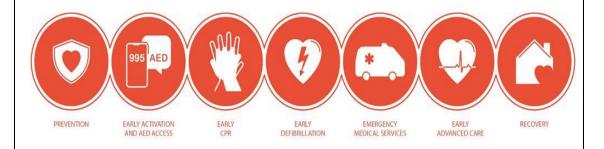


Life Support Course for Nurses



Singapore Resuscitation and First Aid Council

2022

Life Support Course for Nurses

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LSCN Apr 2019

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Introduction

The Life Support Course for Nurses (LSCN) is conceived and designed with the firm belief that there is a need for a custom made course for Singapore nurses. Though in every way an excellent course, the Basic Cardiac Life Support (BCLS) course does not impart sufficient knowledge and skills for nurses to resuscitate collapsed patients in hospitals and clinics.

The LSCN therefore aims to bridge the gap between BCLS and ACLS. The objectives are to impart:

- 1. critical background knowledge pertinent to the care of collapsed patients in hospitals and clinics, especially cardiac arrest patients
- 2. in-depth knowledge and skills in cardiopulmonary resuscitation
- 3. knowledge and skills in the treatment of ischemic heart disease and cardiac arrhythmias.

At every stage during the conception and creation of this course, we have kept in mind the realities that general ward nurses face daily in their work. In certain critical treatment issues, this course follows policies and guidelines given by the National Resuscitation Council, Singapore. However, recognizing that the work environment of Singapore nurses varies widely, we aimed to provide a general framework for which local practice can be adapted in other areas.

In this book, American nomenclature has been adopted for epinephrine (adrenaline), lidocaine (lignocaine, xylocaine) and norepinephrine (noradrenaline). It is conceivable that during the course, instructors may use these names interchangeably.

Conspicuously missing is the resuscitation of an arrested major trauma victim. The subject is a course unto itself and beyond the scope of the LSCN. We hope that nurses who are interested in this subject will attend relevant courses like Basic Trauma Life Support course etc. Pediatric and Infant Resuscitation which is important to a select group of nurses, is appended as an annex module.

Our nurses applying their newly acquired knowledge and skills to subsequent resuscitations define the ultimate success of this course. Very often, it is not just about whether the patient survived the arrest, but that the health care team can end that resuscitation attempt knowing that they have done their best for that patient.

Dr Tham Kum Ying July 1999

Preface to 2006 Edition

Since its launch in 1999 under the auspices of the Singapore National Resuscitation Council, the Life Support Course for Nurses (LSCN) has trained more than 200 nurses from various health care institutions, both in the public and private sectors. Among these nurses, many have become instructors for the course and gone on to train other nurses. The success of LSCN is due largely to the dedication and enthusiasm from the nurses themselves and the support from all the participating institutions.

The 2001 edition incorporated recommendations from 2 sources: (a) the American Heart Association Guidelines 2000 for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care and (b) National Resuscitation Council, Singapore. This 2006 edition again incorporates recommendations from ILCOR and also from National Resuscitation Council, Singapore. Since 2001, one of the significant events that impacted the Singapore healthcare practice scenario was the Joint Commission International (JCI) accreditation exercise which almost all the restructured hospitals participated in. The JCI exercise helped to reinforce the need for clinical staff to be properly trained in life support and resuscitation and gave participation in BCLS, AED, LSCN and ACLS courses a huge boost.

Regardless of the changes, LSCN remains true to its founding principle – that it is a custom made course for Singapore nurses and it aims to bridge the gap between BCLS and ACLS. Hence, all revisions and editions are written with this important principle in mind, so that LSCN will remain relevant to the nurses and nurses can continue to identify with it.

To all the nurses, doctors and institutions that have supported the LSCN and contributed to its success, we want to say a big thank you. We look forward to many more years of happy and fruitful collaborations.

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Dr Tham Kum Ying April 2006

Chapter 1. CHAIN OF SURVIVAL

Introduction

Cardiac disease is the third leading cause of death in Singapore. Cardiac arrest may occur as out of hospital cardiac arrest (OHCA) or in-hospital cardiac arrest (IHCA). Ongoing education efforts are underway to train members of the public to perform cardiopulmonary resuscitation (CPR). Community automated external defibrillators (AED) and ambulance dispatcher-guided CPR help public first responders render CPR. Nurses are often the first responders for IHCA. All nurses should be trained and prepared to initiate resuscitation when faced with a cardiac arrest within their vicinity, hand-over to and assist code blue teams when they arrive.

CPR includes a series of assessments and interventions that support cardiac and pulmonary functions. If CPR is performed correctly, (1) cardio-pulmonary function may be restored, or (2) cardio-pulmonary function may be maintained until advanced life support (ALS) is provided.

Chain of Survival

The essential steps for helping a cardiac arrest victim are illustrated in a system called the "Chain of Survival". The seven rings in this chain are: Prevention, Early Recognition and Access, Early CPR, Early Defibrillation, Emergency Medical Services (Ambulance), Advanced Cardiac Life Support and Recovery. (See Figure 1)



Figure 1 – The Chain of Survival

First Ring: Prevention

Prevention is better than intervention. A healthy diet and lifestyle can reduce the risk of heart diseases. Going for regular medical check-ups can help detect problems early and prompt the individuals to seek treatment early and adjust their diet and lifestyles.

Second Ring: Early Activation and AED Access

Call for ambulance (dial 995) and get an AED if visible and near-by. Follow the SCDF dispatcher's instructions.

Third Ring: Early CPR

The brain cells start dying within 4-6 minutes of cardiac arrest. CPR must be initiated as soon as possible to provide blood and oxygen flow to the brain and heart.

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Fourth Ring: Early Defibrillation

Automated external defibrillators (AEDs) are increasingly available in the community, at lift lobbies, void decks, gymnasium, shopping malls, hotels, airports and schools etc. Apply onto the casualty and be ready to defibrillate.

Fifth Ring: Emergency Medical Services (Ambulance)

Quick access to the scene and transport to the hospital makes a difference to the casualty's chances of survival.

Sixth Ring: Advanced Cardiac Life Support

Medical teams will provide advanced cardiac life support at the hospital with the use of Intravenous/Intraosseous (IV/IO) medication and delivery of oxygen via mechanical systems (i.e. Bag-Valve Masks and ventilators).

Seventh Ring: Recovery

To ensure a good survival outcome after OHCA requires continued community and medical support (i.e. social services, physio/occupational therapy, nutritional guidance, etc.) and therapy to re-habilitate the individual back to normalcy.

Emergency cardiac care includes all responses necessary to deal with sudden and often life-threatening events affecting the cardiovascular and pulmonary systems, in a patient. The components of emergency cardiac care are basic life support and ALS that include:

- 1. Recognizing early signs of heart attack and activating Emergency Medical Services (EMS) system, efforts to prevent complications, reassurance of the patient, and prompt availability of monitoring equipment.
- 2. Providing immediate basic cardiac life support (BCLS) at the scene when needed.
- 3. Defibrillating as quickly as possible if indicated.
- 4. Transferring the patient to hospital where definitive cardiac care can be provided.
- 5. Effective advanced cardiac life support and post cardiac arrest care.

The central issue in emergency cardiac care is optimal patient survival. Survival of cardiac arrest depends on a series of critical interventions. The concept of "Chain of Survival" is the best approach to the treatment of persons with cardiac arrest.

Advanced Cardiac Life Support (ACLS) is another important link (6th Ring). ACLS brings equipment to aid ventilation, establish intravenous access, administer medications, control arrhythmias and provide critical support. Post cardiac arrest care is part of the 6th ring and 7th ring (recovery) and includes: -

- 1. Continuous ETCO2 monitoring.
- 2. Targeted temperature management 33-36°C
- 3. Avoid excessive ventilation. Maintain SaO2 94-99%.
- 4. Maintain mean arterial pressure (MAP) > 65 mm Hg; SBP > 90 mm Hg
- 5. Glucose control. >10mmol should be treated but hypoglycaemia must be avoided.
- 6. Early percutaneous coronary intervention (PCI) as indicated.
- 7. Maintenance dose of antiarrhythmics
- 8. Extracorporeal CPR (eCPR) in selected patients with reversible aetiology

It is now routine for Emergency Departments and Intensive care units to monitor continuous ETCO2 and institute of targeted temperature management for the first 24 hours after return of spontaneous circulation.

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Summary

BCLS should be initiated when cardiac or respiratory arrest occurs. To increase the chances of success, the chain of survival must start with

- (1) Prevention.
- (2) Early Activation and AED Access.
- (3) Early CPR.
- (4) Early Defibrillation
- (5) Emergency Medical Services (Ambulance)
- (6) Advanced Cardiac Life Support
- (7) Recovery

Any delay could be fatal and should be avoided.

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Chapter 2: UPDATE ON BCLS 2016/2018/2022

Update on Cardiopulmonary Resuscitation for 2016/2018/2022

2006 Recommendations for Adults	2011 Recommendations for Adults	2016- 2018 Recommendations for Adults	Rationale
Recognition of cardiac arrest and activation of the EMS	No change	No change	The major cause of cardiac arrest in an adult is ventricular fibrillation and early defibrillation is the treatment of choice.
 2. Airway	2. AirwayOpen airwayNo changeCheck airwayOmitted	2. Airway Open airway Omit initial head-tilt chinlift Check airway Omitted	To reduce delay in starting chest compression.
3. Look, listen and feel for breathing • If no breathing, give two breaths	3. Look, listen and feel for breathing • Omitted 2 initial breaths • If no breathing or abnormal breathing, check pulse and commence CPR Gasping is considered abnormal breathing	 3. Look for rise and fall of chest (new change) Check breathing by looking for rise and fall of chest. Listen and feel for breathing is not required No change Gasping is considered abnormal breathing	 To reduce delay in starting chest compression. In the majority of cardiac arrests, the critical initial elements of CPR are chest compressions and early defibrillation.

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2006 Recommendations for Adults	2011 Recommendations for Adults	2016- 2018 Recommendations for Adults	Rationale
 Inspiratory time: 1 second per breath Inspiratory volume: 400-600ml of air and Rescue breathing: 12 breaths per minute 	No change	Post intubation 10-12 breaths / min, every 5 to 6 seconds 4. Breathing Post intubation 10-12 breaths / min, every 5 to 6 seconds	 Excessive high pressure and volume can overcome the esophageal opening pressure, causing air to enter not only the lungs but also the stomach. This causes gastric distension and predisposes patient to the risk of regurgitation and aspiration. During CPR, blood flow to the casualty's lungs is much less than normal, hence the victim needs less ventilation. Normal ventilation with enough volume to make the Casualty's chest rise is recommended. Giving rescue breaths over a shorter time will help to reduce the duration of essential interruptions.

2-2 LSCN Apr 2019

2006 Recommendations for Adults	2011 Recommendations for Adults	2016- 2018 Recommendations for Adults	Rationale
			Too many breaths or too large a ventilation volume increases the pressure in the casualty's chest, which in turn reduces the amount of blood refilling the heart. This reduces the blood flow generated by the next cycle of chest compression, reducing its effectiveness. Survival is thus compromised.
5. Compression	5. Compression	5. Compression	
	Pulse checkFor healthcare workers who are trained	Pulse checkNo change	The number of chest compressions delivered per minute during CPR is an important
Rate	Rate	Rate	determinant of return of
 "Approximately" 100/min 	At least 100/min	• 100-120/min	spontaneous circulation
	• 30:2 (1 or 2 rescuer)	No change	(ROSC). 30
	Location	Location	compressions to 2 ventilations decrease
	Lower half of sternum. Place middle finger on the notch and index finger next to middle finger.	Lower half of sternum. Place the heel of your hand on the lower half of the sternum.	the number of interruptions and reduce the likelihood of hyperventilation. On stopping chest

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2006 Recommendations for Adults	2011 Recommendations for Adults	2016- 2018 Recommendations for Adults	Rationale
Depth	Depth	Depth	compression, the coronary flow decreases substantially; on resuming chest compression, several compressions are necessary before the coronary flow recovers to its previous level. It is important to minimize interruptions to chest compressions.
Recovery Position Adult and Child Infant – Left Lateral Position	No change	Removal of Recovery position for (adult, child and infant) Reassessment – if both normal breathing and pulse are present, maintained casualty in supine position. Continuously monitor the casualty until help arrives	blood flow by increasing intrathoracic pressure and directly compressing the heart against the vertebrae. Compressions generate blood flow and oxygen delivery to the heart and brain. • Full chest recoil allows an adequate volume of blood to refill the heart, so that the blood flow with the next compression is maximized.

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2006 Recommendations for Adults	2011 Recommendations for Adults	2016- 2018 Recommendations for Adults	Rationale
	Compression-Only CPR: Significantly better than the outcome of giving no CPR	No change	Untrained rescuers or rescuers unwilling to provide mouth-to-mouth ventilations are to continue compressions-only CPR until victim starts to move (signs of life) / AED arrives/ professional help arrives. Trained layer rescuers and healthcare professionals are encouraged to perform both chest compressions with ventilations.
6. Foreign Body Airway Obstruction Heimlich manoeuvre: No change but more emphasis on bending victim forward. When casualty becomes unresponsive: Perform 30 chest compressions	6. Foreign Body Airway Obstruction No change	 6. Foreign Body Airway Obstruction Provide 1st Ventilation → if chest rises → Check for pulse and normal breathing 	

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2006 Recommendations for Child/Infant	2010 Recommendations for Child/Infant	2016- 2018 Recommendations for Child/Infant	Rationale
1. Sequence of ActionStart CPR for 2 minutesActivate EMS	No Change	 1. Sequence of Action Recognition of cardiac arrest and activation of the EMS Get AED 	The reason for this approach is that in most children and infants the major cause of cardiac arrest is hypoxia or shock.
Airway Open Airway Head tilt, Chin lift Check Airway Check for foreign body, remove it if present	2. AirwayOpen AirwayNo changeCheck AirwayOmitted	Airway Open Airway Omit initial head-tilt chinlift Check Airway No change	
 3. "Look, listen and feel" for breathing If not breathing, give two breaths 	 3. "Look, listen and feel" for breathing Omitted If not breathing, check pulse and commence CPR 	 3. Look for rise and fall of the chest (new change) Check breathing by looking for rise and fall of chest Listen and feel for breathing is not required No change 	To reduce delay in starting chest compression.

