Updates in ACLS

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Updates in ACLS

ANCC Accredited NCPD Hours: 1.00 hrs
Target Audience: RN/APRN

Need Assessment

Basic life support (BLS) is the generic term for any form of cardiopulmonary resuscitation. Advanced cardiac life support (ACLS) is primarily required for health care professionals working a hospital setting while caring for critically ill. When a sudden cardiac arrest occurs, immediate cardio pulmonary resuscitation is a vital link in the chain of survival. Another important link is early defibrillation, which has improved greatly with the widespread availability of automated external defibrillators (AED). It is always recommended that emergency medical dispatchers receive better guidance on recognizing potential cardiac arrests and agonal breathing to promote cardiopulmonary resuscitation.

Objectives

- Describe the role of advanced cardiac life support during in-hospital cardiac arrest
- Identify any three factors which are to be ensured for in-hospital cardiac arrest patients
- Discuss the benefit of ventilation in cardio-pulmonary resuscitation
- Discuss the role of oxygen therapy in cardiac arrest
- Discuss the advanced airway management in advanced cardiac life support
- Understand the various drugs used in advanced cardiac life support

Goal

The goal of this article is to highlight the updates in the field of cardiology in advanced cardiac life support

Introduction

Advanced Cardiac Life Support (ACLS), the fourth link in the chain of survival concept for sudden cardiac arrest, is highly dependent on the optimal conduct of the earlier three links, i.e. early access, early cardiopulmonary resuscitation (CPR) and early defibrillation. *Arrhythmia management is a cornerstone of ACLS guidelines. Post-resuscitation interventions (i.e. measures carried out after return of spontaneous circulation [ROSC]) to increase the chance of being discharged alive from hospital have assumed increasing prominence.* Of note, it is important to monitor the quality of the various care procedures. The resuscitation team needs to reduce unnecessary interruptions to chest compressions in order to maintain adequate coronary perfusion pressure during the advanced cardiac life support drill. In addition, the team needs to continually look out for reversible causes of the cardiac arrest. [1, Rank 4]

Updates in ACLS

Cardiac arrest may occur outside or within the hospital and is termed out-of-hospital cardiac arrest (OHCA) or in-hospital cardiac arrest (IHCA), respectively.

Out-of-Hospital Cardiac Arrest

In the out-of-hospital environment, the emphasis is on early recognition by the public and access to emergency pre-hospital care, (as shown in figure 1) *prompt initiation of good-quality cardio-pulmonary resuscitation by the bystander, public access defibrillation (PAD),* early advanced care by emergency ambulance crew and transport to hospital emergency departments (ED) that are capable of delivering resuscitation and post-resuscitation care.

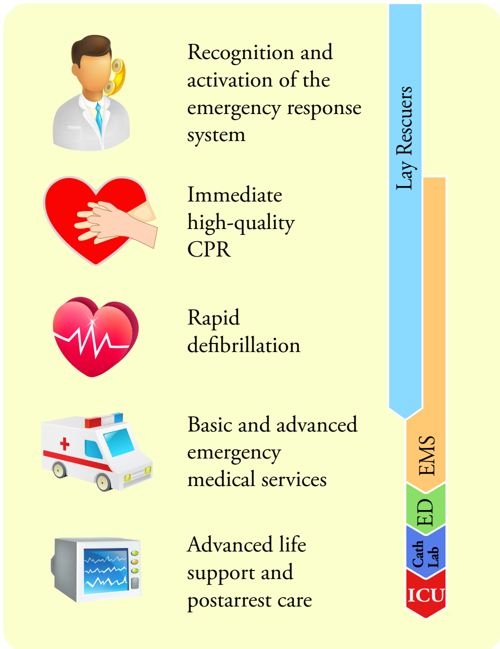

Figure 1: Links in chain of survival

Public Access Defibrillation

Public access defibrillation is another key intervention to improve the time to defibrillation for shockable rhythms in out-hospital cardiac arrest, through increasing the availability of automated external defibrillators (AEDs) in public places. ***Patients with shockable rhythms who received bystander defibrillation had 5.8 times the odds of survival compared to those who did not.***

Emergency Ambulance Response

Pre-hospital emergency medical services should optimise the rapid dispatch of first-responders who are trained in Basic Cardiac Life Support (BCLS) and automated external defibrillator use (as shown in figure 3), as well as paramedics trained in advanced life support protocols, to the scene within eight minutes. ***This can be achieved through strategic pre-placement of ambulances and related vehicular assets, as well as stratified levels of responses for calls of different priority levels.*** [2, Rank 3]

Survival in out-hospital cardiac arrest is critically dependent on prompt institution of the first three links of the chain of survival (as shown in figure 2). ***Pre-hospital factors, including witnessed arrests, initial shockable rhythms, bystander defibrillation and emergency ambulance response times within eight minutes of collapse, have been found to be associated with improved survival outcomes.*** As is well known, ***survival in out hospital cardiac arrests decreases at a rate of 7%–10% with each passing minute without cardio-pulmonary resuscitation after cardiac arrest.***

> **“ Immediate CPR can double or triple chances of survival after cardiac arrest ”**

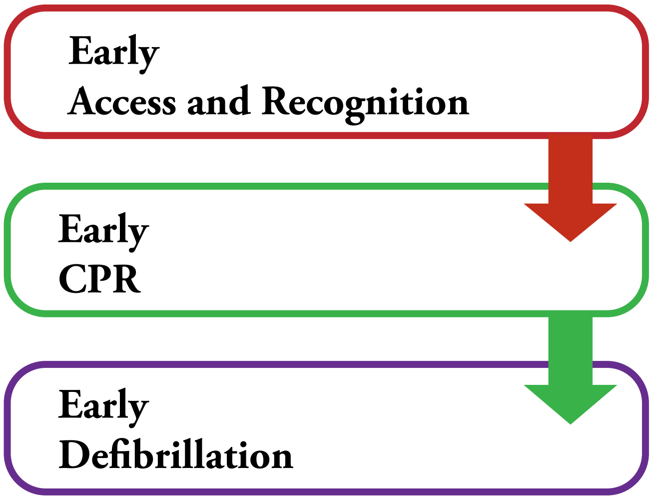

Figure 2: First three links of the chain of survival

The low survival currently seen in many communities reflect the challenges in delivering timely and high-quality pre-hospital resuscitation care. The current overall survival rate of out hospital cardiac arrest in Singapore is 2.5%–3.2%, with witnessed ventricular fibrillation (VF) survival rates at 11%. There remains a need to better understand and address these gaps in the community. In addition, ***ensuring the best standard of advanced cardiac life support in the hospital comprises an integral part of the continuum of resuscitation care for the out hospital cardiac arrest victim.*** [3, Rank 4]

The majority of cardiac arrests managed in emergency departments are out-hospital cardiac arrest. Most of these patients are conveyed by emergency ambulances and a small number by private transport or private ambulances. Some patients may go into cardiac arrest only after arriving at the emergency department. ***Limited data exists on the outcomes of such arrests, although survival factors may include patient acuity, co-morbidities, early recognition and rapid response.*** [4, Rank 5]

