

Cardiopulmonary resuscitation: state of the art in 2011

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Abstract

Failure to recognise the signs of sudden cardiac arrest or impending cardiac arrest will lead to delayed intervention. Cardiopulmonary resuscitation (CPR) must be initiated without delay, irrespective of the level of skill of the caregiver. The 2010 CPR guidelines emphasise the importance of chest compressions, which have now become the first step in the CPR sequence [compressions, airway, breathing or circulation, airway, breathing (CAB), instead of airway, breathing, compressions, or airway, breathing, circulation (ABC)]. Hands-only CPR should be encouraged where untrained caregivers are involved. Although ventilation may be an important step in some cases of arrest, e.g. primary asphyxia, excessive and inappropriate ventilation is detrimental. Rapid defibrillation is an essential life-saving step for specific arrest rhythms. Always consider reversible causes of cardiac arrest, and “find it, flag it, fix it and follow up!” Ideally, and especially during an in-hospital cardiac arrest, skilled providers should strive to work as a team. Team performance must be reviewed regularly to improve CPR efforts and ultimately outcome. Resuscitation and CPR during the perioperative period is unique, and therefore should be tailored individually to each specific clinical situation. A detailed knowledge of the most up-to-date resuscitation algorithms is essential. These are available from any resuscitation council.

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Introduction

The science of cardiopulmonary resuscitation (CPR) is dynamic and ever changing as new evidence continuously comes to light. Modern CPR dates back to 1966, when the first consensus statement was released.

The International Liaison Committee on Resuscitation (ILCOR) was founded in 1992. Its purpose is to review the knowledge and science of resuscitation and offer treatment recommendations. Various professional bodies from around the world make up its members. The American Heart Association (AHA) hosted the first ILCOR conference in 1999 to develop common resuscitation guidelines.

In February 2010, ILCOR, in collaboration with the AHA, coordinated the latest consensus conference. The proceedings of this meeting, the CoSTR 2010 documents (i.e. the 2010 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations), were published in October 2010 and provide the material for regional councils such as the AHA and European Resuscitation Council (ERC) to write their guidelines. The AHA basic life support (BLS)

and advanced cardiac life support (ACLS) courses have gained widespread national and international acceptance, and have set the standard for resuscitation care.

The Resuscitation Council of Southern Africa (RCSA) is a founding member of ILCOR, and has made significant contributions to the international consensus on CPR and emergency cardiovascular care. Its teaching is based on the AHA guidelines.

It is not the author's intention to restate these guidelines in detail, but rather to review the important points, and to put resuscitation into context from an anaesthesiologist's perspective. Paediatric and infant resuscitation will not be reviewed.

Principles of resuscitation

CPR is essentially a set of coordinated actions which aim to improve the chances of survival of a patient following cardiac arrest. The universal ILCOR cardiac arrest algorithm (see Figure 1) forms the basis for all the global resuscitation councils' algorithms.

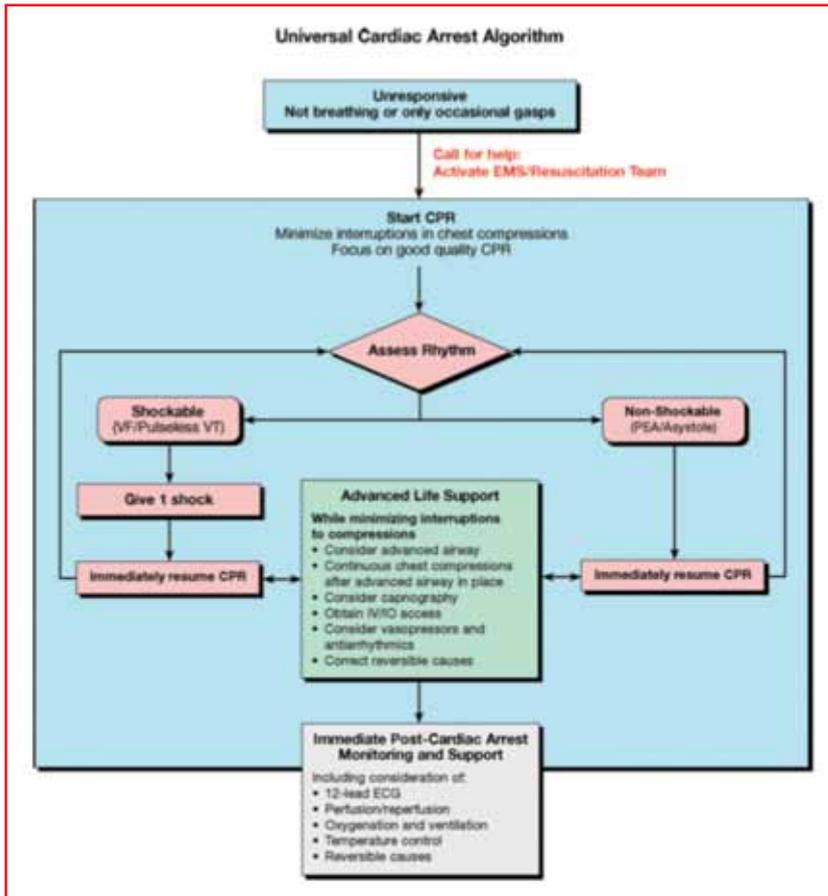


Figure 1: Universal International Liaison Committee on Resuscitation cardiac arrest algorithm

Regardless of the patient, aetiology of arrest, place of arrest or level of care, the fundamental principles of any resuscitative effort are the links of the so-called chain of survival:

- Immediate recognition of arrest and appropriate activation of support systems;
- CPR without delay;
- Appropriate electrical therapy, i.e. defibrillation;
- Instituting advanced care as soon as possible;
- Continued care after cardiac arrest.

The latter is an addition to the 2010 guidelines. Focused post-cardiac arrest care after return of spontaneous circulation (ROSC) has been shown to improve the outcome of patients who have suffered a cardiac arrest.

Initial steps include:

- Optimisation of cardiorespiratory function and re-establishment of vital organ perfusion.
- Consultation with and gaining of expert opinion.
- Transport to an appropriate level of care, i.e. intensive care unit, with continued or stepped-up monitoring.
- Active search for identifiable causes or precipitants. These must be corrected rapidly in order to prevent recurrent arrest.

Subsequently:

- Consider therapeutic hypothermia;
- Identify and treat acute coronary syndrome (ACS);
- Institute lung-protective ventilation;
- Be objective about the prognosis and termination of resuscitative efforts;
- Provide counselling for all parties involved;
- Assist survivors with rehabilitation.

Defining cardiac arrest

The heterogeneity of sudden cardiac arrest (SCA) implies that no single approach to CPR is possible or practical, yet all resuscitation efforts must be based on the chain of survival outlined above. If these are applied effectively, survival can approach 50%. Unfortunately true figures vary greatly, and have been found to be between 5-50%.

Broadly speaking, SCA can be classified according to aetiology (cardiac vs. non-cardiac), circumstances (witnessed vs. unwitnessed), and setting (out of hospital vs. in hospital). Recognition of SCA is not always easy, especially for inexperienced healthcare providers (HCPs).

The 2010 CPR guidelines base the recognition of SCA on assessing unresponsiveness and absence of normal breathing, which implies no breathing, or gasping breaths. “Look, listen and feel” has now been removed from the basic life support (BLS) algorithm.

In the perioperative setting, from induction of anaesthesia to discharge from the care of anaesthesia personnel, cardiac arrest is always a witnessed event with various possible aetiologies (Table I). A number of variables influence the

Table I: Perioperative cardiac arrest

Predictor of perioperative cardiac arrest	p-value (n = 518 294)
American Society of Anesthesiologists score	0.001
End-organ failure	0.020
Diabetes mellitus	0.030
Type of surgery (high vs. low risk)	0.010
Emergency surgery	0.001
Duration of surgery	0.030
Protracted intraoperative hypotension	0.001
Cause of sudden cardiac arrest (haemorrhage vs. all others)	0.001
Time of arrest (working hours vs. after hours)	0.001
Primary electrocardiogram rhythm	0.020

overall incidence of perioperative cardiac arrest. The reported incidence ranges between 4.6-19.7 per 10 000 general anaesthetics.

It is generally accepted that the incidence of SCA is lower during neuraxial anaesthesia (1.8 per 10 000), with more arrests occurring during spinal than epidural anaesthesia (2.9 vs. 0.9 per 10 000 respectively). However, it is important to note that the incidence of SCA directly attributable to anaesthesia is different from the figures quoted thus far. Anaesthesia-related cardiac arrest ranges between 0.5-1 per 10 000 cases. Mortality related to these is approximately 1 per 100 000 anaesthetics.

Cardiac arrest that is attributable to anaesthesia falls into one of two categories: medication related and airway or ventilation related. The single largest aetiological category is associated with the intraoperative use of neuromuscular blocking agents, at emergence or in the recovery room.

The recognition of SCA during the perioperative period can obviously not be based on the 2010 BLS definition, as many patients may be relatively unresponsive and may exhibit abnormal breathing, purely because of "normal" perioperative circumstances.