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2006 Recommendations for Adults	2011 Recommendations for Adults	2016- 2018	Rationale
Addits	Adults	Recommendations for Adults	
 4. Breathing 2 breaths Inspiratory time: 1 second per breath Inspiratory volume: sufficient to make the chest rise visibly Rescue breathing: 20 breaths per min 	 4. Breathing 2 breaths Inspiratory time: 1 second per breath Inspiratory volume: sufficient to make the chest rise visibly. Rescue breathing: 20 breaths per min 	 4. Breathing No change No change Infant (approx. 30mls/breath) 2022 Rescue Breathing Adult & child - No change Infant - 30 rescue breaths per minute (one breath every 2 seconds) 	 Infant collapsed rarely due to cardiac arrest. Most likely due to hypoxia
 5. Compression 30:2 (1 or 2 rescuer) Location: lower half of sternum Child: use heel of one hand or heel of one hand, other hand on top Infant: Place index finger on imaginary nipple line, use middle & ring finger to do chest compression 	 5. Compression No change No change No change No change 	 5. Compression No change No change No change No change 	Child/Infant (Compression ventilation ratio) If there are 2 trained healthcare providers 15:2 Perform 10 cycles of 15 compressions and 2 ventilations within 2 minutes

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2006 Recommendations for Adults	2011 Recommendations for Adults	2016- 2018 Recommendations for Adults	Rationale
 Infant 2cm Child 3cm Approx. 100/min Push hard, push fast Allow chest to recoil fully after each compression Compression-Only CPR: Requires prompt ventilations and chest compressions for optimal resuscitation 	Depth	Depth Infant 3-4 cm Child 4-5 cm 100-120/min for both No change No change	Child and Infant: Cardiac arrest is likely due to hypoxia, hence compression only CPR is not likely to be effectively to be due.
Foreign Body Airway Obstruction Child Perform Heimlich manoeuvre until the object is expelled or the casualty becomes unresponsive When casualty becomes unresponsive: perform 30 chest compress	 6. Foreign Body Airway Obstruction No change No change 	 6. Foreign Body Airway Obstruction Provide 1st Ventilation → if chest rises → Check for pulse and normal breathing No change 	Conscious FBAO (Adult/Child/Infant) – No Change Unconscious FBAO (Adult/Child/Infant) After 30 chest compressions Check for FB → No FB Perform another 30 Chest comp If FB is seen, remove, then Provide 1st Ventilation → if chest rises → Check for pulse and normal breathing

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2006 Recommendations for Adults	2011 Recommendations for Adults	2016- 2018 Recommendations for Adults	Rationale
 Infant Perform 5 back blows and 5 chest thrusts When casualty becomes unresponsive: perform 30 chest compressions 	No changeNo change	 2022 No change Unconscious FBAO (Infant) Perform 30 chest compressions (same as CPR) HTCL, Check for FB → No FB Perform another 30 Chest comp If FB is seen, remove, then Provide 1st Ventilation → if chest rises → Check for pulse and normal breathing 	

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Rescuers CPR with an Advanced Airway

Once an advanced airway (e.g. endotracheal tube) is in place, the compressions need not be synchronized with ventilation. The rescuer performing compression should deliver at least 100-120 compressions per minute continuously, whilst the rescuer delivering the ventilations should ventilate at a rate of 1 ventilation after every tenth chest compression or every 5 to 6 seconds (approximately give 10-12 breaths per minute), each keeping their own timing. This allows uninterrupted chest compressions.

Recovery Position

There are several variations of recovery position, each with its own advantages. No single position is perfect for all casualty. The position should be stable with no pressure on the chest to impair breathing.

Removal of recovery position (Adult/Child/Infant) - 2022 update

National Guidelines 2016; CPR Sequences Across Age Group

CPR Sequence	Adult and Older Child	Child (1-8 year of Age)	Infant (less than 1 year of Age)
Check for danger Establish responsiveness	Tap on casualty's shoulders firmly, shout "Are you okay?"		"Are you okay?"
Call 995, get AED	Immediately	If AED is vi metres	sible or within 100
Open Airway	Omit initial F	lead Tilt – C	hin Lift
Recognition of Cardiac Arrest	Check for normal breathing	(gasping is N	NOT normal breathing)
Pulse Check (for trained healthcare providers only)	Carotid Brachial		Brachial
Start Chest Compressions	If no normal breathing or pulse check (by trained healthcare providers only) within 10 seconds, start chest compressions and perform 5 cycles of CPR (30:2)		
Compression landmarks			Lower half of sternum (just below intermammary line)
Compression Method	Heel of 1 hand, other on top 2 fingers		2 fingers
Compression Depth	4 - 6 cm	4 - 5 cm	3 – 4 cm
Compression Rate	100 – 120 / minute		
Compression: Ventilation Ratio	30: 2 Child & Infant – if 2 trained healthcare providers are present, Compression: ventilation ratio 15:2		
Breathing 2 breaths at 1 second per breath			

Summary

2016/2018/2022 National BCLS guidelines updates:

Guidelines	Adult	Infant / Child	
Chain of Survival	7 Rings to chain of survival – Prevention, Early recognition and early access, Early CPR, Early defibrillation, Early emergency medical services, Early advanced cardiac care which includes post cardiac arrest care and Recovery		
1 rescuer CPR	Activate EMS and get AED for	Activate EMS and get AED for infant CPR, similar to adult setting	
	Omit initial head-tilt and chin-lift	Omit initial head-tilt and chin- lift	
	Check breathing by looking for rise and fall of chest. Listen and feel for breathing is not required	Check breathing by looking for rise and fall of chest. Listen and feel for breathing is not required	
	Compression Rate of 100- 120/mins	Compression Rate of 100- 120/mins	
	Compression depth of 4-6 cm	Compression depth 4-5 cm for Child.	
		Compression Depth 3-4 cm for Infant	
	Minimise interruptions in chest compressions	Minimise interruptions in chest compressions.	
	Avoid excessive ventilation	Avoid excessive ventilation	
	CPR landmark - place heel of hand over lower mid sternum, finger tracing above xiphisternum is not required.	 No change Ventilate 10-12 breaths /min post intubation 	
	Ventilate 10-12 breaths /min post intubation		
FBAO	1 successful ventilation is sufficient	1 successful ventilation is sufficient	
Defibrillation	Perform 2 mins CPR after defibrillation		

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Chapter 3. ACUTE CORONARY SYNDROME

Introduction

Acute myocardial infarction (AMI) and unstable angina are now recognized as part of a spectrum of clinical disease collectively known as acute coronary syndrome (ACS). This syndrome includes ST elevation Myocardial Infarction (STEMI), non ST elevation Myocardial Infarction (NSTEMI) and unstable angina pectoris (UAP).

The common pathophysiological event in ACS is erosion or rupture of an atherosclerotic plaque. This attracts platelets and fibrin, which form a clot, leading to coronary thrombosis and occlusion of the coronary artery.

The definition of acute myocardial infarction is death of a portion of the cardiac tissue which has been acutely deprived of blood and oxygen supply. Early diagnosis of AMI is crucial as mortality is highest in the first few hours of the event and treatment is most effective at the shortest interval possible after the onset of symptoms.

A 12 lead electrocardiogram characteristics (absence or presence of ST elevation) differentiate STEMI from NSTEMI. NSTEMI may present with ST segment depression, nonspecific ST segment wave abnormalities, or even a normal ECG. In the absence of ST elevation, the presence of a suggestive history and positive cardiac enzymes, such as troponin T or I, characterize NSTEMI and distinguish it from STEMI and UAP respectively.

Clinical Presentation

- 1. Chest pain.
- Chest pain results from hypoxia of cardiac muscle.
- The classic description of ischemic chest pain is a squeezing, pressing, vice-like discomfort over the substernal or left precordial region. The pain may radiate to the neck, iaw or shoulders or down the arms.
- A characteristic feature of angina is the gradual build-up of discomfort.
- It may occur over the posterior thorax, interscapular area, lower jaw, neck, throat area or epigastrium.
- 2. AMI, in patients with known coronary artery disease, may present with chest pain that is:
- increasing in frequency or severity over previously stable chest pain.
- occurring at rest.
- unrelieved by rest or sublingual nitroglycerin (GTN).
- 3. Some patients may present with the following:
- epigastric pain especially in inferior myocardial infarction.
- shortness of breath, associated with sweating, pain in the jaw or teeth.
- seizure attacks, confusion, change in mental status due to hypoxemia or sudden deterioration in previously stable medical conditions. Elderly patients and those with diabetes mellitus may present in this manner without complaint of chest pain.
- 4. In some patients, physical examination may be normal. While others may show signs of heart failure or shock e.g. breathlessness, pulmonary edema, low blood pressure.

Evaluation and diagnosis of AMI

Evaluation

The 3 principal parameters for evaluating a patient with AMI are:

- 1. Clinical history
- 2. ECG
- 3. Cardiac markers

Cardiac enzymes, whether CKMB or troponin may not be significantly elevated in the first few hours after the onset of infarction; therefore, early diagnosis frequently depends on the clinical history and ECG.

In 2000, the European Society for Cardiology and the American College of Cardiology advocated a slightly different approach to diagnose ACS. They emphasized cardiac enzymes rise and fall, AND 1 of the following:

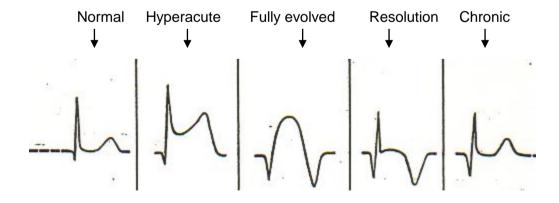
- History of cardiac ischemic symptoms, or
- ECG changes like ST elevation or depression, Q wave, new bundle branch block, tall peaked T wave or
- Findings during coronary artery intervention

ECG Diagnosis

The ECG leads placed over the site of infarction will record the following changes:

- Pathological Q waves reflecting tissue necrosis. Q waves are significant if more than 0.04 second and at least one-third the height of the R wave in the same QRS complex.
- Elevated ST segments reflecting tissue injury. In anterior leads ST elevation needs to be 2mm or more, and in inferior ST elevation needs to be 1mm or more to diagnose AMI.
- 3. T wave inversion reflecting tissue ischemia.

Fig. 3.1 ECG changes in AMI



However it is important to note that these changes are only seen when reperfusion therapy (e.g. thrombolytic therapy) is not instituted.

Some patients with AMI may record a normal initial 12 lead ECG. If the history is strongly suggestive of AMI, it is important to repeat the 12 lead ECG 15 to 30 minutes later. An example of a 12 lead ECG (Fig. 3.2) showing AMI changes is given on page 3-4

ST changes in AMI

Area of infarction	Leads showing ST elevation
Left ventricle massive anterior wall	V2-V6, I, aVL
Left ventricle antero-septal wall	V2-V3
Left ventricle antero-lateral wall	V3-V6, I, aVL
Left ventricle inferior wall	II, III, aVF
Right ventricle	Right chest leads V4-V6
Posterior wall	Posterior leads V7-V9

Fig. 3.2 Anterior-lateral AMI showing ST elevation in leads V2-6

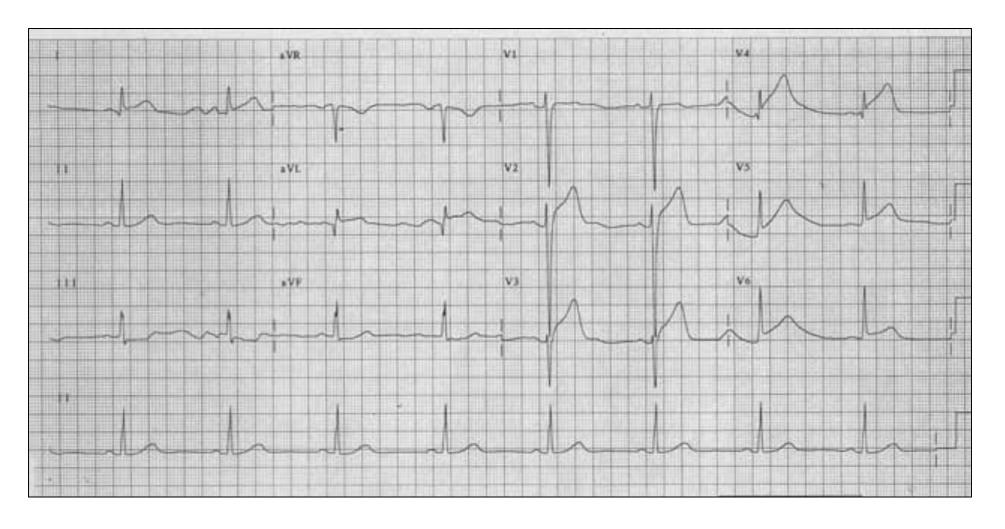
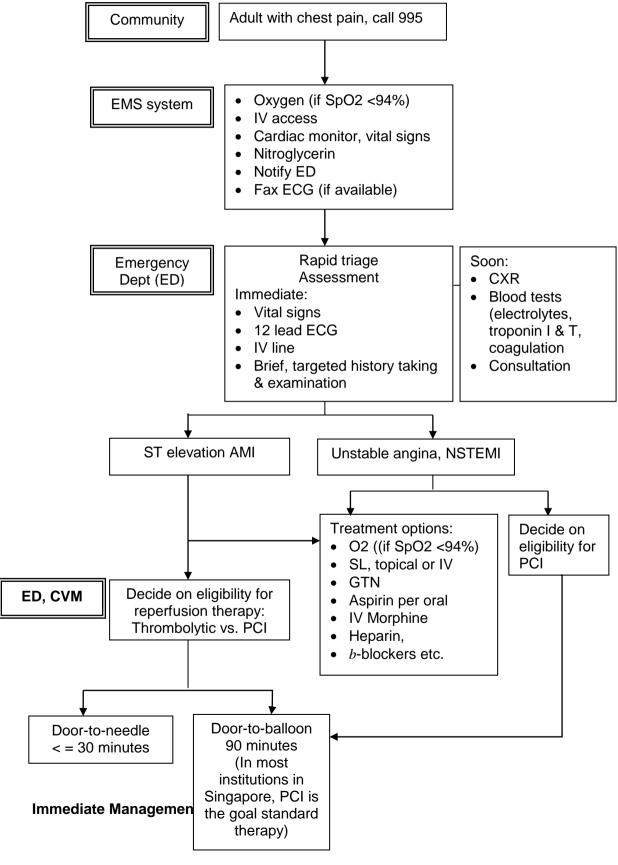


Fig. 3.3 Acute Myocardial Infarction Management Algorithm

Recommended for early management of patients with chest pain and possible AMI. The initial therapy in the ED may vary according to resources and institutional regulations.