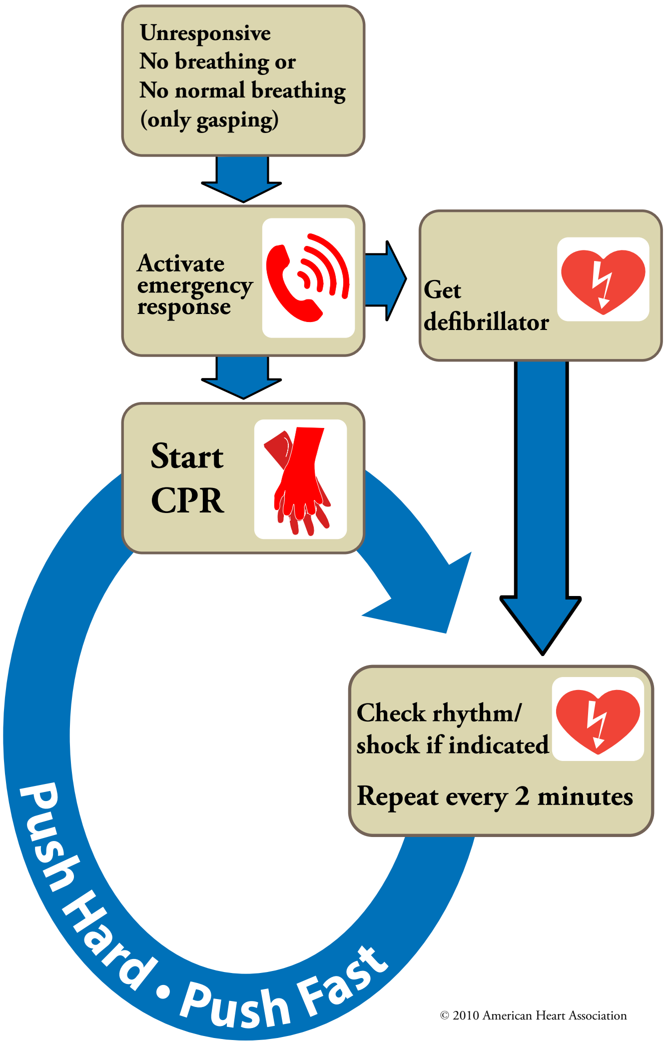

Figure 3: Adult BLS algorithm

In-hospital Cardiac Arrest

A significant number of patients sustain cardiac arrests in the hospital environment. Some inpatients may have had a period of deterioration and an unwitnessed arrest. There may be significant variations in the time of detection of these cardiac arrests in the hospital. ***It is important to have a system for early recognition of patients at risk for cardiac arrest,*** (as shown in figure 4) ***through the use of early warning systems and detection of unexpected cardiac arrests, as well as a system of rapid response.***

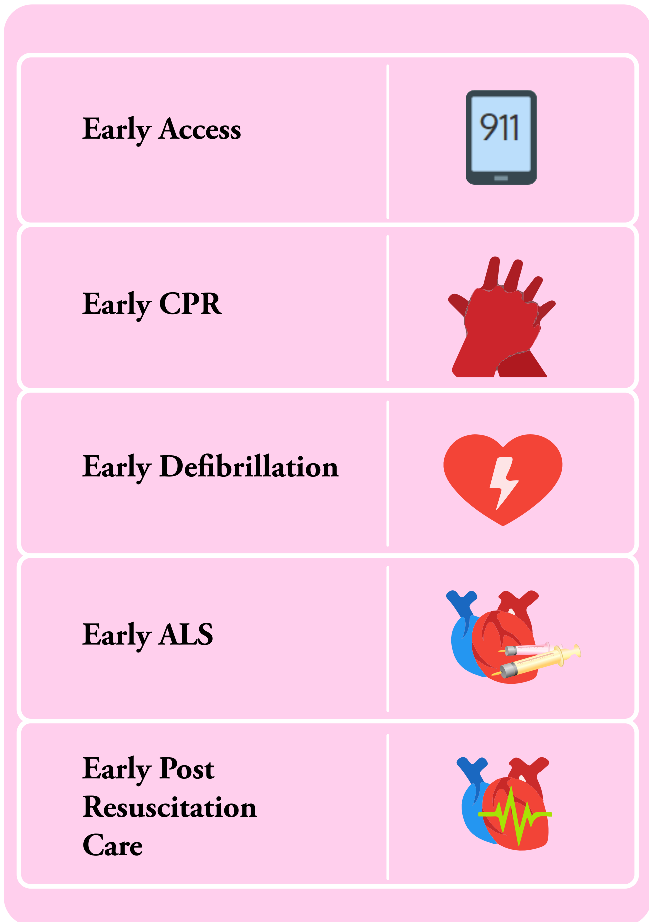

Figure 4: Five links of the chain of survival

For all in-hospital cardiac arrest patients, it is important to ensure the presence of the following (as shown in figure 5):

» Early detection of cardiac arrest.

» A system of calling for help within all clinical care areas, including inpatient wards, ambulatory clinics, operating theatres, intensive care units and public areas of the hospital.

» Staff who are immediately available to perform cardio-pulmonary resuscitation and operate an automated external defibrillators or defibrillator in the automated external defibrillators mode, as well as staff trained in advanced cardiac life support interventions, at all times.

» Resuscitation equipment, including defibrillators, advanced airway devices and drugs, is to be brought to the patient in the shortest possible time; the listing and layout for this equipment should preferably be standardised within each institution. [5, Rank 4]

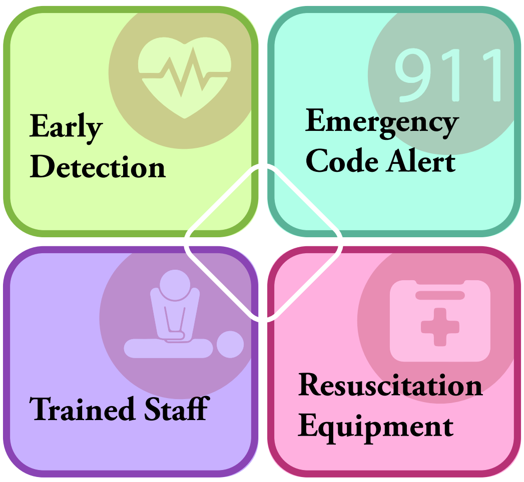

Figure 5: Important factors in early management of cardiac arrest

Medical Emergency and Rapid Response Teams

Various institutions have introduced medical emergency teams, rapid response teams, cardiac arrest teams or Code Blue teams to address the need to manage cardiac arrests. Institutions should have the capability to mobilise these teams promptly, so as to provide better oversight and management of resuscitations. Furthermore, it is important to ensure that all staff with patient care rights already possesses a strong foundation in good basic and advanced life support skills.