Patients can typically deteriorate to pulseless arrest very rapidly, under circumstances wholly dissimilar to all other SCA scenarios. Patients in the perioperative period present with a variety of pathologies, making each resuscitation unique. Hypovolaemia is an exceedingly common contributor to SCA. Unlike other SCA scenarios, the most common dysrhythmias that are encountered are bradycardia and asystole. Fortunately, the outcomes are less dismal during the perioperative period. This is because a direct aetiology can usually be rapidly identified and corrected.

During the perioperative period, anaesthesiologists are primarily tasked with supporting homeostasis. By simply applying basic medical science, SCA can be avoided in most cases.

Cardiac arrest, during the perioperative period, is relatively rare, and unfortunately it is difficult to exactly apply current CPR guidelines to this unique period in patient care. Fortunately, modern monitoring gives us a unique advantage in identifying cardiac arrest, and thereby guides our interventions.

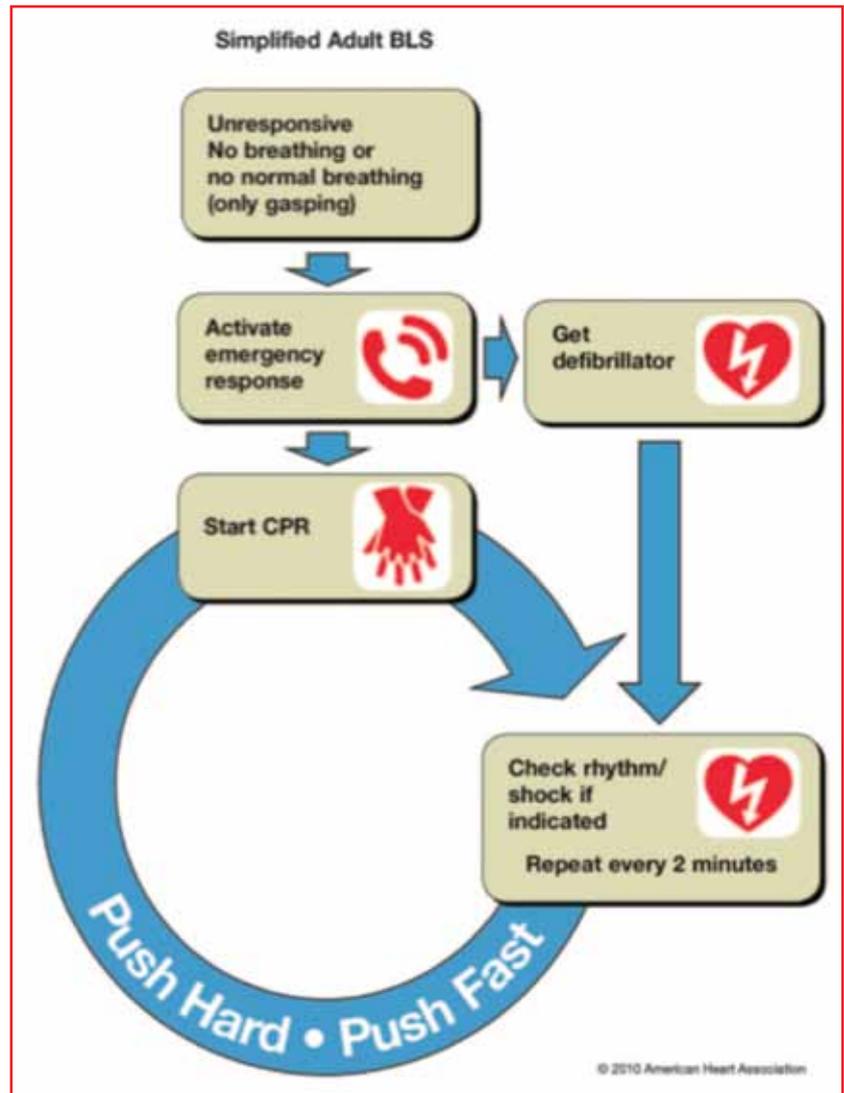


Figure 2: American Health Association basic life support algorithm

The fact that all cardiac arrests attributable to anaesthesia are related to airway management and medication administration is important in prevention strategies.

Adult basic life support

BLS skills lay the foundation for CPR (Figure 2 and Table II), and play an important role in determining ROSC and ultimately survival. There is simply no substitute for well-performed BLS. There is a distinction between BLS for the layperson and the HCP.

Recognition of arrest

The first step in any resuscitation scenario is to timeously recognise cardiac arrest (see Table III) and to activate some form of emergency response system. For out-of-hospital arrest, this would imply ensuring the safety of the scene, and contacting the appropriate emergency services.

Table II: Summary of basic life support for the healthcare provider

Component	Adults	Children	Infants
Recognition	Unresponsive		
	No breathing, not breathing normally, e.g. gasping	No breathing or only gasping	
	No pulse palpated within 10 seconds (^a HCP only)		
CPR sequence	^b CAB	CAB	CAB
Circulation			
Compression rate	At least 100 per minute		
Compression depth	At least 5 cm	At least one-third antero-posterior depth; ± 5 cm	At least one-third antero-posterior depth; ± 4 cm
Chest wall recoil	Allow complete recoil between compressions		
Compression interruptions	HCPs rotate compressors every two minutes Minimise interruptions in chest compressions Attempt to limit interruptions to < 10 seconds		
Airway	Head-tilt/chin-lift (HCP suspected trauma: jaw thrust)		
Breathing			
Compression:ventilation ratio	30:2 (one or two rescuers)	Single rescuer 30:2 Two rescuers 15:2	Single rescuer 30:2 Two rescuers 15:2
Ventilations if rescuer untrained or trained and not proficient	Compressions only		
Ventilations if rescuer advanced airway provider (HCP)	One breath every six to eight seconds (0-8 breaths per minute) Asynchronous with chest compressions About one per breath Visible chest rise		
Defibrillation	Attach and use ^c AED as soon as available Minimise interruptions in chest compression before and after shock Resume CPR beginning with compressions immediately after each shock		

a = healthcare provider, b = circulation, airway, breathing, c = automated external defibrillator

During in-hospital arrest or perioperative arrest, this will imply getting support from the available staff, or activating a resuscitation team, if such an entity is available.

Delays in defibrillation occur in one out of seven cases of cardiac arrest in the perioperative arena. This has been associated with lower rates of survival in the periprocedural period, but not with lower survival rates for cardiac arrest in the operating room. The immediate availability of a defibrillator is absolutely essential in any healthcare environment, especially in theatre.

Table III: Diagnosis of cardiac arrest

Witnessed arrest: Unresponsive patient with no, or lack of, normal breathing. During sudden cardiac arrest, occasional gasps may be incorrectly interpreted as breathing efforts. These guidelines are not applicable during general anaesthesia. Cardiopulmonary resuscitation should also be immediately commenced in situations where cardiac output has critically decreased.
Dilated pupils. Usually within a minute after sudden cardiac arrest. Various drugs that are used in anaesthesia may make this sign obsolete.
Central cyanosis.
No bleeding from surgical wounds.
Profound hypotension.
Sudden decrease in end tidal CO ₂ .
Appropriate electrocardiogram trace, e.g. ventricular fibrillation.

These life-saving devices must be maintained and tested regularly, and a thorough knowledge of their operation is of cardinal importance. Figure 3 shows how rapid defibrillation and CPR affect coronary perfusion pressure.

The 2010 guidelines have de-emphasised checking for breathing and pulse. Studies have shown repeatedly that lay persons and HCPs alike often have difficulty in doing this. The motivation behind this is to minimise any delays in starting chest compressions. If a pulse check is performed

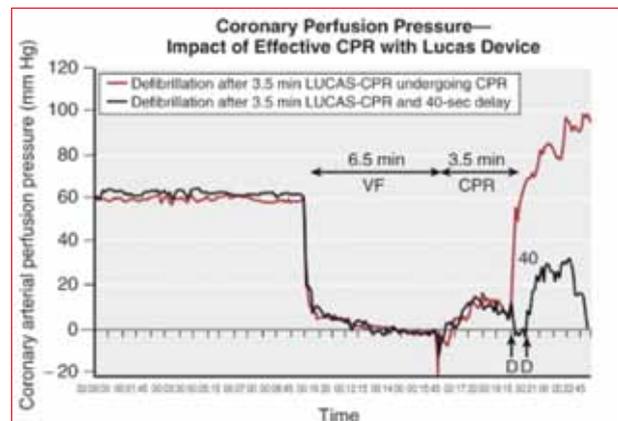


Figure 3: Lund University Cardiac Arrest System (LUCAS)