ECG Monitoring

ECG monitoring records and allows early detection of arrhythmias complicating AMI. Serious ventricular arrhythmias occur most commonly within the first 4 hours of myocardial infarction. The most lethal arrhythmia is ventricular fibrillation, which occurs in about 10 to 15 % of patients with AMI. For classification and treatment of arrhythmias please refer to Chapter 6.

Intravenous Access

Intravenous access must be established for drug administration and fluid therapy. Peripheral venous cannulation should be the first choice of access.

Oxygen Therapy

Hypoxemia may aggravate myocardial damage, increase the risks of complications and should be corrected promptly. Supplemental oxygen through nasal prongs or facemask should be given especially for patients if SpO2 is \leq 94% and/or on thrombolytic therapy. Patients who have difficulty maintaining their airway may require advanced airway support.

Relief of Pain

Pain relief is important because it helps to reduce the secretion of catecholamines, thereby reducing the risk of arrhythmias. Sublingual GTN or IV morphine may be prescribed. It is important to note that intramuscular injections should be avoided because:

- 1. Absorption may be erratic as a result of hypotension.
- 2. Subsequent thrombolytic therapy may cause a muscular hematoma.
- 3. Damaged muscles release enzymes that may interfere with the interpretation of cardiac enzymes.

Morphine should be used with caution in unstable angina/NSTEMI because IV Morphine was associated with increased mortality in a large registry. It is also not recommended for use in patients with hypotension.

Reperfusion Therapy

PCI is the preferred reperfusion strategy provided it is performed in a timely manner with a door-to-balloon time <90 minutes by an experienced team. Various strategies to decrease door-to-balloon time include faxing of ECG by paramedics to ED, activation of the ED to standby for the patient, ECG at ED triage for walk-in patients with chest pain, activation of PCI lab by ED physician, rather than waiting for the cardiologist to review the patient. PCI is an invasive procedure involving the insertion of a stent across a blocked coronary artery to dilate the vessel. This improves blood flow to the myocardium, limits infarct size, thus reducing chest pain. The immediate outcome depends on a number of factors but the overall success rate is about 90 to 95%. A stent may be left in the coronary artery to maintain longer patency.

Thrombolytics have increased risk of hemorrhagic stroke, PCI has lower risk, and hence PCI is the reperfusion strategy of choice in the elderly and those at risk for bleeding complications.

Thrombolytic Therapy

The key factor in treatment of AMI is the restoration of interrupted blood flow to the ischemic myocardium and normalization of the coronary blood flow. Thrombolytic therapy induces dissolution of fibrin clot. It has a major impact in limiting infarct size and reducing mortality. Ideally these agents should be given within the first 6 hours of onset of symptoms for maximum benefit. Some recent data suggest that thrombolytic therapy up to 12 hours after onset of symptoms may still reduce mortality. There is still a role for thrombolytic therapy in hospitals which do not have PCI capabilities. After successful thrombolysis, the patient needs to be transferred to a tertiary centre for angiography.

The preferred fibrinolytic regime is accelerated recombinant tissue plasminogen activator (rt-PA) with heparin. Other new lytic agents include reteplase (rPA), TNK-tPA, lanoteplase (nPA) and saruplase.

Guidelines on Contraindications and Cautions for Thrombolytic Use in AMI Contraindications

- 1. Previous hemorrhagic stroke at any time, other strokes or cerebrovascular events within 1 year.
- 2. Known intracranial neoplasm.
- 3. Active internal bleeding (does not include menstruation).
- 4. Suspected aortic dissection.

Cautions / relative contraindications

- 1. Severe uncontrolled hypertension on presentation (BP > 180/110 mmHg).
- 2. History of prior cerebrovascular accident or known intracranial pathology not covered in contraindications.
- 3. Current use of anticoagulants in therapeutic doses (INR > = 2-3), known bleeding diathesis.
- 4. Recent trauma (within 2-4 weeks) including head trauma or traumatic or prolonged CPR (>10 min) or major surgery (<3 weeks).
- 5. Non-compressible vascular puncture.
- 6. Recent internal bleeding (within 2-4 weeks.
- 7. For streptokinase / anistreptase, prior exposure (especially within 5 days-2 years)
- 8. Pregnancy.
- 9. Active peptic ulcer.
- 10. History of chronic severe hypertension.

Coronary Artery Bypass Grafting (CABG)

CABG is a surgical method for enhancing delivery of blood flow to the previously ischemic myocardium. A vessel e.g. leg vein is harvested and grafted onto the heart, thereby bypassing the arteriosclerotic coronary arteries and restoring blood flow.

Chest pain protocols

Patients presenting to the ED with a history of atypical chest pain with few risk factors for ACS will need evaluation with serial troponins and ECGs at least 6 hours apart (this is commonly known as chest pain protocol). These protocols improve accuracy in identifying patients requiring inpatient admission or further diagnostic testing while maintaining patient safety, reducing LOS and cost.

Patients with STEMI or NSTEMI or those with typical sounding chest pain in the presence of multiple cardiac risk factors are NOT candidates for chest pain protocol. These patients should be admitted and evaluated expediently.

Anti-Platelet Therapy

It is believed that platelets play an important role in thrombogenesis. Three groups of anti-platelet medications are currently in use: aspirin, clopidogrel (Plavix®) and glycoprotein IIB/IIIA receptors. Studies have already shown that aspirin and clopidogrel in addition to percutaneous coronary intervention improves patients' outcome.

Adjunctive Therapy that may be useful are:

- β-blockers
- Vasodilators Nitroglycerin
- ACE inhibitors

Complications of AMI

Arrhythmias

Refer to chapter 6.

Cardiogenic shock

Cardiogenic shock is characterized by shock due to a cardiac cause, whereby:

- Hypotension Systolic BP less than 80 to 90 mmHg or reduction of 70 mmHg or more.
- 2. Oliguria, urine <30 ml /hour.
- 3. Clinical signs of hypoperfusion e.g. mental obtundation, pallor, sweating, tachycardia.

Prognosis is grave if cardiogenic shock is due to massive loss of functioning myocardium (40% or more). Treatment includes using inotropic agents, which enhances the heart's pump capabilities, are used to improve the contractility and correct the hypotension e.g. dopamine, dobutamine or norepinephrine. Should that not suffice, an Intra-aortic balloon counterpulsation (IABP) may be used.

Myocardial Rupture

Myocardial rupture may occur in about 10 % of fatal transmural infarct. It is associated with massive hemopericardium, cardiac tamponade and pulseless electrical activity. Attempts to resuscitate this condition often fail.

Acute Pulmonary Edema

Patient will often be very ill and distressed with dyspnoea, tachypnoea, tachycardia and sweating. Some patients may have a pink frothy discharge from the nose and mouth.

Management includes:

- 1. placing the patient in an upright position.
- 2. providing supplemental oxygen.

- 3. administering intravenous frusemide and morphine.
- 4. administering intravenous vasodilator if there is hypertension, or
- 5. administering intravenous inotrope if there is hypotension.
- 6. mechanical ventilation if indicated.

Mitral Regurgitation

AMI may involve the chordae or papillary muscle, giving rise to impairment of the mitral valve resulting in regurgitation. Frank papillary muscle rupture may lead to immediate hemodynamic compromise, acute pulmonary edema and often sudden death. On the other hand a subtle disruption of the mitral valve apparatus may produce a mild mitral regurgitation with little or no hemodynamic disturbance.

Intra-aortic balloon counterpulsation and a combination of inotropes and vasodilators may be required to improve patient's hemodynamic status before surgical intervention.

Interventricular Septal Rupture

Rupture of the interventricular septum often produces a significant left to right shunt. The patient may present with cardiogenic shock or pulmonary edema. The murmur produced may be similar to that of acute mitral regurgitation. Insertion of a Swan-Ganz catheter enables differentiation between the two conditions. Often intra-aortic balloon counterpulsation with inotropes and vasodilators are employed to improve the patient's hemodynamic status. Early surgery for repair of ventricular septal defect has been advocated.

Right Ventricular Infarction

Right ventricular infarction is more common in inferior myocardial infarction than in anterior infarction. About 30% of patients with acute inferior myocardial infarction have an associated right ventricular infarction. Half of these patients may have right ventricular damage severe enough to cause failure of the entire right heart and pulmonary circulation. The right ventricular end-diastolic pressure is markedly increased resulting in a raised right atrial and jugular venous pressure. The patient may present with hypotension with distended neck veins. A raised JVP in the absence of pulmonary venous congestion (no crepitations in the lungs) should alert one to the possibility of right ventricular infarction. Treatment includes fluid challenge and inotropic agents. Diuretic should be avoided.

Summary

Acute coronary syndrome develops as a result of an abrupt rupture of the atherosclerotic plague. Early diagnosis is of utmost importance as mortality is highest and treatment is most effective. For patients presenting within 12 hours of symptom onset and ECG findings consistent with STEMI, reperfusion should be initiated as soon as possible. Better patient outcome and prognosis is obtained with percutaneous coronary intervention.

Chapter 4. AIRWAY MANAGEMENT

Introduction

Airway management is one of the important cornerstones in resuscitation of a collapsed patient. In this chapter, basic and advanced airway management will be discussed together with a review of importance of oxygen, adjuncts used in airway management and different methods of oxygen administration.

The importance of oxygen to human tissue

All living tissue requires oxygen to function. This is more so in cells of the brain, heart and kidneys. For example, brain cells will die when deprive of oxygen for about 4 to 6 minutes. Therefore in a collapsed patient delivery of oxygen is of prime importance. Anyone in cardio-respiratory distress should receive supplemental oxygen, which may prevent a cardiac or respiratory arrest.

Approach to the Patient with Potential Airway Problem

Basic life support emphasises the importance of a systemic approach to a patient with cardiac or respiratory arrest. The initial step is to establish unresponsiveness. Next is to determine the presence of spontaneous respiration.

When spontaneous respiration is present, the patient should be given supplemental oxygen. In the absence of spontaneous respiration, follow the algorithm of unconscious foreign bodies airway obstruction.

Possible causes of upper airway obstruction:

- 1. Foreign bodies e.g. secretions, blood, dentures, food particles
- 2. Posterior displacement of the tongue
- 3. Swelling of the pharynx, larynx or epiglottis e.g. acute epiglottis, laryngeal oedema

Manoeuvres to Open the Airway

Head tilt and chin lift

When there is acute airway obstruction, opening the airway is of top priority during resuscitation. Upper airway obstruction in an unconscious patient is the result of the loss of tonicity of the submandibular muscles, which provide direct support to the tongue and indirect support to the epiglottis. Posterior displacement of the tongue occludes the airway at the level of the pharynx. The epiglottis may occlude the airway at the level of the larynx.

The basic technique for opening the airway is head tilt and chin lift with anterior displacement of the mandible (see Fig 4.1). In a trauma patient with suspected neck injury, the initial step for opening the airway is the modified jaw thrust (Fig. 4.2).

These manoeuvres should be attempted before any airway adjunct is used. If the patient is breathing spontaneously, proper airway positioning may be all that is required. In some instances, an oropharyngeal or nasopharyngeal airway may be needed to maintain airway patency.





Fig. 4.1 Head tilt and chin lift





Fig. 4.2 Jaw thrust

Adjuncts for Airway Management

Oropharyngeal airway

The oropharyngeal airway is a J-shaped device that will hold the tongue away from the posterior wall of the pharynx when it is properly placed. Oropharyngeal airway facilitates suctioning of the pharynx and prevents the patient from biting and occluding the endotracheal tube. The most commonly used is the Guedel type.

a. Determination of size of oropharyngeal airway

The size is based on the distance, in millimetres, from the flange to the distal tip. The following sizes are recommended:

Large adult: 100 mm (Guedel size 4) Medium adult: 90 mm (Guedel size 3) Small adult: 80 mm (Guedel size 2)

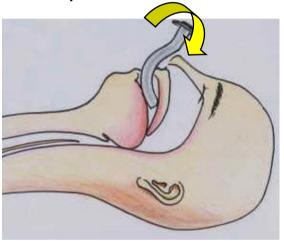
A practical way to determine the appropriate size is to align the airway from the corner of the patient's mouth to the tragus of his ear (See Fig 4.3). The second method is to measure from the middle of the incisor to the angle of the jaw.





Fig. 4.3 Determining the correct size of oropharyngeal airway

b. Techniques of insertion



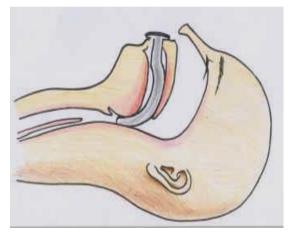


Fig 4.4 Insertion of oropharyngeal airway

- 1. If necessary the mouth and pharynx should be cleared of secretions, blood, or vomitus, using a rigid pharyngeal suction tip (Yankauer).
- 2. The airway is first inserted backward as it enters the mouth.
- 3. As the airway transverses the oral cavity and just before the junction of the hard and soft palate, the operator rotates the airway into its proper position thereby preventing injury to the soft palate.
- 4. Another method is to depress the tongue with a tongue depressor before the airway is inserted with its curve facing the tongue.
- 5. The indication that the airway is in position and of the correct size is the presence of clear breath sounds on auscultation of the lungs during ventilation.
- 6. It should be remembered that, even with the use of the airway, proper head position must be maintained.

c. Complications

1. If the airway is too long, it may press the epiglottis against the entrance of the larynx, producing complete airway obstruction.

- 2. If the airway is not inserted properly, it is possible to push the tongue posteriorly, aggravating the problem of upper airway obstruction.
- 3. To prevent trauma, the operator should make sure that the lips and the tongue are not caught between the teeth and the airway.
- 4. The airway may stimulate vomiting and laryngospasm in the conscious and semiconscious patients and therefore should only be used in the unconscious patients.

Nasopharyngeal airways

These are uncuffed tubes made of rubber or plastic. Their use is indicated when the insertion of an oropharyngeal airway is technically difficult or impossible e.g. trismus, massive trauma around the mouth, mandibulo-maxillary wiring etc.

It is not to be used on patients with suspected fracture of the base of skull as there is a possibility of the airway being introduced intracranically.