All clinical staff of healthcare facilities should also be expected to be currently certified in basic cardiac life support and automated external defibrillation, unless they are excused owing to health reasons. In addition, all acute hospitals should have currently certified basic cardiac life support and advanced cardiac life support-trained personnel who are available 24 hours a day. [6, Rank 3]

> **“ Code blue team: Trained patient care providers who perform resuscitation on any patient who sustains cardiopulmonary arrest ”**

Ventilations in Cardio-pulmonary resuscitation

In contrast to basic cardiac life support, advanced cardiac life support has the benefit of additional manpower, expertise and equipment. In the advance cardiac life support setting, there is no reason to omit ventilations in the provision of cardio-pulmonary resuscitation. However, the question that arises is whether there is a need to synchronise ventilations with compressions during advanced cardiac life support. Prior to advanced airway management in advanced cardiac life support (as shown in figure 6), the recommended rate of ventilation is 10 breaths/minute or one breath every ten compressions when the compression rate is 100–120/minute. Standard cardio-pulmonary resuscitation at 30:2 is equally acceptable. After an advanced airway is inserted, there is no further need to interrupt the compressions and uninterrupted compressions at 100–120/minute with continuous ventilations at 10 breaths/minute should follow.

Care must be taken during the delivery of positive pressure ventilations. Unnecessarily high tidal volumes and ventilation rates are unhelpful, as cardiac output in cardio-pulmonary resuscitation is only about 30% of normal and pulmonary perfusion will thus be mismatched with excessive ventilation. Excessive bag-mask ventilation (BMV) leads to increased intra-thoracic pressures that further decrease venous return and cardiac output. Gastric insufflations, regurgitation and aspiration, as well as diaphragmatic splinting that further impedes effective ventilation, can also occur. The recommended tidal volume is 400–600 mL. If bag-mask ventilation is used, it is recommended that the user should compress the bag by about one-third, sufficient to produce a chest rise over one second. [7, Rank 3]

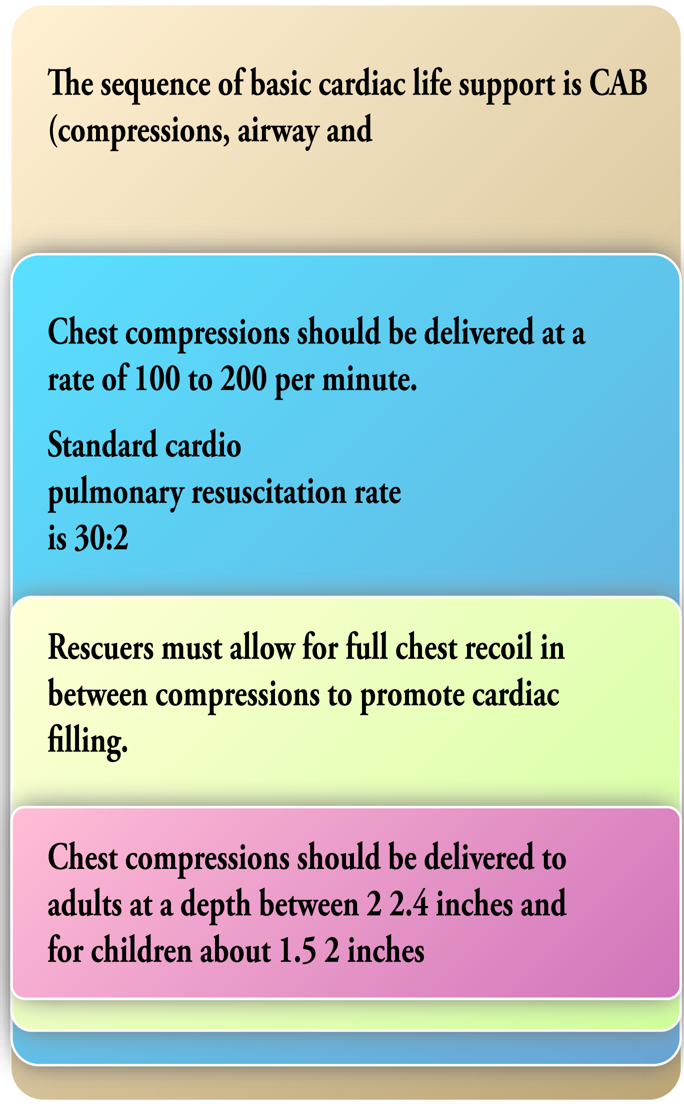

Figure 6: Recommendations in BLS

Rhythm Check Cycles

Once a defibrillator or automated external defibrillators is available, rhythm checks (as shown in figure 7) should be performed immediately and at every 1–2 minutes thereafter. The rhythm may be obtained using either electrocardiography (ECG) monitoring leads or self-adhesive defibrillator pads. Once the pads or ECG leads have been initially applied; cardio-pulmonary resuscitation is interrupted for rhythm analysis.

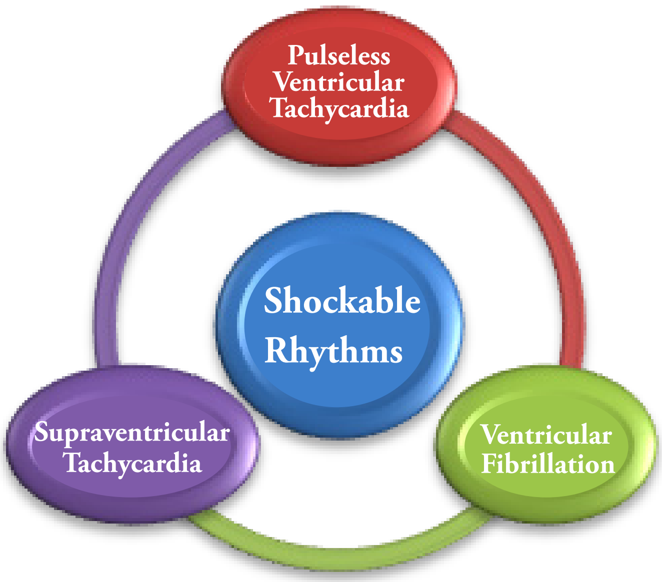

Figure7: Shockable rhythms

During the 1–2 minutes of cardio-pulmonary resuscitation in-between rhythm analyses, there should be no interruptions to chest compressions. A common misconception is that the rescuer should pause chest compressions to analyse the rhythm if the rhythm changes during cardio-pulmonary resuscitation. This practice should be stopped, because reacting to rhythm changes during cardio-pulmonary resuscitation will only increase the number of interruptions to cardio-pulmonary resuscitation (as shown in figure 8). The team should only perform a rhythm check every 1–2 minutes, with good-quality cardio-pulmonary resuscitation maintained throughout. [8, Rank 4]