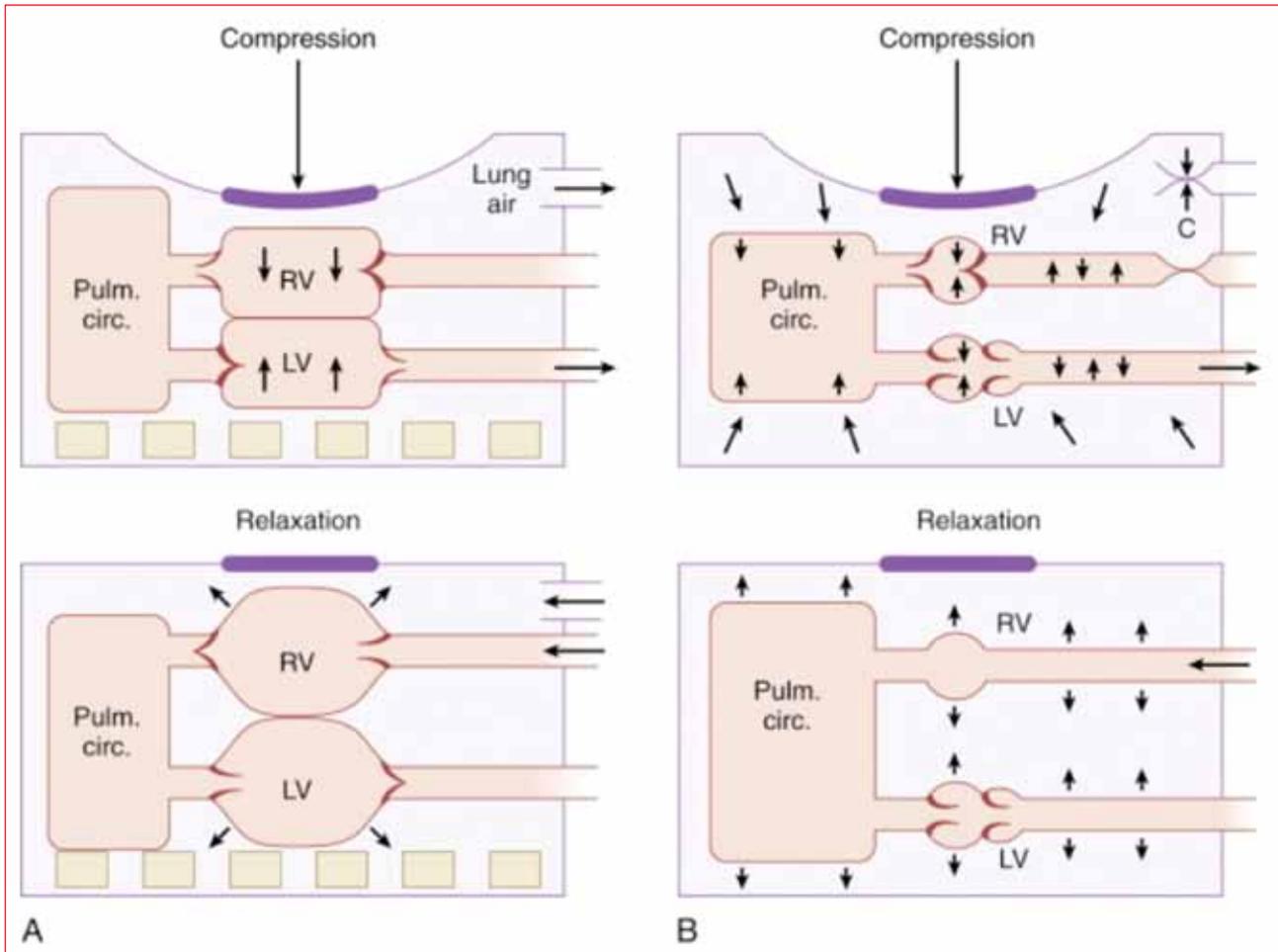


Figure 4: Cardiac compressions (A) and the intrathoracic pressure pump (B)

by an HCP, it should take no more than 10 seconds. There is also no evidence that suggests that checking for breathing, coughing or movement is superior when checking for adequate circulation.

Chest compressions

Effective chest compression is crucial. Whenever possible, the victim should be placed on a firm surface, in the supine position. Backboards are useful, but finding one should not delay CPR, and care must be taken to not displace lines and tubes. Compressions are applied over the lower part of the sternum to a depth of 5 cm. A compression rate of at least 100 per minute is ideal. Fatigue can contribute significantly to inadequate compressions, and it is reasonable to switch chest compressors approximately every two minutes. This should be done during an intervention, e.g. defibrillator application, and the changeover should be limited to less than five seconds.

Figure 4 serves to clarify the mechanism of cardiac compressions (A) and the intrathoracic pressure pump (B). These aid in systemic perfusion and cardiac filling respectively.

The cardiac pump mechanism, in which the heart is compressed between the sternum and the spine, results in the ejection of blood into the systemic circulation. The increase in intrathoracic pressure forces blood out of the thorax.

Cardiac output is markedly decreased during CPR, and at most, is 30% of normal. The majority of the blood flow is directed towards the supradiaphragmatic organs, of which the heart and brain are the most important. Vasopressors improve the flow to these vital organs, but may reduce perfusion of other organs.

Measures of adequate CPR include:

- A sustained diastolic pressure of at least 40 mmHg;
- Coronary perfusion pressure of 20 mmHg;
- An end-expiratory CO₂ of at least 10-20 mmHg.

A sustained end-tidal CO₂ of 10 mmHg or less has an extremely poor outcome, and is most unlikely to result in successful resuscitation.

Compressions take preference over ventilation for the following reasons:

- Lungs usually contain enough oxygen to prevent serious hypoxia. The functional residual capacity consists of approximately 480 ml when breathing air (FiO₂ = 20 %).

- Neurons are more resistant to hypoxia than ischaemia.
- It is usually easier to commence compressions. Hence the emphasis in the current guidelines on “hands-only CPR”. This is especially true for layperson rescuers.
- Compressions may cause spontaneous defibrillation, if initiated before the myocardium becomes hypoxic.

There is no ideal evidence-based compression-to-ventilation ratio. Currently a ratio of 30 compressions to two ventilations is recommended. Once an advanced airway is in place, there is no need to synchronise these.

Airway management and ventilation

In the 2010 CPR guidelines, the old airway, breathing, circulation (ABC) mindset has been replaced with the circulation, airway, breathing (CAB) CPR sequence. This represents a significant change, and reflects growing evidence of the importance of chest compressions. It also serves as a reminder that all airway interventions should be performed swiftly, and should not delay chest compressions.

The simple head-tilt or chin-lift manoeuvre will improve ventilation in most patients. This should only be done in patients with no evidence of head and neck trauma. A jaw thrust, without head extension, is recommended in such cases.

The primary goal of assisted ventilation during CPR is oxygenation and not CO₂ elimination. All rescue breaths must achieve a tidal volume sufficient to visibly raise the chest. This should also be consistent with lung-protective ventilation, by not exceeding a tidal volume of greater than 6-7 ml/kg. Poor lung compliance may require that the pressure relief valve be bypassed, but care should still be taken not to overinflate the lungs.

Excessive ventilation will increase intrathoracic pressure, decrease venous return, and ultimately decrease effective cardiac output. This is important, because even during effective chest compressions, the cardiac output is only about 25-30% of normal.

Ventilation with airway devices

All anaesthetic providers must master the skill of bag-mask ventilation. It can be challenging and requires

Table IV: Characteristics of bag-mask-valve devices

Bag-mask device	Mask
Non-jam inlet valve	Made from transparent material
Pressure relief valve with bypass	Creates a tight seal
Reservoir (high concentration of oxygen)	Fits over mouth and nose
Non-rebreathing outlet valve	Oxygen inlet for insufflation
Standard 15/22 mm fittings	15/22 mm connectors
Adult, paediatric and neonatal sizes	One adult and several paediatric sizes

considerable practice in order to gain competence. Its use is not recommended during lone rescuer CPR, and ideally requires two operators. Table IV lists the characteristics of bag-mask-valve devices.

Bags are commonly one or two litres in volume, and only require one- or two-thirds compression to provide an adequate tidal volume. Remember, only a visible chest rise is required. Each breath is to be administered over one second, and time for complete expiration is necessary. The ratio for ventilations is 2:30 compressions. As soon as an advanced airway device is in place, there is no need to synchronise compressions and ventilations. One breath every seven seconds is adequate.

Endotracheal intubation is not part of the resuscitation guidelines, but may be performed provided it does not delay the CPR cycle. Supraglottic airway devices are relatively easy to use, and do not necessarily require the same level of skill as the placement of an endotracheal tube. The laryngeal mask airway and Combitube® are examples of such devices. With any of these devices in place, compression must be done at a rate of at least 100 per minute, without interruptions.

The standard use of cricoid pressure during placement of airway devices or ventilation is no longer recommended in the 2010 guidelines (Table V).

Table V: The adverse effects of cricoid pressure

Interference with placement of airway devices
Distortion of anatomy in the line of vision
Simulates a swallow reflex, and lower tension of the oesophageal sphincter
Makes intubation difficult
Rupture of oesophagus
Pressure on other structures, e.g. carotid artery
Contraindicated in neck trauma (bimanual cricoid should be done)

Electrical therapy

A delay in the commencement of CPR or defibrillation reduces survival after SCA. Ventricular fibrillation (VF) is the most common initial dysrhythmia in out-of-hospital cardiac arrest, and early defibrillation is the only treatment for it. Mortality increases by 10% for every minute that passes. Immediate CPR prolongs VF, delays the onset of asystole, and extends the window for successful defibrillation.

There is insufficient evidence to either support or refute the application of CPR before defibrillation. However, in monitored patients, as is the case during the perioperative period, the time taken from VF to defibrillation should be under three minutes (see Table VI). A period of immediate CPR before defibrillation may be beneficial after prolonged collapse.