Fig.4.5 Inserting a nasopharyngeal airway



Fig. 4.6 Nasopharyngeal airway in place

a. Determination of size

The sizes are indicated in millimetres. The length of the tubes varies with the internal diameter. Recommended sizes are as follows:

Large adult: 8.0 - 9.0 Medium adult: 7.0 - 8.0 Small adult: 6.0 - 7.0

b. Technique of insertion

- 1. The airway is lubricated with a water-soluble lubricant.
- 2. It is inserted gently close to the midline along the floor of the nose into the posterior pharynx behind the tongue.
- 3. If resistance is encountered, slight rotation of the tube may facilitate the passage at the angle of the nasal passage and the nasopharynx.

c. Complications

- 1. If the tube is too long, it may enter the oesophagus, causing gastric distension and hypoventilation during artificial ventilation.
- 2. Though this type of airway is better tolerated by the semiconscious patient, its use may still precipitate laryngospasm and vomiting.
- 3. The insertion of the airway may injure the nasal mucosa with bleeding and possible aspiration of clots into the trachea. Suction may be needed to remove secretions or blood.
- 4. It is important to maintain head-tilt with anterior displacement of the mandible by chinlift and if, necessary, jaw-thrust when using this airway.

Immediately after insertion of the pharyngeal airway (oral or nasal), check for respiration. If absent or inadequate, artificial positive-pressure ventilation should be initiated with a mouth-to-mask technique, bag-valve-mask, or the oxygen-powered breathing device.

Administration of Supplemental Oxygen

Oxygen content and oxygen delivery are compromised during cardiac arrest. A system that provides high-flow oxygen (100%) should be used during artificial ventilation. If a patient is breathing spontaneously, the administration of oxygen should be titrated according to the PaO2, or oxygen saturation value.

Nursing staff should have an understanding of the oxygen delivery system. This includes:

- 1. oxygen supply (cylinder or piped wall oxygen).
- 2. valve handles to open the cylinder, pressure gauge and flow meter.
- 3. connecting tubing from oxygen supply to the patient's oxygen administration unit.
- 4. humidifier.

Devices Used for Administration of Supplemental Oxygen

1. Nasal Cannula

This is a low-flow system that does not provide sufficient gas to supply the entire inspired volume; therefore, part of the tidal volume must be supplied by room air. The inspired oxygen concentration depends on the flow of oxygen in the unit and the tidal volume of the patient. For every increase from 1 L/min flow, the inspired oxygen concentration will be increased by approximately 4%. The oxygen concentration supplied to a patient with a normal tidal volume by the nasal cannula with a flow of 1-4 L/min is 24-36%.



Fig. 4.7 Nasal cannula

2. Simple facemask

This unit is well tolerated by the adult patient. In order to avoid accumulation of exhaled air in the mask, the minimum oxygen flow must be 5L/min; the recommended flow is 5-8 L/min. Like the nasal cannula, there is dilution of the inspired oxygen by room air. This system provides concentrations of oxygen of 40-60%.



Fig. 4.8 Simple facemask

3. Face mask with oxygen reservoir (Non-rebreathing masks)

This system, in which there is a constant flow of oxygen into the reservoir, will provide oxygen concentrations higher than 60%. A flow of 6L/min will provide approximately 60% oxygen concentration and each increase in flow of 1L/min increases the inspired oxygen concentration by 10%. At 10L/min the oxygen concentration is almost 100%.



Fig. 4.9 Non rebreathing mask

4. Venturi Mask

The Venturi mask provides a high gas flow with a fixed oxygen concentration. Oxygen under pressure is passed through a narrow orifice and, after leaving the orifice, provides a subatmospheric pressure that entrains room air into It delivers a controlled oxygen the system. concentration to a patient. By changing the orifice size of the two adjustable colour-coded diluters (green and white) the oxygen concentration is This type of oxygen delivery is changed. frequently used in chronic obstructive pulmonary possible disease patients with chronic hypercarbia for the treatment of moderate-tosevere hypoxaemia. The administration of high oxygen concentration produces sudden а increase in PaO2, which blocks the stimulant effect of hypoxaemia on the respiratory centre, thereby causing respiratory depression in these patients.



Fig. 4.10 Venturi mask

Oxygen concentrations available are 24%, 26%, 28%, 30%, 35%, 40%, and 50%. The oxygen flow rates to be used for these concentrations are 3 to 15 LPM. The mask with

24% oxygen concentration is used initially. The patient is observed for respiratory depression and PaO2 is evaluated. The oxygen concentration is then titrated to the desired level of PaO2.

* Information obtained from	product	(Multi-vent) leaflet
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O2 flow (LPM)	Oxygen concentration (%)	
Low oxygen concentration diluter (green)		
3	24, 26	
6	28, 30	
Medium oxygen concentration diluter (white)		
9	35	
12	40	
15	50	

5. High Concentrator Mask

It delivers high concentrations of oxygen through the addition of a reservoir bag to achieve oxygen concentration of 60% to 80%. The oxygen flow rate should be administered at 8-12 LPM. Unlike the non-rebreathing mask, this mask does not contain any valves.



Fig. 4.11 High Concentrator Mask

Flow rate as recommended by manufacturer/hospital guidelines.

Bag-Valve-Mask

The bag-valve-mask (BVM) consists of a silicone bag and well-fitting mask that allows manual assisted ventilation. It is non-invasive and therefore easy to learn and use. Some useful tips to remember when using the BVM:

- The correct size mask should cover the bridge of the nose and sit on the chin.
- The patient's airway must be adequately opened with head-tilt and chin lift to minimize insufflations of the stomach.
- The mask can be held with either hand, using the other hand to squeeze the bag.
- The thumb and index finger holds the mask firmly down against the patient's face while the other 3 fingers are spread along the bony part of the jaw, lifting it up to obtain a good seal between mask and face.
- Do not allow the 3 fingers to compress on the soft tissue of the neck.
- A 2-hand technique may be needed to hold the mask if an adequate seal cannot be obtained.



Fig. 4.12 One-hand technique for BVM ventilation



Fig 4.13 Two-hand technique for BVM ventilation

Laryngeal Mask Airway

The Laryngeal Mask Airway (LMA) is a silicone device for airway management. It is an adjunctive airway device composed of a tube with a cuffed mask-like projection at the distal end (see Fig 4.5). It was designed by British anaesthesiologist Archie J. Brain in 1983 as an alternative method of ventilation and provides an end-to-end connection between the natural airway and an artificial airway. The low-pressure cuff provided an airtight seal around the glottis. The LMA provides a more effective seal than the facemask yet is less invasive than endotracheal intubation. It is inserted blindly into the pharynx and inflates with air to form a seal against the laryngeal inlet.





Fig 4.14 LMA sizes 4 and 3 with deflated (left) and inflated (right) cuffs

a. Indications

Indications for use of the LMA are the same as those for assisted ventilation. Additional indications are

- Where the facemask cannot achieve an adequate seal.
- Where intubation is difficult, hazardous or unsuccessful.

b. Contraindications

- morbidly obese patients
- patients at risk for aspiration
- patients with high airway resistance, limited pulmonary compliance
- pharyngo-tracheal pathology e.g. tumour, abscess, haematoma

c. Determination of size

When electing to use an LMA it is important to choose the correct size:

Size 3 - small adult

Size 4 – normal adult

Size 5 – large adult

d. Equipment required for LMA insertion

- 1. 50 ml syringe (for cuff inflation)
- 2. Water soluble lubricant
- 3. Tape to secure LMA
- 4. Bite Block if available

e. Technique of Insertion

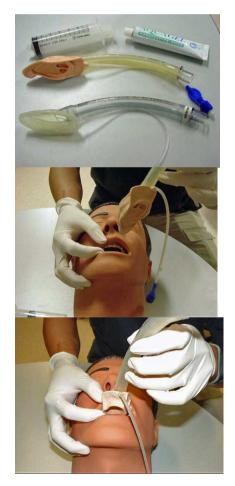
The LMA may be inserted by nurses and doctors trained in its use to achieve a secure airway in all patients who are suddenly profoundly unconscious and require an artificial airway. Many of these patients will also be pulseless.

Step 1 Be sure that the mask is made well deflated, so that it becomes a stiff, flat "shovel". Hyper-extend the head slightly, so the mouth opens. Take the LMA between index and thumb; insert it into the patient's mouth by pressing the mask firmly against the palate.

Step 2 Push the mask further into the mouth with your index finger, pressing the mask so that it stays flat and well in contact with the palate. At the same time, the index finger keeps the tongue out of the way.

Step 3 Push the mask so that it follows the curvature of the pharynx until it enters the epipharynx. The index finger now lies almost horizontal.

Remove the index finger while fixing the connection tube with the other free hand. Then move the mask further downwards until it's tip lies at the entrance of the esophagus.



Step 4 Inflate the cuff with the correct amount of air, thereby allowing the connecting tube to move out slightly as the mask settles. Do not move the mask from its place during cuff inflation. Do not over inflate the cuff. Because of its anatomical shape it should now seal the larynx from the pharynx.

Step 5 Connect to the resuscitator bag and ventilate the patient. Confirm by auscultating over bilateral apices, bases and epigastric region.



Cuff inflation volumes:

Size 3 - up to 20 ml

Size 4 - up to 30 ml

Size 5 - up to 40 ml

Securing the LMA

Do not attach other equipment to the LMA until the cuff has been inflated to avoid accidental displacement of the LMA. The insertion of a bite block to guard against compression of the airway tube is strongly recommended. This can either be in the form of a wad of rolled gauze or an oropharyngeal airway.

Secure the LMA in place with adhesive or cotton tape, ensuring that the airway connector is directed downwards towards the base of the chin. The black line on the spine of the airway tube should face the nose.

The airway tube connector is compatible with all standard ventilation equipment – 15 mm fittings. In the absence of equipment to inflate the lungs, use direct mouth-to-tube inflation.

e. Complications

- 1. Aspiration
- 2. Gastric insufflation
- 3. Partial airway obstruction
- 4. Coughing
- 5. Laryngospasm

a. Maintenance

 Always clean the mask and especially the lumen directly after use with water and soap and a soft brush. Do not use detergents because they may damage the mask. • The mask must be sterilized after cleaning. It is important that the cuff is fully deflated immediately prior to sterilizing. The mask can be sterilized up to a temperature of 136°C. Ensure that the mask is maximally deflated before sterilizing and that no water enters the cuff to prevent damage to the mask.

Endotracheal Intubation

This is an advanced airway technique, which consists of the passage of a tube directly into the trachea. The advantages are to:

- 1. Isolate the airway, preventing aspiration of material into the lower airway.
- 2. Facilitate ventilation and oxygenation.
- 3. Facilitate suctioning of the trachea and bronchi.
- 4. Prevent wasted ventilation and gastric insufflation during positive-pressure ventilation.

Indications for Endotracheal Intubation

- 1. Inability to ventilate the unconscious patient.
- 2. Patient is unable to protect his own airway (coma, areflexia or cardiac arrest.
- 3. For prolonged artificial ventilation.

Endotracheal intubation during cardiac or respiratory arrest should be performed as soon as possible by trained personnel. Adequate ventilation and oxygenation should be provided before and between attempts to intubate. Since there is no ventilation and oxygenation during attempts to intubate the manoeuvre should be completed within 30 seconds. ETT intubation by inexperienced personnel may cause injury to the oropharynx and cause unnecessary interruption in CPR. Hence intubation should be done by the most experienced personnel with minimal interruption to chest compression.

Preparation for Endotracheal Intubation

As with all medical procedures, all equipment must be assembled and tested prior to attempts to intubate. Below is a list of the equipment needed:

- 1. Laryngoscope handle, blades of different sizes
- 2. Endotracheal tubes
- 3. ETT introducer (stylet)
- 4. 10 cc disposable syringe
- 5. Water soluble lubricant
- 6. Gauze
- 7. Adhesive tape to secure ETT
- 8. Suction apparatus
- 9. Bag-valve-mask device
- 10. Oropharyngeal airway
- 11. Gloves
- 12. Magill forceps



1. Laryngoscope

- This device is used to visualise the glottis.
- There are two types of blades,
 - 1) a curved blade (MacIntosh) and
 - 2) a straight blade (Miller, Wisconsin, Flagg etc.); it is a matter of personal preference as to which one is used.
- In general, the curved blade is often used for adults and the straight blade type is preferred in the paediatrics age group.
- There must be available bulbs and batteries for replacement.

2. Endotracheal Tube (ETT)

- The proximal end has a standard 15mm connector that will fit the devices for positivepressure ventilation. The distal end of the tube has a cuff that is linked to a pilot balloon. Inflating the pilot balloon will inflate the cuff. The cuff must always be tested for integrity prior to insertion.
- The size of the ETT refers to its internal diameter in millimetres. It is indicated on the ETT and on the pilot balloon in some brands.

Adult female: 6.5-7.5

Adult male: 7-8

Or as ordered by doctor

- The length of the tube from the distal end is indicated at several levels in centimetres.
- Lubricate the lower portion of the ETT and cuff below the circular ring mark that is usually just above the cuff.
- Tubes of all sizes as well as uncuffed tubes for the paediatric age group must always be available.

3. ETT Introducer (Stylet)

- A malleable stylet, preferably coated in plastic may be inserted through the ETT.
- It helps to shape the ETT to the desired configuration, thus facilitating the insertion of the tube into the larynx and the trachea.
- The end of the stylet must always be recessed at 1-1.5 cm from the distal end of the ETT.
- Lubricate about 1/3 of the stylet with a water-soluble lubricant prior insertion into the FTT

4. Suction apparatus

- The suction unit must be checked that it is working well.
- Ideally, there should be one pharyngeal rigid suction tip (Yankauer) or a large bore catheter for removal of particles and thick fluids.
- There must be a sterile tracheal suction catheter for suctioning the ETT after it is in place.

Role of the assistant in endotracheal intubation

In the specialised areas of the hospital e.g. emergency department, ICU, where doctors are available, endotracheal intubation is usually performed by them. The nurse assists with the following:

- 1. Check all equipment
- 2. Select the appropriate size tube as indicated by the doctor
- 3. Lubricate the ETT and ETT Introducer
- 4. Position the patient
- 5. Prepare suctioning equipment and perform suctioning when necessary
- 6. Inflate the cuff upon the order of the doctor
- 7. Secure tube with tape
- 8. Measure and document exposed length of ETT

Technique of Endotracheal Intubation

Positioning the Patient

- The key to a successful intubation is proper patient positioning.
- Three axes, those of the mouth, the pharynx, and the trachea, must be aligned in order to achieve direct visualisation of larynx.
- To accomplish this, the head is extended and the neck flexed (i.e. the "sniffing position"). The head must not be allowed to hang over the end of the bed/trolley.
- In many cases it is helpful to place layers of linen under the patient's occiput to elevate it a few inches above the level of the bed. This provides proper flexion of the neck; extension of the head is affected by the individual performing the intubation.