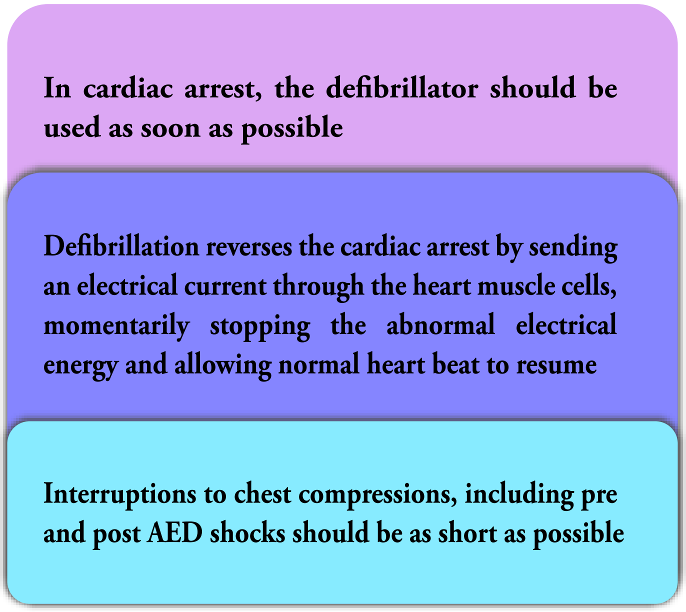

Figure 8: Recommendations in defibrillation

Drugs that may be used in refractory ventricular fibrillation/ polymorphic ventricular tachycardia (VT) include amiodarone and lignocaine. Successful conversion with reversal of spontaneous circulation should be followed by an infusion of either amiodarone or lignocaine in the recommended doses. [9, Rank 3]

Nonshockable rhythms: pulseless electrical activity/asystole

In the absence of a shockable rhythm/asystole, (as shown in figure 9) other rhythms may be seen on the electrocardiogram and a pulse check lasting no more than ten seconds may be performed. If no pulse can be reliably felt, then the rhythm is a form of pulseless electrical activity (PEA). Cardio-pulmonary resuscitation should be restarted immediately for 1–2 minutes before the next analysis.

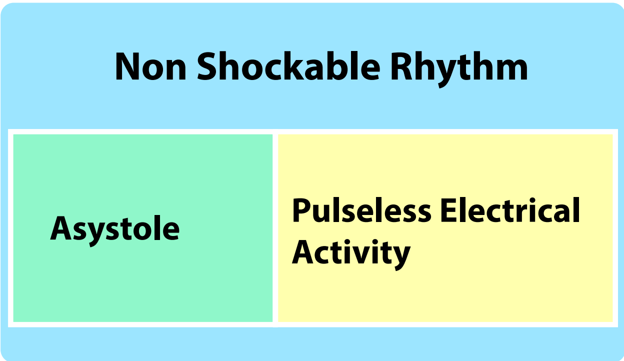

Figure 9: Non shockable rhythm

If the rhythm monitor shows a flat line, perform the following checks to identify fine ventricular fibrillation (do not take more than ten seconds to complete the checks):

» Check the connections to ensure that the leads are attached properly.

» Ensure that the cardiac monitor is not on paddle mode.

» Select different leads on the rhythm monitor to look for the presence of any fine ventricular fibrillation along the different axis.

» Ensure the amplitude of the electrocardiogram tracing is normal.

If the electrocardiogram rhythm remains a flat line despite these checks, the patient is in asystole. Commence cardio-pulmonary resuscitation immediately for 1–2 minutes before the next analysis. [10, Rank 5]

Oxygen Therapy in Cardiac Arrest

The basic objective of ventilation in advanced cardiac life support is to ensure oxygenation of tissues. Although there have been no studies directly comparing fractions of inspired oxygen delivered during resuscitation with survival, indirect studies have found that patients with higher PaO_2 levels during resuscitation were more likely to achieve reversal of spontaneous circulation. From the currently available literature, it is recommended that 100% oxygen or the highest available oxygen concentrations be used during cardiac arrest resuscitation.

After reversal of spontaneous circulation, hyperoxaemia should be avoided during post-cardiac arrest care. Several studies have found poor neurological and survival outcomes with hyperoxaemia following reversal of spontaneous circulation. After reversal of spontaneous circulation, oxygen therapy should be titrated to normoxia (SpO_2 94%–98%) as soon as possible. [11, Rank 3]

Airway Control and Management

An open airway is crucial for the delivery of oxygen to the lungs and tissues.

Access to the airway should be ensured within a few minutes of the start of resuscitation. All healthcare workers should be familiar with the skills of basic airway techniques (as shown in figure 10). Basic airway opening techniques and principles include the following:

» ***The head-tilt, chin-lift manoeuvre*** remains the basic initial airway opening method.

» The ***classical or modified jaw thrusts*** are alternative methods, particularly in suspected cervical spine injury (e.g. patient with a history of fall or diving before cardiac arrest).

» Once opened, the airway needs to be cleared of secretions or foreign material. A blunt-tipped stiff suction catheter is recommended over flexible suction catheters.

» Airway adjuncts help to maintain patency of the oral or nasal air passages:

» ***Oropharyngeal airways*** prevent the tongue from occluding the upper airway and it may be used in unresponsive patients with no cough or gag reflex, during bag and mask ventilation

» ***Nasopharyngeal airways*** may be used if a clenched jaw prevents insertion of the oral airway; it has been known to cause bleeding in up to 30% of cases and should be used with caution in the presence of craniofacial injury. [12, Rank 4]

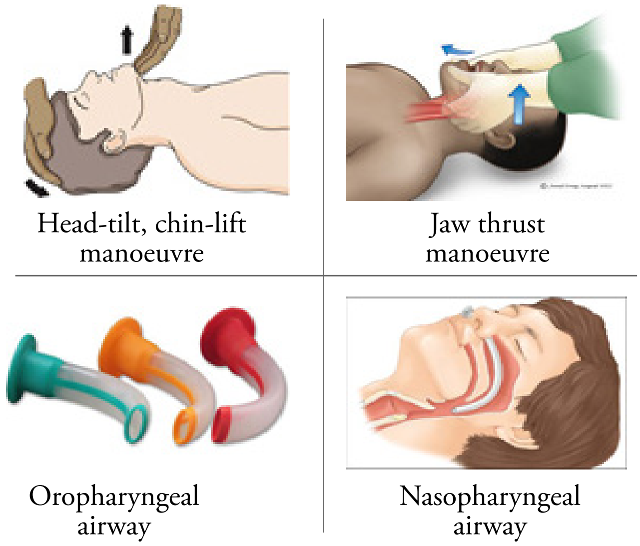

Figure 10: Basic airway opening techniques

Placement of Advanced Airways

The aim of advanced airway management in advanced cardiac life support is to ensure a secure passage for oxygen delivery and ventilation. The placement of endotracheal tubes (ETTs) or supraglottic airways is discussed below (as shown in figure 11).

Endotracheal Tube

Endotracheal intubation remains a core skill in advanced airway management. The benefits of endotracheal tube placement include allowing a definitive patent airway, suction of secretions, reliable oxygen delivery and protection from aspiration of gastric contents. It is indicated when bag-mask ventilation is ineffective or if the patient is in coma or cardiac arrest (absence of gag reflex).

Cricoid Pressure

Cricoid pressure may offer some protection from aspiration and gastric insufflation. However, it may also impede ventilation and interfere with intubation. The routine use of cricoid pressure prior to intubation – a practice prior to the 2010 guidelines revision – is no longer recommended.