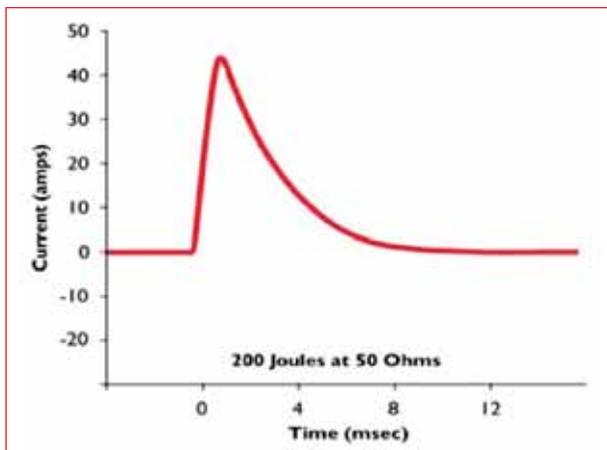
Table VI: Importance of early defibrillation

Majority of sudden cardiac arrest victims have ventricular fibrillation
Ventricular fibrillation is successfully treated with defibrillation
Delayed defibrillation reduces survival
Ventricular fibrillation is followed by asystole (poor outcome)

Defibrillation shocks are typically characterised by the amount of energy applied, e.g. 200 J. Alternatively, they may be described in terms of discharge waveform, i.e. monophasic or biphasic.

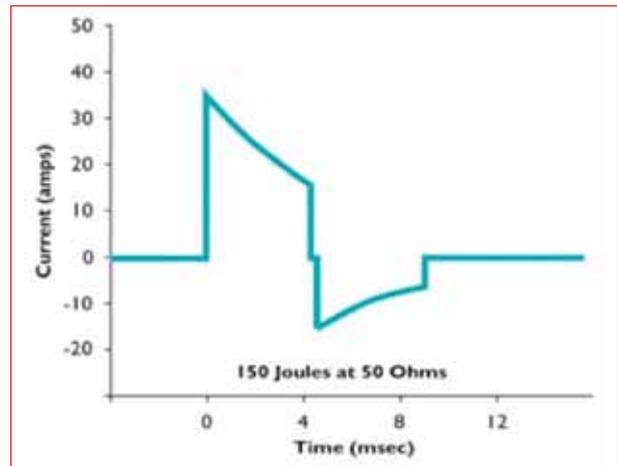
Defibrillation waveforms are generated by the discharge of energy (stored in a capacitor) through a patient. The value of the capacitor (charge), discharge path, and elements of that path determine the shape of the waveform. Waveform shapes are used to classify defibrillation technology. Waveforms describe the delivery of energy, or current, as a factor of time. It is worth noting that, while we may choose an energy setting in joule, it is actually the resulting current (ampere) that defibrillates or cardioverts.

Using energies of up to 360 J, monophasic defibrillators can deliver upwards of 60 A. A body of accumulated data shows that substantial post-shock myocardial dysfunction results from high-energy defibrillation. Figure 5 depicts a typical monophasic waveform.

**Figure 5:** A typical monophasic waveform

With biphasic truncated exponential (BTE) waveforms, the energy delivery occurs in two phases. The first phase, seen as the positive waveform deflection, is indicative of current flow from the sternal to the apical paddles. The second phase, as depicted by the negative deflection, indicates a reverse direction of current flow. Offering the promise of smaller size and longer battery life, the BTE waveform was originally developed for the implantable cardiac defibrillator, where impedance variations are a virtual non-factor.

BTE waveforms generally match the clinical performance of monophasic technology at lower energy levels. Figure 6 depicts a typical BTE waveform.

**Figure 6:** A typical BTE waveform

The rectilinear biphasic waveform was specifically developed for external defibrillation. It sought to improve upon the earlier biphasics by eliminating high peak currents and stabilising its shape in the face of varying patient impedance levels. The rectilinear biphasic waveform is characterised by two distinct attributes.

The first attribute is an initial phase that reduces peak currents by delivering a constant current essentially. This is achieved by controlling the total impedance of a defibrillation circuit, i.e. the impedance of the patient and of the defibrillator. Where patient impedance is high, a series of digitally controlled resistors lowers the defibrillator resistance to maintain the constant current flow. Conversely, when it is low, the defibrillator resistance is raised.

The second attribute is the fixed duration of each of the phases. Regardless of patient impedance, the first and second phases are always six and four milliseconds, respectively. Fixed waveforms yield more consistent performance. Figure 7 depicts a typical rectilinear biphasic waveform.

It is the average current that defibrillates. Studies have found that high-peak currents result in myocardial

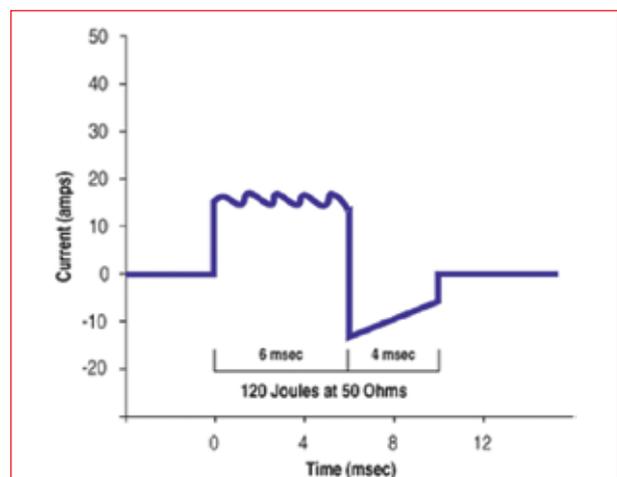
**Figure 7:** A typical rectilinear biphasic waveform

Table VII: Dysrhythmias and recommended electrical therapies

Dysrhythmia	Electrical therapy
Ventricular fibrillation	Defibrillate with 150 J ^a BP, or 360 J ^b MP
Pulseless ventricular tachycardia	Defibrillate with 150 J BP, or 360 J MP
Monomorphic ventricular tachycardia with pulse	Cardioversion 100 J
^c Unstable regular and narrow QRS tachycardia	Cardioversion 50 J
^c Unstable irregular and narrow QRS tachycardia, e.g. atrial fibrillation	Cardioversion 120 J
Paediatric	^d 2 J/kg, max of 10 J/kg has been used
Open chest (internal paddles)	50 J

a = biphasic (when using biphasic devices, consult the manufacturer for recommendation. Usually, initial biphasic doses of 100-120 J are used, and increased as needed)

b = monophasic

c = poor or deteriorating haemodynamics, e.g. hypotension, syncope, loss of consciousness and chest pain

d = the actual upper safe limit for defibrillation in children is not known, and doses as high as 10 J/kg have been used

dysfunction. While the BTE waveform has a lower peak current than the monophasic waveform, the peak current for a rectilinear biphasic waveform is 65% lower than that of the monophasic technology. Modern defibrillators deliver a current based on the stored energy. There is some evidence that current-based defibrillation may be superior to energy-based defibrillation.

Table VII provides a summary of the electrical therapies recommended for different dysrhythmias.

Since 1995, the AHA has recommended the development of automated external defibrillator (AED) programmes especially for lay rescuers. These aim to minimise the delay in “time to first shock”.

AEDs are reliable computerised devices that use voice and visual prompts to guide safe defibrillation. These devices are commercially available, and can markedly improve CPR success. Some data seem to indicate that the use of AEDs during in-hospital SCA could improve survival. Table VIII summarises the operational steps required for successful automated external defibrillation.

Table VIII: Operational steps: automated external defibrillator

Activate: Power on the automated external defibrillator. This activates the voice prompts for guidance in all subsequent steps. Some devices may power up automatically when the lid of the case is opened.
Attach: Choose the correct adhesive pads, and place these on the patient's bare chest. Use paediatric pads or system for patients less than eight years of age, if available.
Analyse: Always remain clear of the patient during analysis. Some devices will automatically analyse the rhythm, while other may require you to push a button.
Administer: The automated external defibrillator will advise whether or not a shock is required. Clear the patient. Only then, press the shock button.

For attachment of the defibrillator pads (Figure 8) or application of the paddles, the following positions may be used:

- Anterolateral (default) ;
- Anteroposterior;
- Anterior-left/right infrascapular.

**Figure 8:** Self-adhesive defibrillation pads: paediatric, left; adult, right

To reduce transthoracic impedance, defibrillation paste or gel must be used with handheld paddles. Water-based lubricants (such as K-Y jelly) or saline are not recommended.

SCA is not common in children, and the aetiologies are notably diverse and frequently preventable. VF is also uncommon in children (5-15%). As in adults, rapid defibrillation still markedly improves survival. The actual upper safe limit for defibrillation in children is not known, and doses as high as 10 J/kg have been used.

For children between one and eight years of age, the use of an AED is recommended, with the paediatric dose attenuator system. Manual defibrillation is recommended for infants (younger than one year).

There have been case reports of fires ignited by sparks from incorrectly used defibrillators. One of the common reasons is defibrillation in the presence of oxygen-enriched air. Oxygen tubing must be removed from the patient during defibrillation. (“I’m clear, you’re clear, all clear, oxygen clear!”). The use of self-adhesive pads and ensuring good chest wall contact can further minimise spark ignition during defibrillation. When manual pads are used, gel pads

are preferable to electrode pastes and gels, because they may spread between the paddles, creating the potential for a spark.