Pre-oxygenation

- Use the BVM to pre-oxygenate the patient, preferably to SaO2 >95%.
- If necessary suction the mouth and pharynx prior to intubation.
- It is important to remember that each intubation attempt should be no more than 30 seconds.



Laryngoscopy

- The mouth is opened with the fingers of the right hand.
- The laryngoscope is held in the left hand and blade inserted in the right side of the mouth displacing the tongue to the left.
- The blade is then moved slightly towards the midline and advanced to the base of the tongue.
- Simultaneously, the lower lip is moved away from the blade using the right index finger. Avoid pressure on the lips and teeth.
- By exerting upward traction of the handle the glottic opening is visualised. The handle must not be used with a prying motion, and the upper teeth must not be used as a fulcrum.
- When the curved blade is used, the tip of the blade is advanced into the vallecula (i.e. the space between the base of the tongue and pharyngeal surface of the epiglottis).
- The tip of the straight blade is inserted under the epiglottis.
- The routine use of cricoids pressure is not recommended as it may obstruct the trachea. If used to assist with cord visualization, the pressure should be adjusted, relaxed or released if it impedes ventilation or intubation.

Inserting the ETT

- An assistant may pull down patient's right lip corner to help the passage of ETT into the oral cavity.
- The tube is advanced through the right corner of the mouth and under direct vision, passed between the vocal cords.

Removing the stylet

- If a stylet has been employed, it should be removed from the tube at this time before the ETT reaches its final position.
- The tube should be advanced to 1-2.5 cm distal to the vocal cord. This will place the tip of the tube about halfway between the vocal cords and the carina.
- This position allows for some displacement of the tip during flexion or extension of the neck without extubation or displacement into a main stem bronchus.





Advance the tip of the curve blade into the vallecula





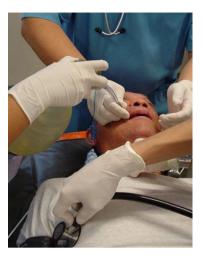
Confirming and securing the ETT

- Following inflation of the cuff with 5-8ml of air, the patient is ventilated with resuscitator bag or ventilator.
- · Connect the ETT to the resuscitator bag.
- ETT placement is confirmed by auscultation over:
 - epigastric region
 - bilateral apices of the lungs
 - bilateral lateral aspect of the chest at the midaxillary line

and / or

- attach end-tidal CO2 detector, note colour change OR obtain continuous ETCO2 tracing
- Note ETT marking at the patient's lip. The marking will usually be 21 cm (for women) and 23 cm (for men).
- Wipe away secretions and secure ETT with adhesive tapes.
- If the face is wet, or patient has a mustache/beard, the ETT should be secured with a cloth tape tied round the back of the head.
- Suction and ventilate as indicated.
- Ventilate at the rate of 10-12 breaths per minute.
- Finally, a CXR is performed to confirm tube placement.

In the event of oesophageal intubation, remove the ETT immediately and pre-oxygenated the patient before the next intubation attempt again.



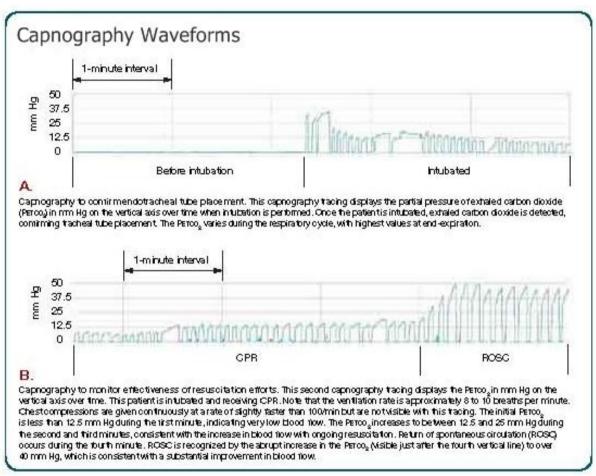


marking



ETT secured with a tie

Continuous quantitative waveform capnography (ETCO2) is recommended for confirmation of ETT placement, monitoring of intubated patients for ETT dislodgement and CPR quality and prognostication. A continuous waveform capnography serves as a physiologic confirmation of endotracheal intubation and effective chest compressions. A sudden increase in ETCO2 indicates return of spontaneous circulation (ROSC). CPR should be halted and pulse check should be performed. On the other hand, ETCO2<10 mm Hg after 20 minutes of CPR indicates poor prognosis and CPR should be terminated.



Highlights of the 2010 AHA Guidelines for CPR and ECC

Possible Complications of Endotracheal Intubation

- 1. Trauma to the:
 - oral cavity (lips, tongue, teeth).
 - pharyngeal and trachea mucosa.
 - larynx and its vocal cords.
- 2. Pharyngeal-oesophageal perforation.
- 3. Intubation of pyriform fossa.
- 4. Vomiting and aspiration of gastric contents.
- 5. Stimulation of pharynx and larynx in a non-arrest patient resulting in release of epinephrine and norepinephrine giving rise to hypertension, tachycardia and arrhythmia.
- 6. Intubation of the right main bronchus.

Trouble-shooting post-intubation desaturation

If the patient desaturates while on mechanical ventilation, do the following:

- 1. Check that the O2 saturation probe is on the finger and that the wave form is good. If the SaO2 is still low, then
- 2. Disconnect the ETT from the ventilator circuit, connect to a bag and manually bag the patient. If the SaO2 improves, the problem is with the circuit. Check or change it. If the SaO2 does not improve, then the problem could be from the ETT or more distal.
- 3. Check the ETT position because it may have dislodged into the right main stem bronchus or oesophagus. Auscultate to ensure equal air entry. Check end-tidal CO2 tracing. Order a chest Xray if needed.
- 4. Suction the ETT and clear the secretions
- 5. If there is hypotension, deviated trachea and unilateral decreased air entry, hyperresonance to percussion, a tension pneumothorax could have developed. Perform immediate needle decompression in the second intercostal space mid-clavicular line. If there is a rush of air, proceed to insert a chest tube on the ipsilateral side. Tension pneumothorax is a clinical diagnosis and there is no time to do a chest Xray first.
- 6. Assess if the patient has woken up and is biting the ETT or fighting the ventilator. Sedate and paralyse the patient if needed.

Cricothyrotomy

This technique allows rapid access to the airway for temporary ventilation and oxygenation in patients with difficult airway. It creates an opening in the cricothyroid membrane with a scalpel.

Technique of Cricothyrotomy

- 1. The area is cleansed.
- 2. A horizontal incision is made at the level of the cricothyroid membrane.
- 3. The scalpel handle is inserted through the incision and rotated 90 degrees.
- 4. A paediatric ETT, the largest possible, is inserted through this opening.
- 5. Ventilation is done with a bag-valve unit.
- 6. This technique should not be used beyond 45 minutes as it will cause carbon dioxide retention.

Complications associated with Cricothyrotomy

- 1. Haemorrhage
- 2. False passage
- 3. Perforation of the oesophagus
- 4. Subcutaneous and mediastinal emphysema

Tracheostomy

Tracheostomy should ideally be performed under controlled condition in the operating room by a skilled doctor after the airway has first been secured by an endotracheal tube or cricothyrotomy.

Summary

Appropriate airway management is critical in the resuscitation of a collapsed patient. All nursing staff must be conversant in basic airway management and competent in assisting in and maintenance of advanced airway management.

Chapter 5. ELECTRICAL THERAPY

A. Defibrillation

Introduction

Ventricular fibrillation (VF) is the most frequent initial rhythm in sudden cardiac death and defibrillation is the only definitive treatment. Defibrillation passes an electric current through the heart to depolarise all abnormal pacemakers, thus abolishing all electrical activity temporarily. This enables the sino-atrial node to resume function as the primary pacemaker and re-establish sinus rhythm. The sooner the shock is applied after the onset of VF, the greater the chance of success and survival.

Synchronised direct current (DC) shock (or synchronised cardioversion) is indicated for patients in whom the tachyarrhythmia has caused hemodynamic impairment or when it is not responsive to anti-arrhythmic drug therapy.

There are no major changes recommended regarding defibrillation and cardioversion in the 2015 AHA Guidelines for CPR and ECC (Emergency Cardiovascular Care). Emphasis on early defibrillation integrated with high-quality, uninterrupted CPR is still key to improving survival from sudden cardiac arrest.

Defibrillation (Non-Synchronised DC Shock)

Indication – VF and pulseless Ventricular Tachycardia (VT)

The presence of a pacemaker and implanted cardioverter-defibrillator are not a contraindication to defibrillation. Care must be taken to ensure the paddle or multifunction electrode pad are applied at least 4 fingers breadth away from the implanted pacemaker

Paddles / Multifunction Pads

When using manual paddles, one will open a package of conduct gelled pads "defib pads" and apply these gelled pads to the patient's chest and place the defibrillation paddles as shown in Fig 5.1.

In the case of multifunction pads placement, carefully pull off the protective cover from the pads and connect the cable accordingly. These pads have diagrams that instruct users on the correct placement of pads.

Check the expiry date of the gelled pads and multifunction pad before use.



Defib Pads Multifunction Pads



Placement of Paddles / Multifunction Pads placement

For ease of placement and teaching, the anterolateral pad position is a reasonable default electrode placement.

- 1. Anterolateral position
- One (sternal) to be placed to the right of the upper sternum just below the right clavicle.
- One (apex) to the left of the nipple in the mid axillary line.

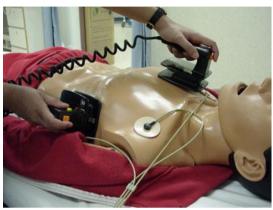




Fig. 5.1 Anterolateral placement of defibrillator paddles (L) and multifunction pads (R)

- 2. Anterior-posterior position
- One pad is positioned anteriorly over the precordium just to the left of the lower sternal border.
- The other is positioned on the rear of the patient behind the heart.



Fig. 5.2 Anterior-posterior placement of defibrillator paddles

How much electric current actually passes through the heart during defibrillation is dependent on

- 1. The energy setting of the defibrillator (measured in joules).
- 2. The amount of resistance the patient's thorax puts up against the current, known as **transthoracic impedance**, which is in turn dependent on
 - electrode (paddle) size.
 - contact material between the paddle and skin.
 - distance between the paddles.
 - amount of pressure the paddles are applied to the chest.

If the transthoracic impedance is high, a shock set at too low an energy level may not generate enough current to defibrillate successfully. To reduce transthoracic impedance, it is important to use a conductive gel, cream or pads between the paddles and the chest, and to apply hand-held paddles firmly to the chest.

Biphasic Waveforms and Energy Levels

Biphasic waveform defibrillators are used by most Singapore hospitals today. In biphasic cardioversion, the current flows in a positive direction for a specified duration, after which it reverses and flows in a negative direction.

Current research confirms that biphasic shock energies at or less than 200J are safe and effective for VF termination. In general, the energy does not need to be increased but the defibrillator's manufacturer's recommendations should be followed.

Recommended Joules for Biphasic Defibrillation

Biphasic defibrillation for Adult	Initial (1st) shock and all subsequent shocks – 120 to 200 joules or energy level recommended by manufacturer/hospital guidelines
Defibrillation for Infant and Children	2, 4, 4 Joules/kg (biphasic)

Procedure

- 1. Confirm VF by checking more than one lead and/or increase in ECG size. In some instances, coarse VF can be present in some leads while very small undulations or a straight line is present in others.
- Apply defibrillation pads on the patient's chest or apply conduction pads to the patient's chest and place the defibrillation paddles as shown in Fig. 5.1.
 Application of pads to the chest wall must be done quickly with minimal interruptions to chest compressions.
- 3. Shave if there is a lot of chest wall hair to ensure pads can effectively stick to the chest.
- 4. Remove any transdermal medication as they may overlap with the defibrillation pads. This may result in sparking and potential skin burns.
- 5. Select the energy required. Make sure the synchronizer is OFF.
- 6. Press the charge button. The monitor will display the energy level as selected and sound an alarm when the capacitor is charged.

- 7. Continue chest compressions while the defibrillator is charged, this will minimise the pre-shock pause.
- 8. Remove the paddles from their sockets and place them firmly on the patient's chest.
- 9. Make sure all personnel stand clear of the patient. Ventilation must be stopped momentarily during defibrillation.
- 10. Deliver the shock by depressing both discharge buttons simultaneously.
- 11. Commence CPR immediately following defibrillation for 2 minutes.
- 12. If patient remains in VF or pulseless VT, please refer to Algorithm 1 in chapter 6 for further management.
- 13. Once the VT or VF is converted, note the rhythm on the monitor screen and print one rhythm strip.

Synchronised Cardioversion or DC Shock

Synchronized cardioversion is recommended for fast atrial fibrillation, fast atrial flutter and supraventricular tachycardia. Synchronization means that the defibrillator will deliver the shock near the peak of R wave of the QRS complex to avoid the risk of inducing ventricular fibrillation.

Procedure

- 1. Explain procedure to patient and the potential need for repeating the procedure.
- 2. Remove dentures if necessary.
- 3. Prepare intravenous sedation and analgesia as ordered.
- 4. Monitor the patient on an ECG lead (usually lead 2), which has a tall prominent R wave to ensure that the shock is triggered off at the right time. Ensure that intravenous line is working.
- 5. Administer 100% supplemental oxygen.
- 6. Ensure that resuscitative drugs, equipment and suction apparatus are ready.
- 7. Activate synchronizer switch. Run an ECG strip to confirm the countershock is programmed to occur at the 'R' wave. If the 'R' wave is not prominent enough, the defibrillator will not pick up the 'R' wave and no current will be discharged when the discharge buttons are activated.
- 8. Alert all personnel to stand clear prior to the synchronized DC shock.

Safety Precautions

- 1. The patient must not be in contact with wet or metallic surface. These can conduct electric currents to the rescuer.
- 2. Floor surface should be clean and dry to prevent electrical hazard.
- 3. Avoid coating the paddles with excessive ECG gel to prevent spark escape.
- 4. Ensure that there is no direct oxygen source directed at the patient's chest e.g. If patient is on oxygen mask ensure that it is on the face and not on the chest. If BVM is performed, remove resuscitator away from patient.
- 5. Members of the resuscitation team must not be in direct contact with the patient during defibrillation. The operator must shout "All clear!" before defibrillating.
- 6. All charging and discharging of the capacitors must be done with the paddles on the patient's chest or in the defibrillator's sockets. The paddles must not be waved about in the air.