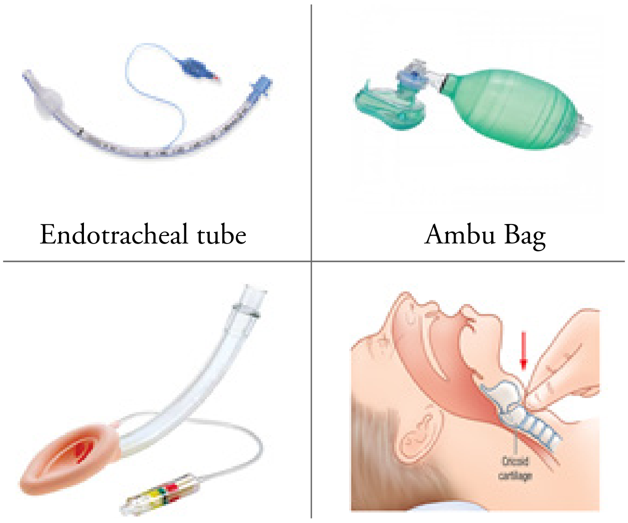

Figure 11: Advanced airway management equipments

Confirmation of Tube Placement

Following placement of an endotracheal tube, confirmation of placement is paramount, as missed oesophageal intubations are more harmful than no intubation. The expected standards for confirming correct placement clinically include (as shown in figure 12) bilateral chest expansion; misting and demisting of endotracheal tube during bag-mask ventilation; and five-point auscultation, beginning from the epigastric area, bilateral upper lobes and lateral chest area bilaterally.

In addition to clinical assessment, continuous waveform capnography is recommended as a reliable method of confirming and monitoring correct placement of an endotracheal tube. If continuous waveform capnometry is not available, non waveform carbondioxide detectors, oesophageal detector devices or ultrasonography used by an experienced operator are reasonable alternatives. Chest X-ray (CXR) should also be performed to assess the depth of endotracheal tube after a patient has achieved reversal of spontaneous circulation (of note, CXR cannot differentiate between an endotracheal tube in the oesophagus and one in the trachea, as both are midline structures). [13, Rank 4]

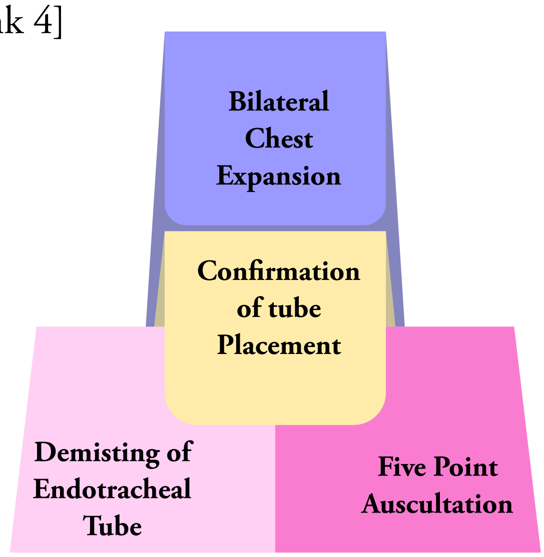

Figure 12: Confirmation of ET tube placement

Complications

Endotracheal intubation performed by an unskilled provider can result in the following:

» Trauma to the oropharynx with bleeding.

» Prolonged interruptions to compressions/ventilations.

» Prolonged hypoxemia with resulting cerebral hypoxia.

» Failure to diagnose misplacement/ displacement of tube, as recognised in 6%–25% of instances.

Therefore, there is a need for regular training and retraining in these procedures to ensure currency of skills. Institutions must determine the frequency of such training and maintenance of competence standards for physicians who are expected to perform advanced airway placements. Vital signs monitoring during intubation for cardiac arrest situations is of limited value, as peripheral circulation is impaired and SpO2 measurement is ineffective or inaccurate. [14, Rank 4]

Criteria Related to ACLS

The other set of criteria corresponds to reported prognostic factors in out-of-hospital cardiac arrest patients. First, the duration of no-flow was shorter in advanced cardiac life support patients as compared to no-advanced cardiac life support patients. In the latter group, our study reports a median duration of no-flow of 15 min, exceeding the possibility of survival without intervention according to the model described by Larsen. The validation of no-flow duration for the decision to initiate advanced cardiac life support needs further research. Patients presenting cardiac arrest at home or in nursing home are less likely to be resuscitated compared to other places. The importance of location on cardiac arrest resuscitation and outcome has been emphasized. Cardiac arrest patients present better outcome if it occurs in a public place. Actually, the circumstances explain this result with a higher percentage of shockable rhythm, the presence of bystanders and the application of automated external defibrillators. Although poorly studied, the social pressure exerted by families or bystander could influence the medical decision in different ways. However, families or relatives could influence the decision differently. Indeed, they represent an important source of information about the medical condition of the patient or do-not resuscitate order and hence can influence on the decision to resuscitate or not. At the same time, the decision to initiate resuscitation in the presence of relatives could influence their psychological outcome. Asystole was associated with the absence of advanced cardiac life support, whereas a shockable rhythm or automated external defibrillator application was more frequent in the advanced cardiac life support patients. This parameter represents also a described prognosis factor in out-of-hospital cardiac arrest in the field or after admission to intensive care unit. [15, Rank 5]

However, the results show a large overlap between the 2 groups, meaning that a large proportion of patients share similar characteristics. The key question is to understand why does a physician start advanced cardiac life support or refrain to initiate resuscitative efforts in patients presenting similar characteristics. Apparently numerous prognosis factors (age, sex, no-flow, location, initial rhythm) are incorporated to establish a prognosis in a few seconds. In the absence of recommendations, it sounds like a subjective decision based on the perception of the potential outcome of the patient. Thus, a decision to withhold advanced cardiac life support in a patient having a potential good outcome would be a major ethical concern.

Even if the emergency physicians filled out the form after each intervention, there is a possible discrepancy between what is declared and what is actually done. It was impossible to evaluate several parameters such as the influence of the medical condition on care or the quality of basic life support. Moreover, the influences of relatives or the social pressure during interventions have not been analyzed. These situations probably resulted in initiation of advanced cardiac life support, whereas it was considered futile by the emergency physicians. [16, Rank 4]

Vascular Access and Drug Therapy

The use of drugs in cardiac arrest patients serves as an adjunct to the earlier components of care, viz. airway control and ventilation management, ensuring good-quality chest compressions with minimal interruptions and prompt arrhythmia management. The special features concerning the use of pharmacological agents during cardiac arrest management are elaborated below. [17, Rank 3]

Routes of drug delivery (as shown in figure 13)

- **Peripheral large-calibre veins,** especially the antecubital and external jugular veins, are the commonest routes to be used.
- **Intraosseous cannulation** can allow drug delivery to non-collapsible venous plexuses in the bone marrow. This may be rapidly inserted as an alternative route of access if intravenous (IV) access is not easily available. Rates of drug delivery are expected to be similar as for the intravenous route.
- **Central venous lines,** either through the subclavian or internal jugular veins, shorten medication access to the central circulation. Central lines, however, cannot be used for rapid infusion of fluids. The insertion of a central line is also likely to result in interruptions to chest compressions. Be aware of complications of central

line insertion such as pneumothorax, and bleeding into the pleural space.

» **Endotracheal administration** of drugs is no longer recommended because drug levels achieved are suboptimal and doses required to achieve blood levels that are similar to the intravenous route may be about 3–10 times as much.