Synchronised cardioversion

Synchronised cardioversion refers to a shock delivered on the R wave of a QRS complex, with the aim of converting an abnormal rhythm back to a sinus rhythm. This synchronisation avoids shock delivery during the relative refractory period of the cardiac cycle, when a shock could produce VF. It is also the reason why the precordial thump is no longer recommended.

Table IX describes the steps that must be followed for the correct administration of synchronised cardioversion.

Figure 9 shows where synchronised cardioversion fits into the AHA tachycardia algorithm.

Transcutaneous pacing

Transcutaneous pacing (TCP) is the treatment of choice when a patient has a symptomatic bradycardia with signs of poor perfusion. Many defibrillator manufacturers now include a pacing mode in manual devices. Practitioners must be familiar with the indications, techniques and hazards of TCP (Tables X and XI).

Table IX: Stepwise synchronised cardioversion

Sedation of patient when appropriate. If unstable or rapidly deteriorating, proceed immediately with cardioversion.
Turn on the defibrillator and attach the monitor leads. Ensure that a proper rhythm is displayed. Position the pads or paddles.
Have an assistant engage the sync function of the defibrillator. Look for marks on the QRS complex. It may be necessary to adjust the gain.
Select the appropriate starting energy level (escalate as needed) ^a Atrial fibrillation: 120 J ^a Ventricular tachycardia (with pulse): 100 J ^a Supraventricular tachycardia and regular atrial flutter: 50 J
Clear the patient. Charge the defibrillator.
Deliver the shock by holding down the discharge buttons on the manual pads, or by pressing the button on the defibrillator.
Check the monitor. If the tachycardia persists, increase the energy level.
Activate the sync mode after delivery of each synchronised shock. Most defibrillators default back to the unsynchronised mode after delivery of a synchronised shock. This default allows an immediate shock in cardioversion, which produces ventricular fibrillation.

a = When using biphasic devices, one should consult the manufacturer for specific recommendation. Initial biphasic doses of 100-120 J are usually used, and escalated as needed.

Figure 10 shows where TCP fits into the AHA bradycardia algorithm.

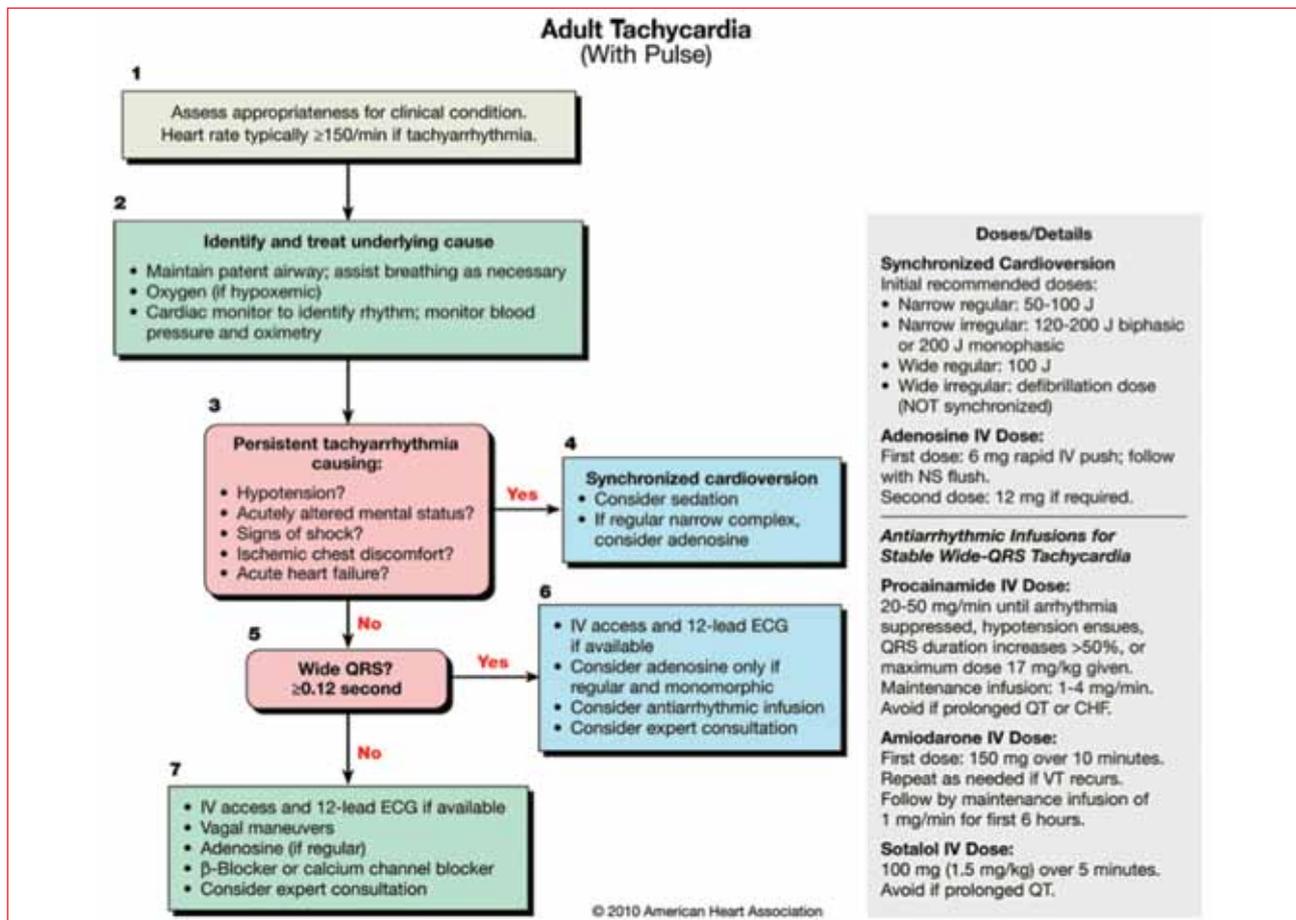


Figure 9: American Heart Association tachycardia algorithm

Table X: Indications for transcutaneous pacing

Haemodynamically unstable bradycardia
In the setting of acute coronary syndrome: <ul style="list-style-type: none"> • Symptomatic sinus bradycardia • Mobitz type II second-degree ^aAV block • Third-degree AV block • New or alternating bundle branch block or bifascicular block
Bradycardia with symptomatic ventricular escape
Overdrive pacing of refractory tachycardias
Precautions with transcutaneous pacing: <ul style="list-style-type: none"> • Contraindicated in hypothermia and asystole • Sedation and analgesia may be indicated • Carotid pulse is unreliable to confirm mechanical capture

a = atrioventricular

Table XI: Stepwise transcutaneous pacing

Place pacing electrodes as per package instructions.
Switch to pacing mode.
Set demand rate to 60/minute. External pacemakers have either fixed rates (asynchronous pacing) or demand rates. Start or switch on the pacer.
In a stepwise manner, increase the current output, and set it at 10 % above the dose at which consistent electrical capture is observed.
Confirm mechanical capture by detecting a reliable peripheral pulse or by pulse oximetry.