Complications

- 1. VF after cardioversion.
- 2. Pain over the anterior chest.
- 3. Skin irritation from the defibrillation gel.
- 4. Paddle burn marks due to insufficient gel applied or multiple DC shocks.

Nursing Responsibilities in the Care of Defibrillators

- 1. Charge the defibrillator when it is not in use.
- 2. Adequate ECG monitoring discs, recorder paper and defibrillator pads must be available.
- 3. ECG cable and leads are in good working condition.
- 4. Perform defibrillation function test according to the manufacturer's instructions for the defibrillator.

Summary

Early defibrillation enhances patient's chance of recovery from VF and pulseless VT. Nurses have to be familiar with the procedures of synchronized and non-synchronized shocks and the safety precautions to be taken.

B. Automated External Defibrillation

Overview of Automated External Defibrillators

The generic term "automated external defibrillators" (AED) refers to external defibrillators that incorporate a rhythm analysis system. Some devices are "fully automated" and some are "semi-automated" or "shock-advisory" defibrillators. All AEDs are attached to the patient by two adhesive pads and connecting cables. The pads serve 3 functions –

- to record the rhythm
- · to deliver the electric shock and
- to pace the heart

A fully automated defibrillator requires the operator to attach the defibrillatory pads and turn on the device. The AED then analyses the rhythm. If VF (or VT above a pre-set rate) is present, the AED charges its capacitors and delivers a shock. Semi-automated or shock-advisory devices require the operator to press an "analyse" control to initiate rhythm analysis and a "shock" control to deliver the shock. The shock control is pressed only when the device identifies VF and advises the operator to press the shock control.

Operational Steps

All AEDs can be operated using four simple steps:

- 1. Turn on the power
- 2. Attach the device.
- 3. Initiate analysis of the rhythm.
- 4. Deliver the shock if indicated.

Interruption to CPR during analysis

Emergency personnel must not touch the patient while the AED analyses the rhythm, charges the capacitors and delivers the shocks. Chest compression and ventilation must cease while the device is operating. This allows accurate analysis of the rhythm and prevents accidental shocks to the rescuers. The time from activating rhythm analysis to delivery of a shock averages 10 to 15 seconds.

Minimise interruption to CPR

- Continue CPR while applying pad, stop CPR when about to discharge defibrillator
- 2. Commence CPR for 2 mins immediately following defibrillation before checking rhythm.

Advantages of AEDs

- 1. The AED interprets the rhythm and does not require a person to do so.
- 2. The training for AED users only involves recognition of cardiac arrest, proper attachment of the device and adherence to the memorized treatment sequence.
- 3. This approach allows "hands off "defibrillation.
- 4. Adhesive defibrillation pads may also offer consistently better paddle placement during a prolonged resuscitation attempt.
- 5. Even with minimally trained personnel, early defibrillation is practical and achievable

AED Use in Children and Infants

To defibrillate a child of 1-8 year old with an AED, the rescuer should use a paediatric dose-attenuator system if one is accessible. The dose attenuator will decrease delivered energy to a dose more suitable for a child of 1-8 year old (i.e 50-75J). If the AED does not have a paediatric dose-attenuator system, the rescuer should use a standard AED.

For an infant (<1 year old), a manual defibrillator is preferred. If a manual defibrillator is not available, a defibrillator with a dose attenuator is mandatory.

C. Cardiac Pacing

A pacemaker is a device that delivers an artificial stimulus to the heart causing electrical depolarization followed by a cardiac contraction. External pacing is for temporary emergency use until transvenous pacing or other definitive therapy can be initiated. In most modern defibrillators, the external pacemaker is an optional device that can be attached to the machine. It consists of a pulse generator, a cable and two electrodes to be attached to the patient. Each electrode has a central area of conductive jelly and an outer area of adhesive.

Indications

It is used to treat situation e.g. complete heart block whereby bradycardia is causing life-threatening symptoms or patient is not responding to drug therapy.

Pulse Generator

Components of the generator are:

- 1. On-off switch
- 2. Pacing rate setting
- 3. Output current (in milliamperes) 40 to 100 mA
- 4. Sensor to sense intrinsic QRS activity
- 5. Mode of pacing
- a) Fixed the pacemaker will deliver continuous regular electrical stimuli regardless of any natural intrinsic stimuli.
- b) Demand the pacemaker will sense intrinsic QRS activity and inhibit the pacing stimulus. If QRS activity is not sensed, the pacemaker will pace at the selected rate.

Electrode Placement

- 1. Anterior-Posterior Position
- Negative electrode left anterior chest, halfway between xiphoid process and left nipple line.
- Positive electrode on left posterior chest, beneath the scapula and lateral to the spine.
- 2. Anterolateral Position
- Negative electrode on left chest and axillary over fourth intercostal space.
- Positive electrode on right chest, subclavicular area.



Anterolateral Position

Chapter 6. ARRYHTHMIAS TREATMENT ALGORITHMS

Introduction

Cardiac arrest occurs with one of four arrhythmias: ventricular fibrillation (VF), pulseless ventricular tachycardia (VT), pulseless electrical activity (PEA) and asystole. When faced with a cardiac arrest, doctors and nurses involved in the resuscitation must recognize the rhythm and treat it immediately. The treatment of each arrhythmia entails a complex set of decisions with which all personnel must be familiar. Hence, the need for treatment algorithms which facilitates comprehension and execution.

1. VF / Pulseless VT

The VF/ pulseless VT algorithm is the most important for adult because majority of cardiac arrests collapse in VF or pulseless VT. When VT does not generate a pulse, it is treated as for VF. The definitive treatment for VF/pulseless VT is defibrillation. VF and pulseless VT has the best prognosis of the 4 cardiac arrest rhythms. Over 80% of people in whom defibrillation will be successful have achieved this by one of the first 3 shocks. Drug therapy is only an adjunct. The emphasis therefore is on early recognition, early CPR and early defibrillation.

After a shock has been delivered, the ECG monitor may show a straight (isoelectric) line for a few seconds. This does not mean that the rhythm has reverted to asystole because VF/pulseless VT may appear shortly. Only when a "straight" line persists for more than one sweep (width) of the ECG monitor, then should treatment for asystole be started.

Transdermal patches should be removed and the defibrillator paddles should be 4 finger breadths away from implanted pacemakers.

2016/2018 New Guidelines

A. Energy Level

The biphasic energy level is usually between 120J to 200J, depending on the manufacturer/hospital recommendation.

B. Pulse Check

Pulse check immediately after delivering a shock is de-emphasized. Resume chest compression immediately post shock, regardless of what the patient's rhythm is, continue chest compression for 2 mins. This is because immediately after successful defibrillation, cardiac contraction is poor and external chest compression will augment cardiac output until the heart resumes normal contraction. After 2 mins, or earlier if the patient shows signs restored perfusion (eg. moving limbs, moaning) stop chest compression, check rhythm and pulse.

6-1

Refractory VF/Pulseless VT (Fig. 6.1)

The algorithm for refractory VF / pulseless VT management is as follows:

1. Shock-CPR cycle

Start with basic life support by assessing responsiveness, activating code team, calling for defibrillator, opening the airway, checking pulse and performing chest compression. Giving 2 breaths is now omitted to minimize delay in starting chest compression.

The moment the defibrillator arrives, attach the leads. Interrupt chest compression briefly to check the rhythm. If the monitor shows VF or VT, defibrillate once with 120J to 200J, depending on the manufacturer/hospital recommendation for biphasic defibrillators.

After the first shock continue chest compression for 2 mins before assessing rhythm. If the patient remains in VF or VT, shock again and continue chest compression for 2 min. This should be repeated in a shock-CPR, shock-CPR cycle until there is spontaneous return of circulation or the rhythm changes to PEA or asystole. Chest compression should be continuous and only briefly interrupted to analyse rhythm and deliver shock.

2. IV access & drugs

Concurrently a member of the team should establish IV access and start IV normal saline. For refractory VF/pulseless VT (ie. no cardioversion after the first shock) drugs may be given immediately once IV access is established and no longer needs to be coordinated with shocks. After any drug is given, chest compression needs to be done to allow the drug to circulate. Adrenaline is given IV 1mg (1:10000) rapid bolus every 3-5 min.

2a. Anti-Arrhythmic Agents

Anti-arrhythmic agents are given at the same time as the first adrenaline. Amiodarone is administered IV 300 mg rapid bolus. Amiodarone may be repeated once after 3-5 mins if VF/pulseless VT is refractory and is administered IV 150 mg rapid bolus. Lignocaine may be given as an alternative to amiodarone and is given IV 50-100 mg (1-1.5mg/kg) rapid bolus repeated once at the same dose after 3-5 mins if VF/pulseless VT is refractory. Thereafter only adrenaline is repeated.

After return of spontaneous circulation, antiarrhythmic should be continued as an infusion. Either IV amiodarone infusion 1 mg/min x 6 hrs, then 0.5 mg/min x 18 hrs *OR* IV lignocaine infusion 1-4 mg/min x 24 hrs.

3. Definitive airway

Other members of the resuscitation team should secure the airway and breathing with intubation. If unable to intubate, support with BVM ventilation. Intubation attempts should not interrupt chest compressions or defibrillation.

2. Pulseless Electrical Activity (Fig. 6.3)

Pulseless electrical activity (PEA) means that when the ECG monitor shows a non-VF/VT and non-asystole rhythm, this rhythm is unable to generate any cardiac output and the patient is pulseless. Cardiac compression, airway management, ventilation and intravenous access must be instituted and adrenaline given.

In the absence of any reversible cause, PEA has a poor prognosis. Only adrenaline needs to be given every 3-5 minutes in PEA. Atropine is no longer recommended for routine use in the management of PEA because it is unlikely to have a therapeutic benefit. Sodium bicarbonate is not longer recommended for prolonged cardiac arrest. It may still be given if the cause of the PEA is tricyclic antidepressant poisoning or hyperkalaemia.

3. Asystole (Fig. 6.3)

Asystole generally carries an extremely poor prognosis. Sometimes fine VF may be mistaken for asystole and therefore it is important to check with another lead, increase the ECG size (gain) or stop ventilation for a few seconds to assess the rhythm. When in doubt, however, it is advisable to continue with chest compression and reassess after 2 minutes of CPR (5 cycles).

Besides cardiac compression, airway management, ventilation and establishment of intravenous access with administration of adrenaline.

After 15 to 20 minutes of resuscitation, the chances of recovery from asystole are dismal. Exceptions to this are cases of near drowning, poisoning, and less common locally, hypothermia, all of which should be excluded before resuscitation is abandoned. Decision for termination of resuscitation depends on established local hospital protocols. It is suggested that if end tidal CO2 is < 10 mm Hg after 20 mins of resuscitation (poor prognosis), resuscitation efforts can be stopped.

The key in management of cardiac arrest is to search and treat any reversible causes. The common reversible causes are listed in this table.

Causes	Treatment	Causes	Treatment
1. H ypovolemia	Fluid resuscitation	Tablets (drug overdose) / intoxication	Antidote
2. H ypoxia	Ventilation	2. T amponade, cardiac	Pericardiocentesis
3. H ydrogen ion – Severe Acidosis	Give sodium bicarbonate 1 mEq/kg	3. T ension pneumothorax	Needle compression of chest
4. H yperkalaemia	Remove potassium/dialysi s	4. Thrombosis (ACS, AMI)	ResuscitationRevascularisationPCI
5. Hypokalaemia	Replace potassium		,
6. H ypothermia	Warm patient	5. T hrombosis (pulmonary)	thrombolytics / Surgery

Summary

VF/pulseless VT when recognized and treated early affords the patient the best chance of recovery. It is thus important for health care professionals to be thoroughly familiar with its recognition and treatment.

Key changes in advanced cardiovascular life support for 2015 include the following:

- Continuous quantitative waveform capnography is now recommended for confirmation and monitoring of ETT tube placement and CPR quality.
- IV access, drug administration, and intubation, while still recommended, should not cause interruption to chest compressions or delay shocks.

Fig. 6.1 Algorithm1

Management of Ventricular Fibrillation (VF) / Pulseless Ventricular Tachycardia (VT)

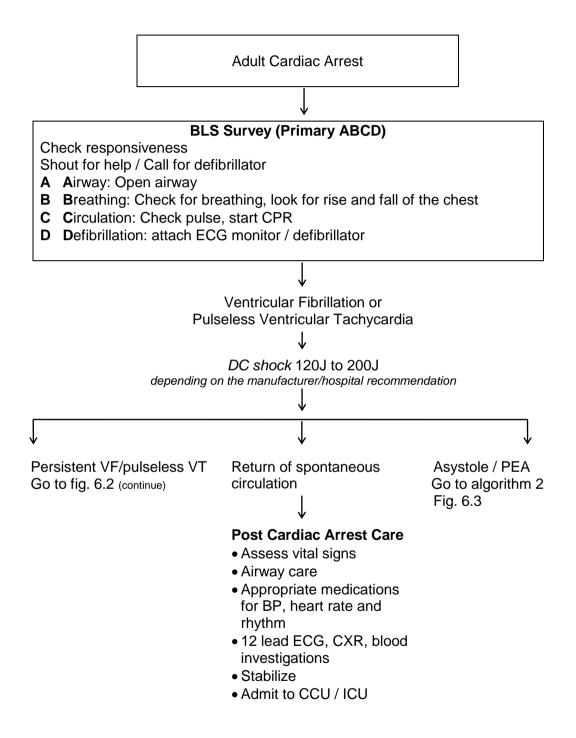
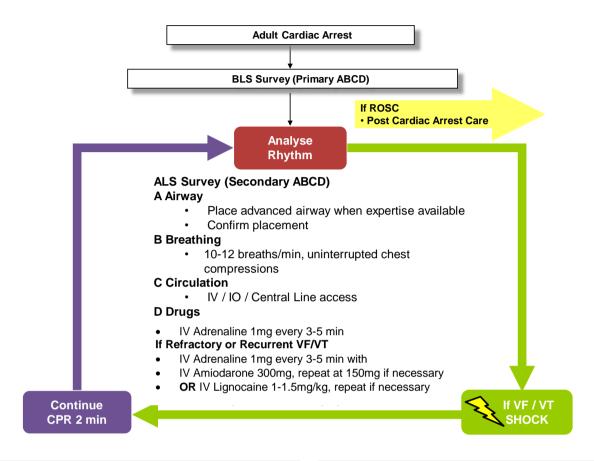


Fig 6.2 Algorithm1

Management of Ventricular Fibrillation (VF) / Pulseless Ventricular Tachycardia (VT)



Consider causes that are potentially reversible

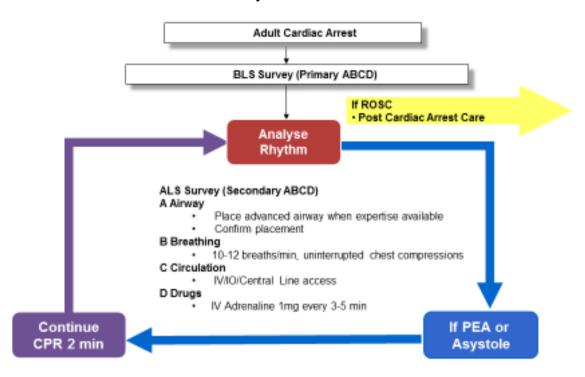
- **H**ypovolaemia
- Hypoxia
- Hydrogen ion acidosis Tension pneumothorax
- Hyper-/hypokalaemia
- Hypothermia
- "Tablets" (drug OD, accidents)
- Tamponade, cardiac
- Thrombosis, coronary (ACS)
- Thrombosis, pulmonary (embolism)

Note:

- Sequence is shock-CPR, shock-CPR etc. 1.
- In refractory VF (ie. failed first shock), both 2. drugs should be given together once IV is established and need not be coordinated with shock.
- 3. Drug administration needs to be followed by CPR to ensure it circulates.
- Post-cardiac arrest care includes targeted temperature management, avoid hyperoxia, amiodarone infusion 1mg/minx6 hrs, then 0.5mg/min x 18 hrs.