» **Implanted vascular access devices,** such as peripherally inserted central catheters or Port-a-caths, may be used with appropriate aseptic technique and care to avoid air emboli. [18, Rank 4]

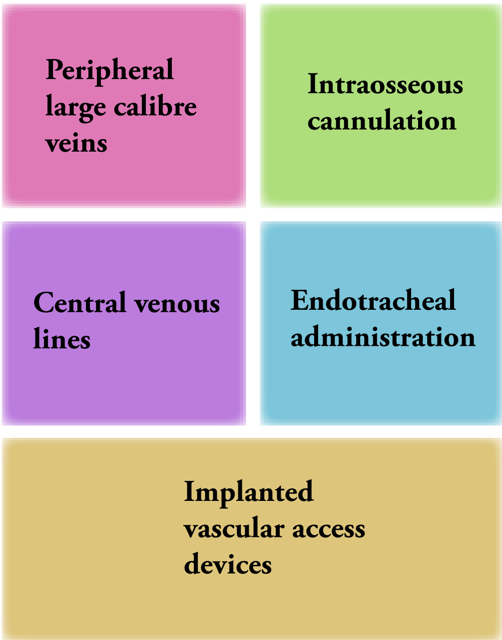

Figure 13: Routes of drug delivery

Prolonged circulation time during Cardiopulmonary resuscitation

Circulation time during cardio-pulmonary resuscitation is prolonged. Drugs will take 30–60 seconds of good-quality cardio-pulmonary resuscitation to reach the central circulation and peripheral vasculature to exert their effects. Each drug dose should be flushed with a 20-mL bolus of normal saline. The administration of drugs should not interrupt cardio-pulmonary resuscitation or delay the delivery of shocks, bearing in mind that the effect of the drug may not yet be evident until after the next cycle of cardio-pulmonary resuscitation, owing to the time it takes to reach the central circulation.

Common drugs used in resuscitation (as shown in figure 14)

» **Adrenaline** is indicated for asystole, pulseless electrical activity and refractory ventricular fibrillation. It should be administered in a 1.0mg dose diluted to 10 mL (1:10,000) for bolus administration every 3–5 minutes.

» **Amiodarone** is given intravenously at a dose of 300-mg bolus in refractory or recurrent ventricular fibrillation/ pulseless ventricular fibrillation or pulseless ventricular tachycardia that persists or recurs after one or more shocks. This may be repeated at a dose of 150 mg, if necessary.

» **Lignocaine** is an alternative to amiodarone for patients in cardiac arrest due to refractory or recurrent ventricular fibrillation/ pulseless ventricular tachycardia. It is usually given at a dose of 1–1.5 mg/kg body weight bolus.

Figure 14: Common drugs used in resuscitation

Sodium bicarbonate (NaHCO3) may be given for specific indications such as hyperkalaemia and tricyclic antidepressant overdose. It is usually given quickly as 1–1.5 mL/kg body weight (i.e. 50–100 mL) of intravenous 8.4% NaHCO3. It is no longer recommended for routine administration in resuscitation from prolonged cardiac arrest. [19, Rank 3]

Pre-Arrest Algorithm

Prognostic factors associated with survival after in-hospital cardiac arrests are an important focus of ongoing research. Patients admitted to hospital have increasingly complex conditions and present unique challenges when managing in-hospital cardiac arrest. Clinicians have to rapidly process many factors related preadmission status (including age, sex, co-morbidities) and factors related to the arrest itself (whether the arrest was witnessed or monitored, initial rhythm) to determine the effectiveness of ongoing pulmonary resuscitation.

Potentially Unstable Dysrhythmias

Advanced cardiac life support includes the management of potentially unstable dysrhythmias, such as tachydysrhythmia and bradydysrhythmia, in patients who are not in cardiac arrest. These dysrhythmias have the potential for deterioration, leading to impaired cardiac output, shock and cardiac arrest and should thus be managed promptly. However, it must be remembered that other forms of shock, including hypovolaemic, haemorrhagic, distributive, obstructive and cardiogenic shock, may be present in the patient. Dysrhythmias constitute only one mechanism of cardiogenic shock. Rhythm control is part of the holistic management of the patient, which should include considerations of other mechanisms of shock and underlying conditions

The general approach to dysrhythmia management has been standardised across the various algorithms. The management of each dysrhythmia is based on clinical stability. In general, hypotension, altered mental status, chest pain, breathlessness, signs of

“ Determination of hemodynamic status is central to arrhythmia management in the in-patient setting ”

shock and heart failure are considered serious signs and symptoms that may be attributable to impaired cardiac output caused by the rhythm, thus suggesting haemodynamic instability. Patients should be managed with vital sign and electrocardiogram monitoring in clinical areas equipped for resuscitation. Supplemental oxygen should be given as necessary. A 12-lead electrocardiogram should be obtained as far as possible. If time does not allow, the cardiac monitor may be utilised for rhythm assessment. [20, Rank 4]

Wide Complex Tachycardia

The majority of wide complex tachycardias (WCTs) are due to ventricular tachycardia. A small subset is due to supra ventricular tachycardia (SVT) with aberrant conduction. Clinical (i.e. patient with a history of ischemic heart disease) and electrocardiogram features specific to ventricular tachycardia (i.e. atrio ventricular dissociation capture beats and fusion beats) exist, but in the presence of clinical doubt, the patient should be managed as for ventricular tachycardia. [21, Rank 4]

Unstable Patients

Patients with wide complex tachycardias who have serious signs and symptoms of instability should be treated rapidly with urgent synchronised cardioversion as follows (as shown in figure 15):

- Explain the procedure to the patient.
- Give analgesia and sedation carefully in titrated doses (beware that drugs used in procedural sedation may have negative effects on haemodynamics).
- Energy levels should begin at 100 J; evidence for different energy levels for monophasic or biphasic defibrillators is lacking.
- Ensure safety as for defibrillation; if the shock is unsuccessful, the energy level may be escalated and repeated (150/200J) subject to the maximum energy level permitted by the individual defibrillator.
- Once converted, an infusion of amiodarone at 1 mg/min for the first six hours followed by 0.5 mg/min for the next 18 hours, or lignocaine at 1–2 mg/min for 24 hours should be given. [22, Rank 4]

Explain the Procedure

Analgesia and Sedation

Safe and Clear Defibrillation

Amiadarone /Lignocaine Infusion

Figure 15: Cardioversion in unstable WCTs

Stable Patients

If the patient is haemodynamically stable with no serious signs and symptoms, obtaining a 12-lead electrocardiogram first may provide additional useful information regarding the aetiology of the wide complex tachycardias.

» Patients with monomorphic ventricular tachycardia may be managed with intravenous amiodarone 150 mg diluted in 100 mL of D5W, given slowly over 10–15 minutes and repeated once if the first dose is unsuccessful.

» Upon conversion to sinus rhythm, continue with an infusion at 1 mg/min over six hours followed by 0.5 mg/min over the next 18 hours, up to 900 mg/day.

» Alternatively, IV lignocaine at a dose of 1–1.5 mg/kg body weight may be administered at a rate of 10 mg/min and the dose repeated, if necessary, up to a maximum of 3 mg/kg.