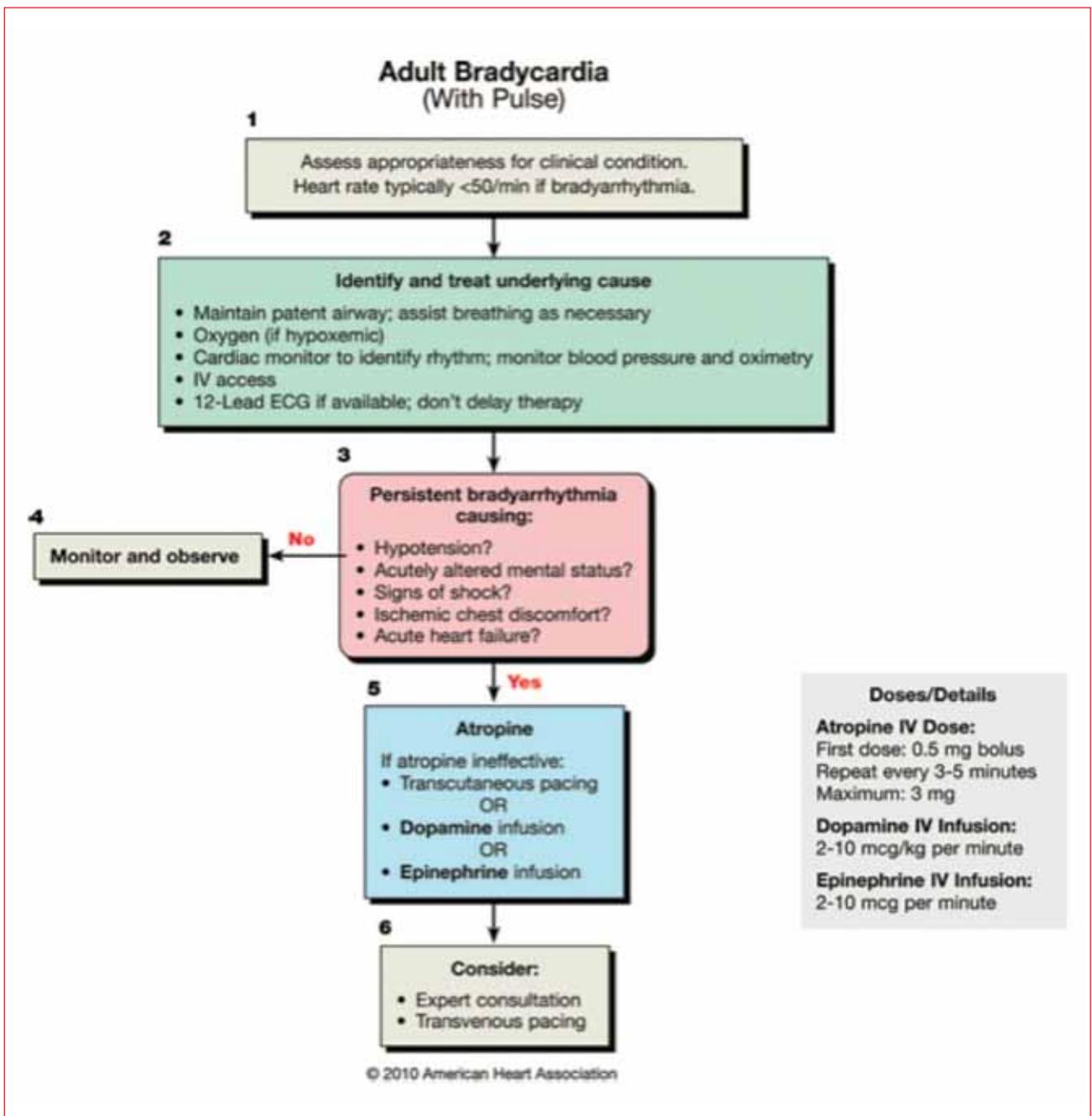


Figure 10: American Heart Association bradycardia algorithm

Perioperative cardiac arrest: an anaesthesiologist's perspective

Cardiac arrest during anaesthesia is a rare event (see Table XII), but is distinct from SCA in other settings. It is usually witnessed and often anticipated. The response to an arrest is also more timeous and focused. Outcome is fortunately markedly better, which can be attributed to detailed knowledge about the patient as well as the vast number of resources available in the theatre environment, which can be rapidly called upon.

Table XII: Procedure to be followed for cardiac arrest during anaesthesia

Inform the surgical team. If possible, all surgery should be stopped.
Place the theatre table in the Trendelenburg position.
Stop the administration of all anaesthetic drugs.
Ventilate with 100% oxygen.
Initiate basic life support or advanced cardiac life support resuscitation.
Seek reversible causes of cardiac arrest.

Figure 11 illustrates the ERC in-hospital resuscitation algorithm, and Figure 12 the new circular AHA ACLS algorithm.

Escalating care

When increased support is required to maintain cardiorespiratory function, serious consideration should be given to escalating the level of monitoring in parallel. Timeous insertion of an arterial line and central venous catheter will assist in the evaluation of decompensating patients in the perioperative period. However, the insertion of these monitors should not take precedence over supportive measures.

Pre-arrest (rescue)

Practitioners caring for patients undergoing surgery need to have a very clear comprehension of how to identify a patient

Table XIII: The mechanisms of hypoxaemia

Low alveolar oxygen tension	Increased alveolar-arterial gradient
Low inspired oxygen	Right-to-left shunt
Alveolar hypoventilation	^a V/Q mismatch
Diffusion hypoxia	Low mixed venous oxygen tension: decreased cardiac output, increased oxygen consumption, decreased haemoglobin concentration
Increased oxygen consumption	Diffusion defect

a = ventilation-perfusion

in a crisis and need to act promptly. Failure to “rescue” is an all too common “cause” of perioperative cardiac arrest.

SCA in the perioperative period typically occurs as a consequence of either hypoxaemia (Table XIII), or the progression of a circulatory process.

A frequently overlooked factor that contributes to perioperative circulatory compromise (which can very easily exacerbate the condition of a patient in crisis) is overzealous ventilation. This is particularly true for patients suffering from obstructive lung disease. In the latter, the development of auto-positive end-expiratory pressure (auto-PEEP) can literally choke the circulation. The detection and reduction of auto-PEEP leads to rapid improvement, and should be among the first assessments performed in susceptible patients with an unstable circulation.

Ventilatory support should aim to effectively support adequate oxygenation, and practitioners must strive to adhere to the principles of lung-protective ventilation. Simply put, the smallest tidal volumes and lowest rates must be used.

Cardiopulmonary interaction and circulatory management should be approached systematically by addressing the components of cardiac output (stroke volume). (See Table XIV).

Table XIV: The components of cardiac output (stroke volume)

Preload: Optimal cardiac filling, according to the Frank-Starling mechanism
Contractility: Avoiding and correcting factors impeding inotropism
Dysrhythmias
Afterload: Laplace's Law, systemic vascular resistance, pulmonary vascular resistance

Certainly the most common cause of hypotension in the perioperative period is hypovolaemia. Unfortunately, the question: “How much volume is too much?” remains unanswered. It seems reasonable for practitioners to volume resuscitate as long as there is evidence that the patient will respond, and providing there is no hydrostatic pulmonary oedema present.

A reliable indicator of hypovolaemia is pulse pressure variation (PPV), provided that ventilation is with tidal volumes less than 8 ml/kg, and also that the overall respiratory compliance is normal.

The following formula is used to calculate pulse pressure variation:

$$PPV = 100 \times [(PP_{Max} - PP_{Min}) \div (PP_{Max} + PP_{Min}) \div 2]$$

Figure 13 illustrates an approach to volume resuscitation.

The next step in supporting circulatory collapse would be to address contractility or “pump failure”. The management of left and right ventricular failure (LVF and RVF) is quite

