**31

**a.** Pre hospital fibrinolytic therapy (class 2a) is beneficial to eligible patients when transport to hospital is >60 min.

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**b.** The patients with cardiogenic shock or heart failure due to acute MI need to be shifted to centers where PCI/CABG is available if transport time is 30-45 minutes.

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**c.** PCI can be equivalent to fibrinolytic therapy in big centers with experienced operators.

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**d.** PCI is superior to fibrinolytics in patients <75 yrs of age with cardiogenic shock.

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**e.** Heparin 80 mg/kg bolus and 12 mg/kg/hr infusion is indicated as adjunct therapy with fibrin specific lysics (e.g. alteplase) and for all patients undergoing PCI.

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**f.** ACE inhibitors are indicated for patients with Congestive heart failure (CHF), large AMI or EF<40%.
B: ST Depression AMI

Agents that inhibit glycoprotein IIb/IIIa receptors especially short acting ones like Eptifibatide and Triofiban improve prognosis in patients at high risk due to ST depression, positive markers and refractory ischemia; they are also recommended for patients undergoing PCI.

a. Enoxaparin, a low molecular weight heparin (LMWH) is superior to unfractionated heparin (UFH) and is easy to administer without rebound angina.

b. Use of triple anti thrombotic therapy (asprin and clopidrogel*, GP IIb/IIIa inhibitors and UFH/LMWH) is the most effective treatment of ST- depression AMI and unstable angina.

Pharmacology of resuscitation

a. Amiodarone (class 2b) 300 mg IV push is drug of choice for cardiac arrest from VT/VF that persists after multiple shocks. (Lidocaine 1.0-1.5 mg kg\(^{-1}\) is class indeterminate for the same indication).\(^8\),\(^32\),\(^33\)

b. Amiodarone and Sotalol (class 2b) are recommended agents for treatment of stable monomorphic and polymorphic VT.\(^32\)

c. Amiodarone and procainamide (class 2b) are recommended ahead of adenosine for the treatment of stable wide complex tachycardia.\(^32\)

d. Bretyllium\(^**\) has been dropped from the VF/pulseless VT protocol.\(^32\)

*Approximately 20% patients are resistant to Asprin

**Worlds supplies of bretyllium are nearly exhausted

e. Magnesium sulfate (class2b) 1-2 g IV is given in torsades de pointes or when suspected arrhythmia is caused by hypomagnesaemia.\(^31\)

f. Procainamide (class indeterminate) up to 50 mg kg\(^{-1}\) is given to VF/VT victims who respond to shocks with intermittent return of pulse or a non VF/VT rhythm but the arrhythmia recurs.\(^26\)

g. Vasopressin, (single dose 40 IU IV class 2b) may be more efficacious than epinephrine for promoting the return of circulation after cardiac arrest in VF/ pulseless VT resistant to multiple shocks.\(^34\)

h. High dose epinephrine 0.1 mg kg\(^{-1}\) in adults (class indeterminate) does not seem to improve survival or neurological outcome in cardiac arrest patients.\(^35\)

i. Recombinant tissue plasminogen activator (rtpa) improves neurological outcome when given to eligible stroke patients within 3 hours of stroke (class 1).\(^34\)

Sodium Bicarbonate\(^2\)

a. Class 1: In case of known preexisting hyperkalemia.

b. Class 2a: In case of known, pre existing bicarbonate responsive acidosis.

c. Class 2a: In tricyclic antidepressant over dosage.

d. Class 2a: To alkalinize urine in asprin or other drug over dosages.

e. Class 2b: For intubated and ventilated patients with a long arrest interval.

f. Class 2b: Return of circulation after a long arrest interval.

g. Class3: harmful in hypercarbic acidosis.

Conclusions

a. Cerebral resuscitation is the most important goal of ACLS, unless spontaneous ventilation and circulation are started quickly, successful resuscitation cannot occur.

b. Never forget the patient, treat the patient and not the arrhythmia; emergency care providers must constantly review the resuscitation rather than focus on a single resuscitative effort.

c. For resuscitation to be successful there must be an unbroken chain of events starting with the BLS and ending with ACLS; the two are joined by ILS (intermediate life support) which includes use of automatic external defibrillators (AED’s) and some of the advanced airway devices by a variety of out of hospital personnel.

d. Time is critical, the longer it takes to restore the heart beat, the less are the chances of a successful resuscitation.

e. Seek and treat the cause; people who experience arrest in VF need a defibrillator, while as people who arrest in asystole or PEA need a diagnosis followed by an intervention to correct the abnormality. Similarly diagnosis and treatment of acute MI in time is critical.

f. Post resuscitation care is equally important; once the heart has started keep it started.

g. Survival from VF cardiac arrest declines by 7-10% for each minute without defibrillation.

h. Advanced planning is very important and every resuscitation attempt has a structure that evolves over time through various phases; these include anticipation of patient arrival in hospital, transfer to reception of the patient by the resuscitation team, resuscitative efforts by the primary team, maintenance of the patient.
after the return of circulation and notification of the patient’s family of the expected outcome.

a. Good resuscitation requires a careful thought about when to stop resuscitation efforts and even more important, when not to start. We should not resuscitate the patients where disease has reached the end of its clinical course and life stops; nor should we resuscitate people against their wishes.*

b. The chain of survival which includes Early CPR, Early Defibrillation, and Early ACLS should be applied and strengthened in our community, both at our hospitals as well as our homes.

c. Life stick resuscitation is feasible and safe and may be advantageous in patients with asystole or PEA.

d. Resuscitation by chest compressions in VF/pulseless VT by lay rescuers without mouth to mouth breathing is feasible and effective (pilot study did not show any difference in outcome of two groups one resuscitated with both ventilation and chest compressions while as the other group resuscitated with chest compressions only).

*People who have do not attempt resuscitation orders (DNAR)

References


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