Fig. 6.3 Algorithm 2

Asytole and PEA



Consider causes that are potentially reversible

- **H**ypovolaemia
- "Tablets" (drug OD, accidents)
- Hypoxia
- Tamponade, cardiac
- Hydrogen ion acidosis Tension pneumothorax
- Hyper-/hypokalaemia
- Thrombosis, coronary (ACS)
- Hypothermia
- Thrombosis, pulmonary (embolism)

Note:

- Drug administration needs to be followed by 1. CPR to ensure it circulates.
- 2. Post-cardiac arrest care includes - targeted temperature management, avoid hyperoxia.

Chapter 7. DRUGS AND DRUG DELIVERY SYSTEMS

Introduction

This chapter discusses the drugs used in resuscitation, acute myocardial infarction and other cardiac emergencies. These are presented in table 7.1 on the following pages. It is important for nurses to be familiar with the drugs commonly used in resuscitation, especially epinephrine, atropine, lidocaine, amiodarone, and those commonly used in post resuscitation care such as dopamine, nitroglycerine, verapamil and sodium bicarbonate.

A brief discussion on various drug delivery routes is outlined in the subsequent pages. Though peripheral cannulation is by far the commonest, nurses should be aware that there are alternative routes to administer drugs.

It is important to note that without treating the primary disease processes, trying to treat the secondary effects may be futile or even harmful.

Principles in Delivering Drugs

Drugs that have potent effect on blood pressure and heart rate:

- Should not be given as rapid bolus except in cardiac arrest patient i.e. should be given as slow bolus or via infusion.
- Should be tapered / tailed down gradually under ECG and blood pressuring monitoring.
- Should commence with the lowest dose to achieves the desired effect, this will constitute optimal dose.
- After each drug administration flush the line with 10-20mls of normal saline and continue 1-2 minute of chest compression for the drugs to reach the central circulation or before the next shock is given, if needed.
- In the event intravenous access is difficult, access via subclavian or internal jugular central venous line (CVL) or intraosseous (IO) route are alternatives.
- Drug delivery via endotracheal tube route is no longer recommended because drug levels achieve are suboptimal and doses required to achieve blood levels similar to the IV route are about 3 to 10 times as much.



Fig. 7.1 Easy IO™ "gun" and manual IO needle

American Heart Association (AHA), 2015, emphasizes the delivery of high-quality, uninterrupted CPR and early defibrillation for shockable rhythm. Vascular access and drug administration, while still recommended, should not cause significant interruptions in CPR and delay shocks.

Consider the following two scenarios and their rationale for drugs administration.

Cardiac arrest	Pulse is present
Drugs are used to: Help start the heart Preserve coronary and cerebral circulation	Drugs are used to optimize: Cardiac output by optimizing volume, pump function and heart rate Coronary circulation
	The environment for cardiac function

Cardiac Arrest Drugs

The table provides a sample of drugs for the various cardiac arrest situations

VF/ Pulseless VT	Pulseless Electrical Activity	Asystole
Adrenaline Amiodarone Lignocaine Magnesium Others	Adrenaline Others e.g., potassium, calcium and antidotes	Adrenaline

Sodium bicarbonate – routine administration of sodium bicarbonate after prolonged cardiac arrest is not recommended (AHA Guidelines 2010). Administer 50mmol if cardiac arrest is associated with hyperkalaemia or tricylic antidepressant overdose. If continuous infusion of sodium bicarbonate is used, regular blood gas monitoring will be required to guide therapy.

In patient with pulse and blood pressure, drugs are used to optimize cardiac function.

Optimization Drugs

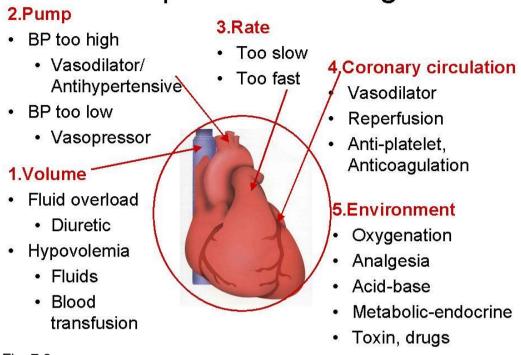


Fig. 7.2

1A. Pump Optimization Drugs: Vasodilator / Antihypertensive

a) Nitroglycerin (GTN)

- Is a venous and arterial dilator.
- Reduces preload, reduces left ventricular filling pressure.
- Is the drug of choice in acute heart failure / acute pulmonary oedema (APO) + ischaemic heart disease (IHD) / acute coronary syndrome (ACS).
- Interaction with Viagra.

b) Sodium Nitroprusside

- Is an arterial and venous dilator.
- Has the potential to induce myocardial ischaemia.
- Is the drug of choice in hypertensive emergencies.
- c) **ß Blockers** drug name usually ends with "olol" e.g.: propranolol, atenolol.
- Reduces heart rate.
- Reduces force of myocardial contractility.
- Should be used with caution in asthma / COLD, & congestive heart failure.

1B. Pump Optimization Drugs: Vasopressor

Catecholamines: "...aline" e.g: noradrenaline

- Common effects: increases BP, heart rate, myocardial contractility.
- Exclude hypovolaemia before using vasopressor to increase pump action/ increase BP.

Adrenaline

Improves coronary and cerebral perfusion pressure*.

- Given as rapid bolus only in cardiac arrest*.
- Given via infusion for symptomatic bradycardia.
- It should be given via a central line

Noradrenaline

- Is the preferred vasopressor for septic & neurogenic shock.
- It should be given via a central line

Dopamine

- Low dose 1-2 μg/kg/min: may not change heart rate or BP.
- Medium dose 2-10 µg/kg/min: increases heart rate and BP.
- High dose 10-20 μg/kg/min: increases heart rate and BP.
- A vasopressor for hypotension from (a) bradycardia (b) after return of spontaneous circulation

Dobutamine

- 2-20 µg/kg/min: increases BP, less tachycardia than dopamine and noradrenaline
- A vasopressor for hypotension with (a) pulmonary oedema, (b) left ventricular dysfunction

2A. Rate Optimization Drugs for Tachycardia

Common effects: reduces heart rate and blood pressure*.

Given as rapid bolus only in cardiac arrest, otherwise give as infusion for tachycardia.

Lignocaine*

- Use for VF, VT, wide complex tachycardia of unknown origin.
- Numbness of mouth and digits is a sign of toxicity.

Amiodarone*

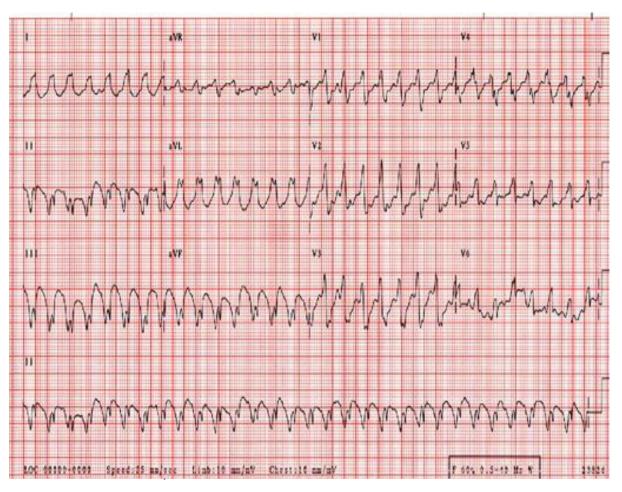
- Use for VF, VT, SVT, atrial fibrillation-flutter.
- Reduces clearance of warfarin, digoxin.

Magnesium*

• Drug of choice for Torsades de Pointes.

All these three drugs can be given as rapid bolus in VF or VT cardiac arrest but should be administered as slow bolus or infusion when used for tachycardia patients with pulse.

Tachycardia with Wide QRS: Amiodarone, Lignocaine

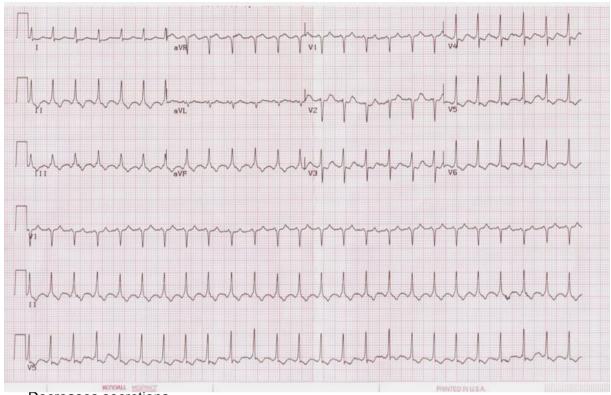


- Avoid concomitant use with ß Blockers.
- Do not use in Wolf-Parkinson-White syndrome.

Beta blockers

Use to slow down narrow complex tachycardia

Tachycardia with Narrow QRS: Adenosine, Verapamil, Diltiazem



- Decreases secretions
- No longer recommended in asystole and PEA cardiac arrest (AHA Guidelines 2010).
- Large doses needed for acute cholinergic poisoning (organophosphate).
- Side effects: similar to adrenaline, can also cause seizure, respiratory failure.

3. Coronary Circulation Optimization Drugs

3.1 Vasodilator

• GTN

3.2 Reperfusion

• Thrombolytic

3.3 Antiplatelet

- Non-specific aspirin
- Specific Clopidogrel (Plavix)
 - Glycoprotein IIb/IIIa inhibitors (GP IIb/IIIa): Risk of haemorrhage

3.4 Anticoagulant

- Heparin
- Low molecular weight heparin e.g. fraxiparine

4. Environment Optimization Drugs

4.1 Oxygen

4.2 Morphine

• Overdose may be reversed with nalxone

4.3 Sodium Bicarbonate (NaHCO3)

- Use in hyperK, tricyclic overdose, pre-existing acidosis
- Do not mix with other drugs
- Side effects: hyperosmolarity, hypernatremia
- CNS acidosis (Paradoxical alkalosis)
- should not be used unless blood gases show severe metabolic acidosis (PH <7.35)

4.4 Calcium

• Use in hyperK, hypoCa, Ca-channel blocker overdose

Summary

In cardiac arrest, drug administration is secondary to other interventions. Basic cardiopulmonary resuscitation (CPR), defibrillation when indicated and airway management must be the rescuer's first priorities. When a pulse is present, drugs are used to:

Optimize

- a. Cardiac output by optimizing
- Volume
- Pump
- Rate
- b. Coronary circulation.
- c. Environment for cardiac function.

The key to management is to identify the primary disease process(es) and use the appropriate drugs, if any, to optimize the deranged process(es).

• Table 7.1 Emergency Cardiac Drugs

(the doses and dilution depends on individual institutions protocols)

Drug	Main Uses	Therapeutic Effects	Dilution / Routes of Administration	Usual Dosages	Side Effects	Contraindications	How Supplied	Comments	Formatted Table
Epinephrine Adrenaline	1st drug in cardiac arrest Indications: VF Pulseless VT PEA, Asystole To maintain heart rate and/or arterial BP	Heart pumps harder ↑ contractile force of the heart - ↑ cardiac output Heart pumps faster - ↑ conduction of SAN, AVN and ventricular → ↑ heart rate Peripheral vasoconstriction ↑ systemic vascular resistance	IV or IO push (1:10,000) Dilute 1mg with 9mls of NaCL 0.9% IV infusion	1 mg q 3-5 min during cardiac arrest resuscitation Infusion: 0.01-1 µg/kg/min to titrate according to blood pressure	Ventricular irritability tachycardia & PVCs Non-collapsed Patients Stroke, Acute MI	None when used in cardiac arrest. Use with caution in pregnant patients.	Preload syringes: -1:10,000, -0.1 mg/ml, 10 ml Ampoules: -1:1000, 1 mg/ml, 1 ml (not for bolus use)	Can be deactivated if mixed with alkaline solutions. Effects can be transient. Monitor vital signs and ECG closely.	
Atropine	Hemodynami cally unstable bradycardia (2nd or 3rd degree heart blocks) Acute cholinergic poisoning (organophosphates)	 Heart pumps harder Heart pumps faster – blocks vagal nerve →↑ conduction of SAN, AVN and ventricle →↑ heart rate; Decreases secretions 	IV push neat Maximum 2.4 mg	Bradycardia: 0.6 mg q 3-5 min up to 2.4 mg Cholinergic toxicity: 1.0- 2.0 mg IV. In massive poisoning, IV infusion of atropine may be needed	Tachycardia, Palpitations, Paradoxical bradycardia (if dose is <0.5mg) Seizures, Hypertension, Respiratory failure	Use caution in AMI.	Preload syringes: 0.1 mg/ml, 10 ml and 1 mg/ml, 10 ml. Vials and ampoules: 0.6mg/ml	If dose of < 0.5 mg given or drug not administered as rapid IV push, a paradoxical bradycardia may occur	

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Drug	Main Uses	Therapeutic Effects	Routes of Administration	Usual Dosages	Side Effects	Contraindications	How Supplied	Comments
Lidocaine	Multifocal PVCs VT VF	Raises fibrillatory threshold thus enhances the effect of DC shock Decrease speed of electrical impulse ↓ heart rate	IV or IO push IV maintenance infusion: 1g/500 ml D⁵or NS (2 or 4 mg/ml)	1-1-5mg/kg Repeat at 3-5min interval as needed Max total dose 3mg/kg Maintenance: 1-4 mg/min, titrate to effect For acute CHF or liver failure patients, reduce loading dose by 50% and start maintenance infusion at 1 mg/min	Hypotension Seizures, Respiratory depression or arrest Widening of the QRS complex Bradycardia leading to cardiac arrest.	Wolfe-Parkinson-White syndrome; severe degrees of SAN, AVN or intraventricular heart block in the absence of a cardiac pace maker. Use caution in bradycardia with PVCs	Preload syringes 100mg/10 ml vials and ampoules: 1% 10 mg/ml, 500 mg in 5 mls.	Can give rapid IV push in cardiac arrest. Do not administer faster than 50 mg/min in a conscious patient. If one agent is unsuccessful after completing 2 doses, you may give the other agent.
Amio- darone	Multifocal PVCs VT- Wide complex tachy VF	Slows conduction through myocardial cells, AV node, accessory pathway Raises fibrillatory threshold Vasodilatation Negative inotrope	IV or IO push Infusion	IV 300mg bolus for VF & pulseless VT Repeat 150mg bolus once after 3-5min IV infusion: 1mg/min X6hr, then 0.5mg/min x18 hr max 2g/day	Hypotension, Bradycardia – cardiac arrest Worsening of CCF Hepatic, thyroid dysfunction	Use with caution In: Bradycardia with PVCs Pregnancy Thyroid disease	Ampoules 150 mg	If one agent is unsuccessful after completing 2 doses, you may give the other agent.