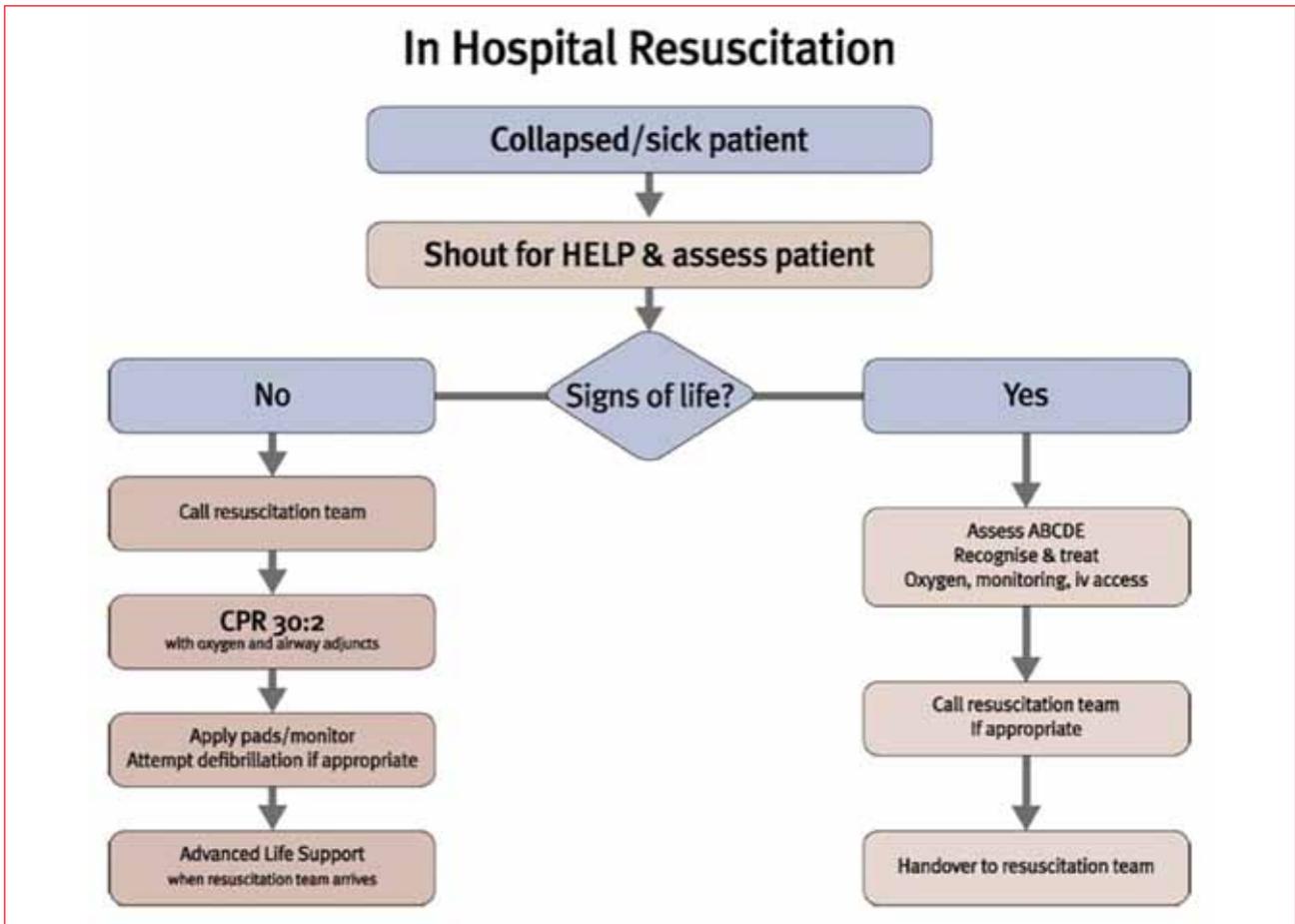


Figure 11: European Resuscitation Council in-hospital resuscitation algorithm

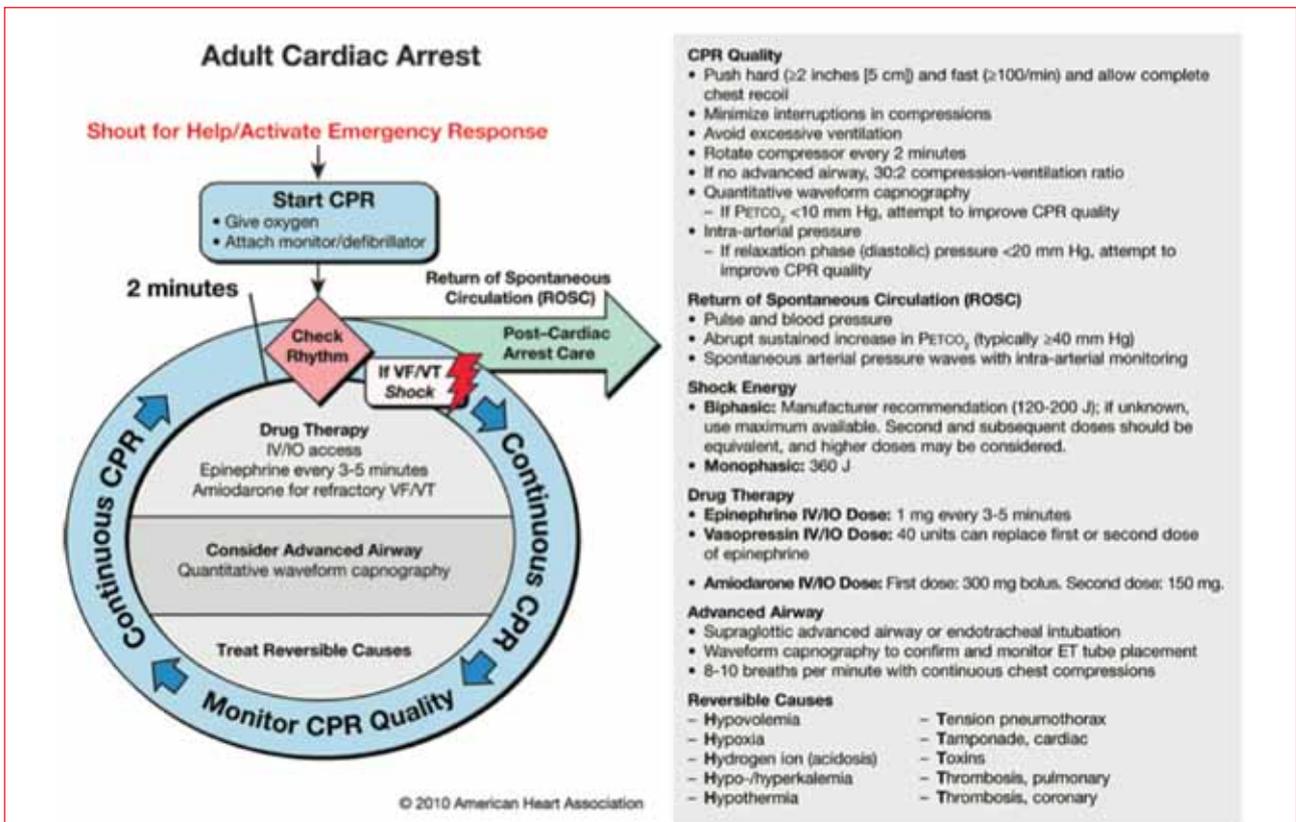


Figure 12: American Heart Association ACLS algorithm

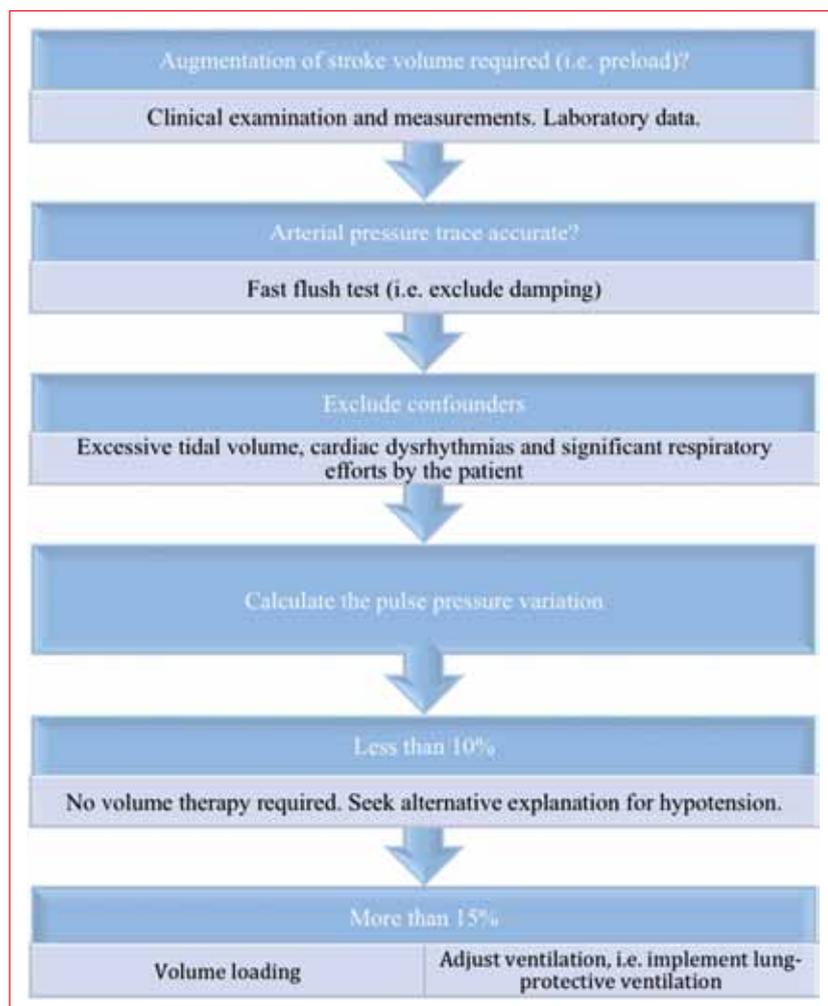


Figure 13: An approach to volume resuscitation

different. However, both require adequate volume to promote filling and adequate output. In short, LVF can be managed optimally by afterload reduction and inotropic support. RVF readily responds to a combination of pulmonary vasodilators and positive inotropes.

Common perioperative crises

- Attributable directly to anaesthesia:
 - Intravenous anaesthetic overdose;
 - Inhalation anaesthetic overdose;
 - High neuraxial block;
 - Drug administration errors;
 - Pharmacogenetic factors, e.g. malignant hyperthermia.
- Respiratory:
 - Hypoxaemia;
 - Auto-PEEP;
 - Acute bronchospasm.
- Cardiovascular:
 - Hypovolaemia;
 - Vasovagal reflex;
 - Tension pneumothorax;

- Anaphylactic reaction;
- Transfusion reaction;
- Electrolyte imbalance (particularly potassium);
- Severe pulmonary hypertension;
- Increased intra-abdominal pressure;
- Pacemaker failure;
- Prolonged QT syndrome;
- ACS;
- Pulmonary embolism;
- Gas embolism;
- Oculocardiac reflex;
- Electroconvulsive therapy.

Post-cardiac arrest care

A new addition to the 2010 AHA guidelines is an algorithm that focuses on continued stepwise care after ROSC (Figure 14). The main goal is to improve patient survival and quality of life. The major contributors to early mortality, usually in the first 24 hours following ROSC, are cardiovascular instability and brain injury.

In the general adult population, the most common cause of cardiac arrest is cardiovascular disease and myocardial ischaemia, and these continue to contribute to mortality, even after ROSC. It is of utmost importance to exclude and promptly treat ACS.

The only intervention shown to improve neurological outcome is therapeutic hypothermia (Figure 15), and this must be considered in all patients who are unable to follow verbal commands following ROSC.

The key components of post-arrest care are:

- Escalation of monitoring and care, i.e. invasive monitoring and intensive care admission.
- Targeted temperature management:
 - Institute therapeutic hypothermia;
 - Treat pyrexia.
- Organ-specific evaluation and support.
- Goal-directed application of vasoactive drugs:
- Mean arterial pressure of 65 mmHg and central venous oxygen saturation (ScvO₂) of 70%.
- Modifying outcomes by protocol-guided medical management, e.g. glucose control.
- Seizure control.
- Prognostication by means of expert neurological assessment and ancillary testing, e.g. brain single-photon emission computed tomography (SPECT).
- Organ preservation for donation and transplant.