» Upon successful conversion to sinus rhythm, a maintenance infusion should be given at a rate of 1–2 mg/min.

» It is reasonable to perform elective synchronised cardioversion if drug therapy fails. Transferring the patient to a monitored setting for this is preferred if the patient remains stable and time allows (as shown in figure 16).

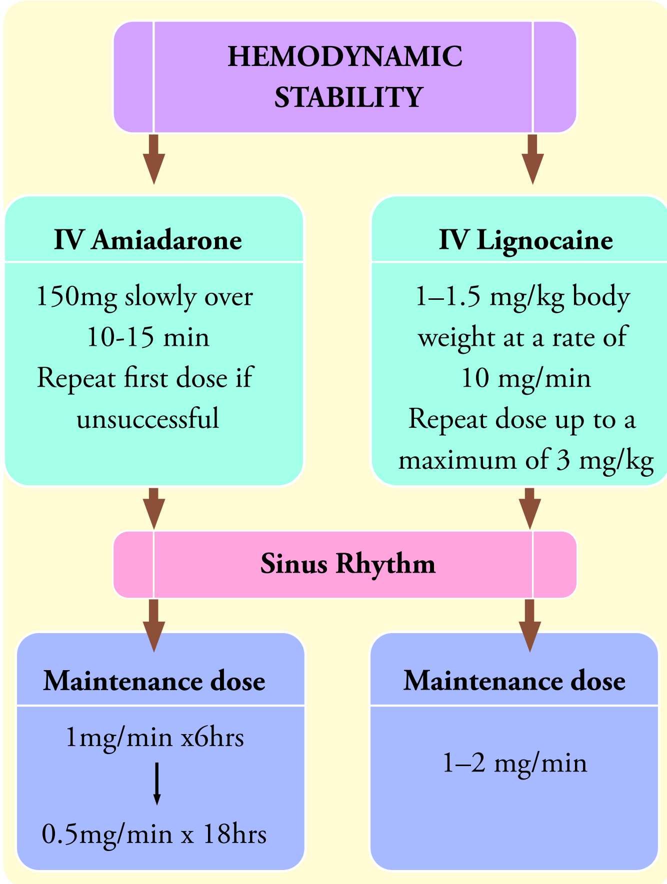

Figure 16: Cardioversion in stable WCTs

Polymorphic ventricular tachycardia is a shockable rhythm and patients presenting with it are often haemodynamically unstable. Synchronised cardioversion may not be successful, as the computer in the defibrillator may be unable to find the R-wave to synchronise the shock to. Defibrillation may be necessary. If the cause of the polymorphic ventricular tachycardia is a prolonged QT interval (torsades de pointes), intravenous magnesium sulphate 1–2 g over 15 minutes should be administered. In patients with polymorphic ventricular tachycardia and normal QT interval, intravenous amiodarone 150 mg over ten minutes may be given. [23, Rank 4]

Narrow Complex Tachycardias

The majority of regular narrow complex tachycardia (NCTs) are due to supra ventricular tachycardia and may also be due to atrio ventricular nodal re-entry or atrio ventricular re-entry tachycardia. Patients presenting with supra ventricular tachycardia usually have a sudden onset of symptoms. Diagnosis is usually made after a 12-lead electrocardiogram is performed. [24, Rank 3]

Unstable Patients

Patients with regular narrow complex tachycardia who have serious signs and symptoms of instability should be treated with urgent synchronised cardioversion. The procedures for synchronised cardioversion are as follows:

» Explain the procedure to the patient.

» Give analgesia and sedation carefully in titrated doses (beware that drugs used in procedural sedation may have negative effects on haemodynamics).

» Energy levels should begin at 50 J; evidence for different energy levels for monophasic or biphasic defibrillators is lacking.

» Ensure safety for defibrillation.

» If the shock is unsuccessful, the energy level may be escalated and repeated.

One may consider the use of intravenous adenosine 6 mg as a rapid bolus dose, followed by normal saline 20-mL flush for haemodynamically unstable patients with regular narrow complex tachycardia. [25, Rank 5]

Stable Patients

If vital signs are stable with no serious signs and symptoms, non-pharmacological cardioversion using vagal manoeuvres, such as carotid sinus massage or the modified Valsalva manoeuvre, may initially be attempted. [26, Rank 3]

Valsalva Manoeuvre

The Valsalva manoeuvre (as shown in figure 17) achieves vagal stimulation by first causing an increase in intrathoracic pressures during patient straining through forced expiration against resistance, followed by sudden reduced intrathoracic pressures when the patient releases the strain. It is during the second phase, when intrathoracic pressures fall back to normal, that there is a sudden return of venous blood back into the central circulation. A surge in cardiac output follows, which triggers the vagal response. The initial phase is performed with the patient sitting in a semi-recumbent position, followed by laying the patient supine and passively elevating the patient's legs once the strain is released. This augments the venous return to the heart and accentuates the surge in cardiac output, thus triggering a more effective vagal response.

When performing the procedure:

- Explain the procedure to the patient.
- Position the patient in the trolley with the head of the trolley raised to a 45° angle.
- Have the patient perform a forced expiration against resistance by blowing against the nozzle of a syringe or using a hand-held manometer for at least 15 seconds.
- Once the patient releases the strain, lower the head of the trolley to lay the patient flat and have an assistant raise the patient's legs to about 45°.
- Monitor the rhythm for conversion. [27, Rank 4]

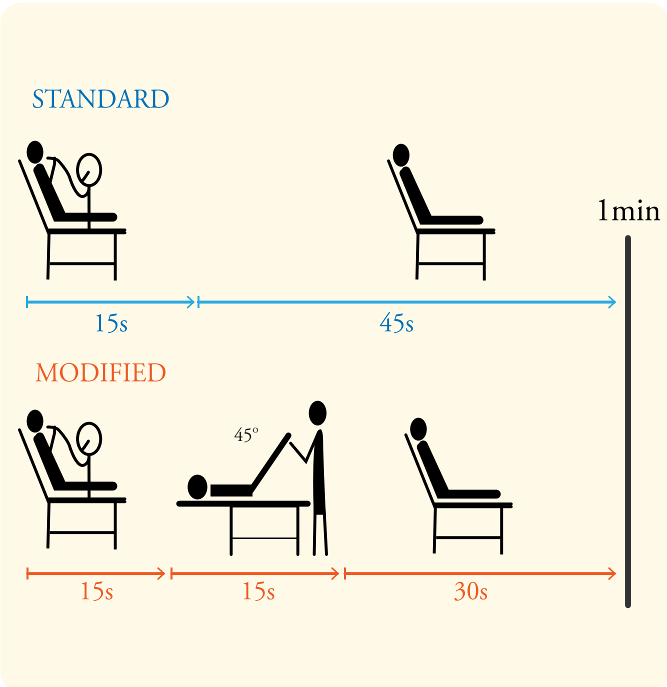

Figure 17: Valsalva Manoeuvre

Pharmacological Cardioversion

In the event of unsuccessful attempts with vagal manoeuvres, pharmacological agents (as shown in figure 18) may be employed to achieve rhythm control.