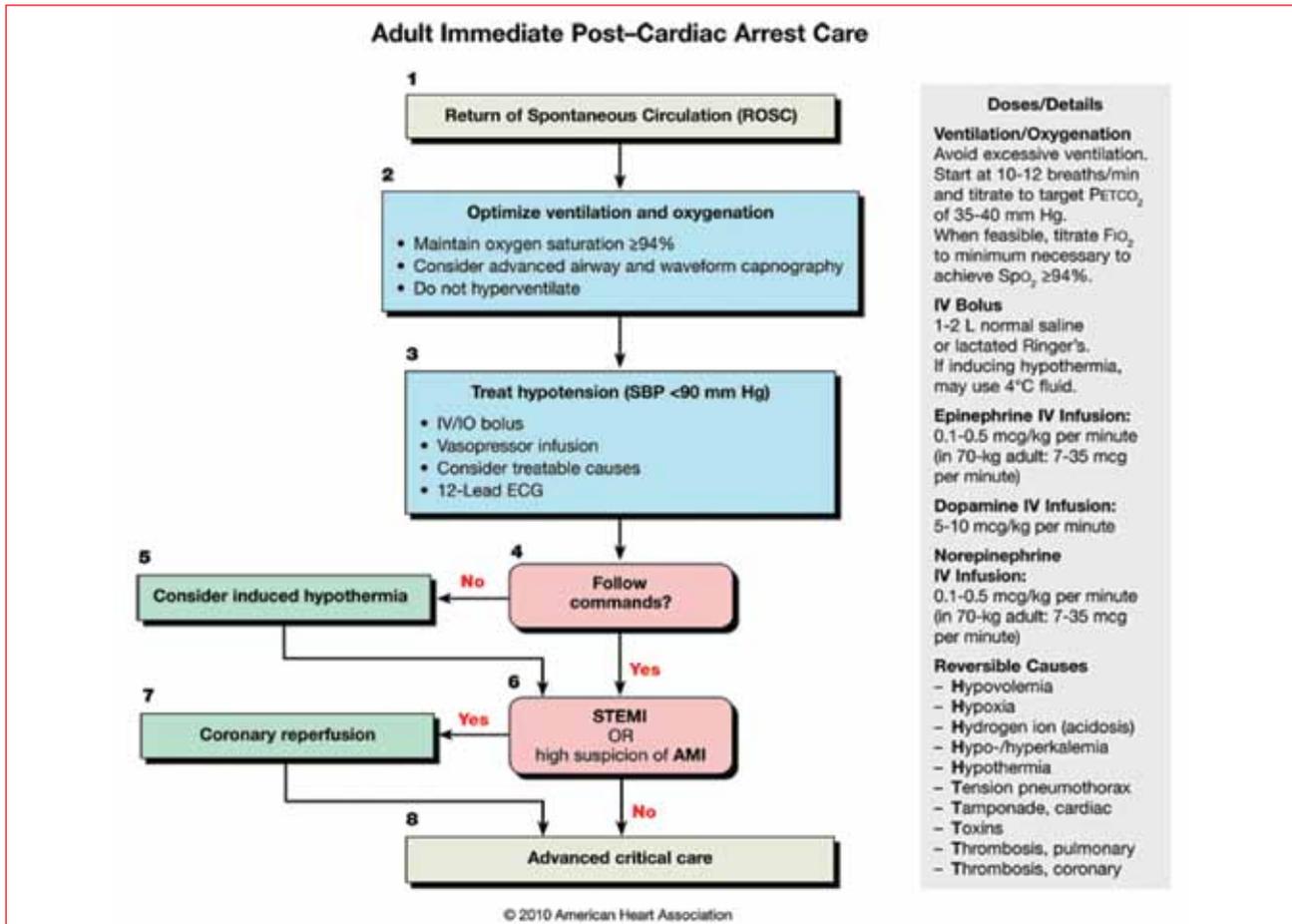


Figure 14: American Heart Association immediate post-cardiac arrest care algorithm

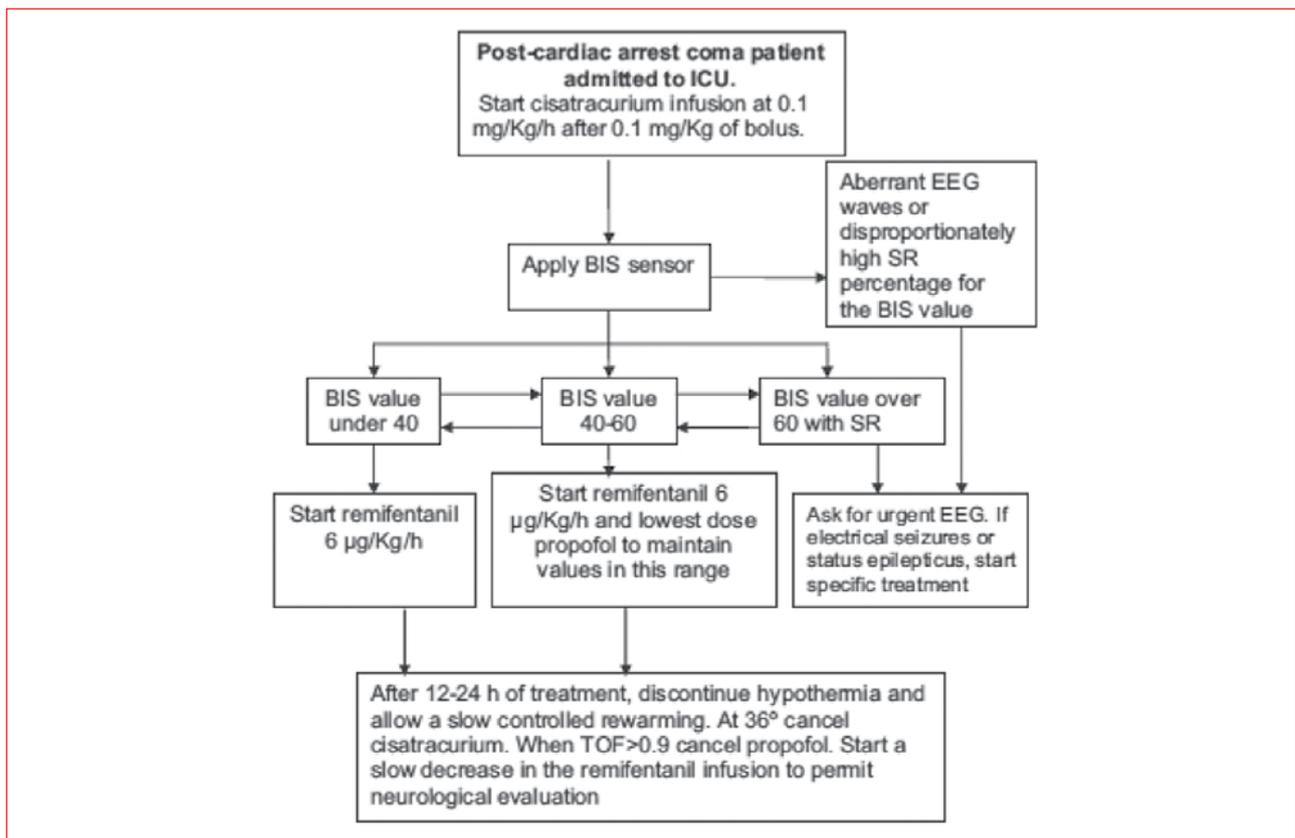


Figure 15: A suggested protocol for post-arrest therapeutic hypothermia

Conclusion

The major focus of the 2010 CPR guidelines is to simplify the entire process, and to improve layperson CPR. Key aspects of the guidelines are:

- A change in the CPR sequence, from ABC to CAB. This change is an attempt to address the reluctance to perform CPR. Most cases of adult SCA are characterised by VF or pulseless ventricular tachycardia.
- The critical components of treatment for these deadly rhythms are chest compressions and early defibrillation. The process of evaluating the airway delays the start of chest compressions.
- There is an emphasis on providing high-quality chest compressions. Push hard and fast. Keep interruptions to an absolute minimum. Allow full recoil of the chest wall. Do not hyperventilate.
- The recommended depth of compression is 5 cm.
- Chest compressions must be administered at a rate of at least 100 per minute.

Healthcare professionals should take note of the following important changes:

- Effective teamwork is essential.
- Capnography is an important tool, not only to confirm intubation, but also to monitor the quality of CPR.
- Therapeutic hypothermia should be considered as part of a multidisciplinary care plan after any cardiac arrest.
- Atropine is no longer recommended in the routine management of PEA and asystole.
- Adenosine may be used for the management of a wide-complex, stable, regular monomorphic dysrhythmia.
- Manual defibrillation is preferred for infants, but an AED may be used if a manual defibrillator is not available.

The use of a defibrillator (manual or AED) is an essential skill for any person responding to an arrest. These devices can be intimidating, but one should be familiar with their use, as direct current countershocks are the only curative therapy for some common peri-arrest rhythms.

Numerous devices are available to aid in airway management and ventilation. Unfortunately, training in their use is lacking, and some may do more harm than good if used incorrectly. In the majority of cases, basic airway management, i.e. head-tilt/chin-lift and bag-mask-valve ventilation, is sufficient to sustain adequate oxygenation.

Anaesthesiologists face a unique challenge in the perioperative setting. They must coordinate a team-based resuscitation. ACLS is optimised when a team leader effectively integrates CPR with advanced life-support strategies.

It is therefore of cardinal importance that we keep up to date with ongoing changes in resuscitation science, and update our skills regularly by attending appropriate refresher courses.

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