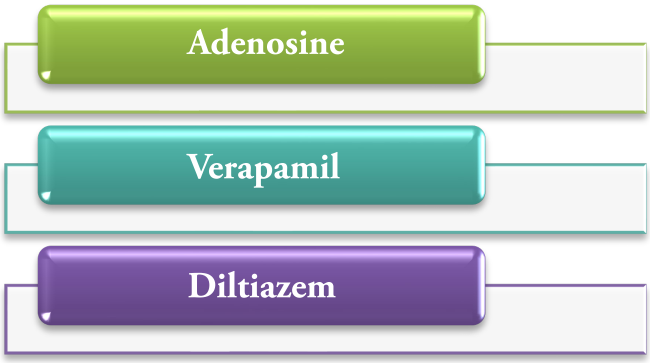

Figure 18: Drugs to achieve rhythm control

- Intravenous adenosine, verapamil and diltiazem are all acceptable drugs of first choice for the conversion of stable patients with supraventricular tachycardia.
- Adenosine for pharmacological cardioversion of supraventricular tachycardia is given as a rapid intravenous bolus of 6 mg via a proximal large calibre vein, followed immediately by a 20-mL saline flush using a three-way connector. This may be repeated at 12 mg if the initial bolus is unsuccessful.
- A simplified method of administration has been described, where the dose of adenosine is first mixed into the saline flush and the mixture administered as a single bolus. There is no difference in success rates, so this method may be considered an alter-

native for adenosine administration.

» Intravenous verapamil, a calcium channel blocker, may also be used for the treatment of supraventricular tachycardia. It is given as an infusion at a rate of 1 mg/min up to a maximum of 20 mg with haemodynamic monitoring at two-minute intervals. The infusion is stopped once sinus rhythm is achieved. In patients with atrial fibrillation (AF) with a fast ventricular rate, it may be used as a rate control agent when infused at the rate of 1 mg over three minutes up to a maximum of 20 mg, again with haemodynamic monitoring.

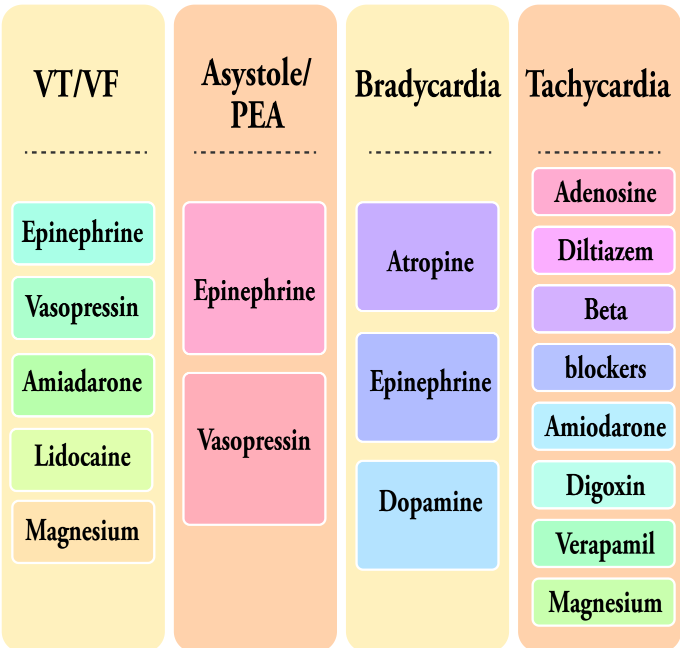

Figure 19: ACLS drugs

» Intravenous diltiazem is a calcium channel blocker. For patients with supraventricular tachycardia, it may be infused at a rate of 2.5 mg/min up to a maximum of 50 mg with haemodynamic monitoring at two-minute intervals. The infusion is stopped once conversion to sinus rhythm is achieved. In patients with atrial fibrillation with rapid ventricular response, it may be infused at the rate of 2.5 mg over three minutes up to a maximum dose of 50 mg, again with haemodynamic monitoring, to achieve controlled ventricular response. It should be avoided in the presence of heart failure owing to a possible negative ionotropic effect (as shown in figure 19).

Electrocardiogram and vital signs monitoring should be continued during the process and for at least two hours thereafter, for early detection of recurrence. [28, Rank 4]

Bradyarrhythmias

Haemodynamically significant brady arrhythmias usually present with a heart rate of below 60 bpm and a blood pressure of less than 90/60 mmHg. Most patients who are symptomatic would tend to have heart rates that are even lower than 50 bpm. Asymptomatic and haemodynamically stable patients generally do not need further treatment for the bradycardia. However, patients with Mobitz Type II second degree or complete heart blocks should be monitored closely even if stable, as they are at risk for sudden deterioration. Unstable patients should be managed in a monitored area and given supplemental oxygen or ventilatory assistance as appropriate. A 12-lead electrocardiogram or rhythm strip should be obtained to analyse the rhythm. [29, Rank 5]

The common drugs that may be used for brady arrhythmias are atropine, dopamine infusion and adrenaline infusion (as shown in figure 20).

» Atropine for bradycardia is given in repeated boluses of 0.6 mg up to a maximum vagolytic dose of 2.4 mg.

» Dopamine infusion is started at a rate of 5–20 mcg/kg/min and increased to a maximum of 20 mcg/kg/min, above which the likelihood of peripheral and splanchnic vasoconstriction may be significant and undesirable.

» Adrenaline infusions for bradycardia are initiated at a rate of 2–10 mcg/min and increased gradually till the target heart rate and/or blood pressure is achieved.

» Cardiac pacing would have to be considered for all patients with haemodynamically significant bradycardias.

In an emergency situation, transcutaneous pacing is the preferred method until transvenous pacing can be organised. Transcutaneous pacing is painful in conscious patients, requiring analgesia and sedation. [30, Rank 4]

Atropine	Dopamine	Adrenaline
Bolus of 0.6 mg up to a maximum vagolytic dose of 2.4 mg	**Started as infusions at a rate of 5 20 mcg/kg/min and increased to a maximum of 20 mcg/kg/min**	**2 10 mcg/min initially and increased gradually**

Figure 20: Drugs in bradyarrythmias

Conclusion

In out-of-hospital cardiac arrest patients, there are several factors independently associated with the initiation of advanced cardiac life support: age, suspected cause, location, duration of no-flow, initiation and duration of basic cardiac life support, use of automated external defibrillator and first recorded rhythm. Thus, emergency physicians seem to initiate advanced cardiac life support according to several criteria. They represent a combination of TOR rules validated in pre-hospital setting and prognostic factors described in resuscitated patients from out-of-hospital cardiac arrest. The TOR rules have been reported in several studies concerning basic life support as well as advanced cardiac life support. Both are considered conservative as only the presence of all criteria leads to stop resuscitation efforts. Emergency physicians in the field are not supposed to take into account the complete list of criteria to decide starting advanced cardiac life support. However, some parameters of the different advanced cardiac life support TOR rules were associated individually with the decision, such as absence of witness, absence of basic life support performed by a bystander and absence of defibrillation. [30, Rank 4]

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