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Drug	Main Uses	Therapeutic Effects	Routes of Administration	Usual Dosages	Side Effects	Contraindications	How Supplied	Comments	Formatted Table
Dopamine (Inotrope)	Cardiogenic shock Septicemic shock Anaphylactic shock Neurogenic shock Hypovolemic shock (after fluid resuscitation has failed to raise BP)	Dopamine: dilates renal & mesenteric vessels (1-2 µg/kg/min) enhancing renal blood flow but NO LONGER recommended in acute oliguric renal failure • increases myocardial contractility (2- 10 µg/kg/min) increasing cardiac output, increasing heart rate • causes peripheral vasoconstriction (>10 µg/kg/min) elevating blood pressure	IV infusion via central / large bore vein	• Dopamine IV infusion : 5-20 μg /kg/min	Low-dose: Hypotension, Tachycardia Moderate-dose: Tachycardia Angina Ventricular arrhythmias. High-dose: Same as moderate Decreased kidney function Hypertension	Hypovolemic patients prior to IV fluid resuscitation	Dopamine 200 mg/5 ml vials	Risk of skin necrosis if drug extravasated. Do not mix with NaHCO3. IV infusion should be titrated to desired effect and gradually tapered when stopping. Nursing implications Monitor BP & cardiac rhythm Monitor drip site closely to observe for extravasation	

Drug	Main Uses	Therapeutic Effects	Routes of Administration	Usual Dosages	Side Effects	Contraindications	How Supplied	Comments	Formatted Table
Nitroglycer in (GTN)	Unstable angina Acute myocardial infarction Pulmonary edema with high BP Hypertensive crisis	Venodilator. Dilates coronary arteries, Relieves spasm Opens up collateral vessels — increases blood flow to the myocardium	Sublingual tablet Transdermal patch Spray IV infusion	SL tablet one stat. Repeat if pain not relieved within 3 to 5 minutes Transderma I patch delivers 0.2 or 0.4mg/hr, lasts 24 hrs IV infusion 10-200 µg/min titrated to response	 Headache Flushing, Tachycardia Hypotension 	Use with caution in patients with hypotension and tachycardia	Sublingua I tablet 0.3-0.4 mg Transder mal patch of 5 or 10mg IV infusion vials of 10mg/10 ml or bottles of 50mg/50 ml	Sublingual route has rapid onset of 1 to 2 minutes with duration of 30 minutes. IV infusion must be delivered via infusion pump Nursing Implications: Monitor BP and heart rate	
Verapamil	PSVT Acute AF or atrial flutter with rapid ventricular response (exclude WPW first)	Ca channel blocker negative inotropic effect— decreases force of cardiac contraction Dilates coronary, systemic and peripheral vessels – increases blood flow and lowers blood pressure	IV or slow bolus	5mg diluted in 5ml given at 1mg/min. If necessary, repeat but should not exceed 10 mg	Hypotension Prolongation of the PR interval, bradycardia-asystole Nodal escape rhythms Heart blocks VF in patient with WPW	Sick sinus syndrome with a functioning pacemaker, heart blocks, hypotension or cardiogenic shock; patients on β blockers because of potentiation of effects	5 mg/2ml ampoules	Nursing implications: Monitor cardiac rhythm & BP	

Drug	Main Uses	Therapeutic Effects	Routes of Administration	Usual Dosages	Side Effects	Contraindications	How Supplied	Comments	Formatted Table
Sodium bicarbo- nate	Profound metabolic acidosis Hyperkalemi a Tricyclic antidepress ant poisoning	Correction of acidosis	IV bolus IV infusion	• 1-1.5 mEq/kg	Hyperosmol arity Hypernatrem ia CNS acidosis (paradoxical alkalosis)	Should not be used unless blood gases show severe metabolic acidosis	Bottles 50 mls or 250 mls. Ampoules 10 mls/20 mls.	Only IV route. No mixing with other drugs.	

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Chapter 8. ORGANIZATION OF RESUSCITATION

Introduction

When a cardiac arrest occurs in specialized areas in the hospital e.g. Emergency Department, ICU, the medical response is usually swift because trained personnel and equipment are immediately available. In the general wards however, the first person to discover a cardiac arrest may be a visitor, a patient or a nurse. Preparation and organization is the only way to overcome these difficulties to give the collapsed patient the best chance for recovery.

In order for resuscitation to be efficient, it must be organized and performed by a team of trained personnel comprising doctors, nurses and health care assistants. The goals of resuscitation are:

- 1. Re-establishing spontaneous respiration and circulation.
- 2. Maintaining and preserving function of vital organs.

American Heart Association (AHA) Phased-Response Approach

The AHA Phased-Response Approach is adopted to provide a systematic approach to resuscitation and it is organized into 4 phases:

Phase I

- anticipation (mental / technical preparation for the code).
- resuscitation before the arrival of the code team.

Phase II

- · resuscitation arrival of the code team.
- resuscitation.
- maintenance (stabilization of the situation).

Phase III

- notification of the family.
- transfer of patient to appropriate facility.
- documentation.

Phase IV

review of the process.

Phase I - Preparation

In the general ward, the ability to respond to a cardiac arrest requires:

- 1. adequate preparation of staff including
 - how to get help when faced with a cardiac arrest.
 - where to locate equipment.
 - how to start the resuscitation.
 - how to use the resuscitation equipment e.g. bag-valve-mask, turn on defibrillator etc.
- 2. adequate equipment preparation including
 - user friendly assembly of equipment e.g. all instruments for intubation in a transparent box.
 - prominent labeling of important drugs.

- prominent display and easy accessibility of resuscitation trolley / cart.
- checking the resuscitation equipment at least daily, if not every shift.

When a new nursing staff joins the ward or department, preparation for and responding to a cardiac arrest must be an important part of the orientation program. Periodic checks should be performed on all staff and equipment to assess their operation readiness.

Phase I – Resuscitation before Arrival of Code Team Action Plan for General Ward (Fig. 8.1)

The following action plan is suggested for any general ward nurse who discovers a collapsed patient:

- assess responsiveness.
- if patient is unresponsive, **shout for help, activate the code button and get the resuscitation equipment.** Foreign body search and Giving 2 breaths is now omitted to minimize delay in starting chest compression.
- place the patient in a supine position and begin the ABC resuscitation.
- if ECG monitor is available, attach the monitor leads and check ECG rhythm. If the monitor shows VF or pulseless VT, then depending on local practice, a state registered nurse may defibrillate and treat according to the VF/pulseless VT protocol.
- if the monitor is not yet available, then Nurse 1 starts bag-valve-mask ventilation and Nurse 2 performs chest compression until the monitor is available to check the rhythm.
- the first state registered nurse at the scene of the cardiac arrest, designated Nurse 1 should assume leadership for resuscitation until a suitably trained doctor arrives to take over.

Phase II - Code Team Arrival, Resuscitation and Stabilization

- 1. When a doctor arrives, it is suggested that he stations himself at the patient's head to take care of the patient's airway and breathing. He is designated as Doctor 1 and leader of the resuscitation until a more senior doctor comes to take over.
- 2. Nurse 1 gives a brief verbal report to Doctor 1 in the suggested format (e.g., SBAR) -
 - Who was the patient i.e. age, diagnosis and relevant background history.
 - Where was the patient.
 - When was the patient found.
 - What was patient's condition e.g. unresponsive, gasping, pulseless etc.
 - What was the initial ECG rhythm.
 - What had been done so far.
 - What response if any, so far.
- 3. With the launch of this Life Support Course for Nurses, all state registered nurses in Singapore will be able to assist in the management of patient's airway and breathing, including rendering the necessary assistance for intubation. This nurse is designated as Nurse 1 during the resuscitation.

- 4. The second doctor (designated as Doctor 2) to arrive should establish intravenous access, defibrillate if indicated and administer intravenous medications.
- 5. The second nurse (designated as Nurse 2) will continue with chest compression until told by Doctor 1 to stop.
- 6. If a third nurse is available, she can help with preparation of intravenous medications and be a "runner" for the team.
- 7. As the leader, Doctor 1 must make all the critical management decisions. Team members may make suggestions but the ultimate responsibility lies with the leader. This is important because performing resuscitation for an unexpected collapse in the general ward is very chaotic. Lack of decisive leadership or lack of respect for leadership will add more disorder to the chaos and this may be detrimental to patient care.

Phase III - Notification of Family, Transfer of Patient and Documentation

- 1. When the resuscitation is proceeding smoothly, Doctor 2 should speak to witnesses and get more details if available. All relevant information, findings and resuscitation progress must be documented. As stated in the chapter on Post Resuscitation Care, proper documentation is very important.
- 2. When spontaneous circulation returns and is maintained, Doctor 2 liaises with the ICU/CCU team to accept the patient. The patient can then be transferred to the ICU/CCU accompanied by a doctor, a nurse and an assistant.
- 3. Generally speaking, the patient is not transferred unless the SBP is maintained at or above 90 mmHg for a continuous 15 minutes.
- 4. At the end of the resuscitation, whether it is successful or not, Doctor 1 and Nurse 1 should speak to the relatives. If resuscitation is prolonged, then either Doctor 1 himself or Doctor 2 should update the relatives periodically, if appropriate.

Phase IV - Review

- 1. After the patient has left the ward, it is advisable for all staff involved in the resuscitation to gather for a debrief led by the most senior doctor. In the general ward, this may not always be possible, in which case, the nurses are encouraged to hold their own debrief. Debrief serves the following purposes:
 - review of the events leading to patient's collapse.
 - audit of resuscitation.
 - ventilation and de-stressing opportunity.
- 2. A successful debrief is one that is concise but done with skill and sensitivity. Learning from the clinical experience is an invaluable way to reinforce those actions that were effective and to improve on performance of those that were not.

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• all equipment and consumables used in the resuscitation must be cleaned and replaced or replenished as soon as possible, and certainly before the end of the shift.

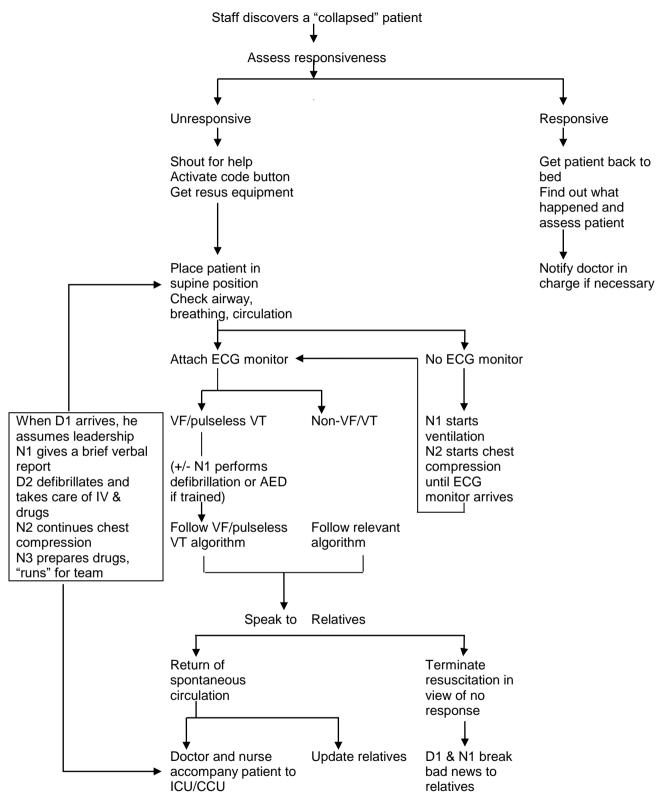
Action plan for specialized area (e.g. Emergency Department, ICU)

- 1. assess responsiveness.
- 2. if patient is unresponsive, shout for help and push the patient into the resuscitation room / area, if necessary.
- 3. ensure that the patient is in a supine position and begin the ABC of resuscitation.
- 4. attach the monitor leads and check ECG rhythm.
- 5. Most specialized areas have their own **resuscitation protocols**, which all staff should be thoroughly familiar and follow accordingly.

Summary

Performing resuscitation in the general ward can be a harrowing and chaotic experience. Prior preparation of equipment and training of all staff can bring about order in the madness, thus maximizing patient's chance of eventual recovery.

Fig. 8.1 Action Plan for Collapsed Patient in General Ward



Key:

N1 = Nurse 1, N2 = Nurse 2, N3 = Nurse 3

D1 = Doctor 1, D2 = Doctor2

Chapter 9. POST RESUSCITATION CARE

Introduction

Full recovery from cardiac arrest is rarely immediate. The restoration of pulse and rhythm marks the start and not the end of a successful resuscitation. The true end point is a fully conscious, neurologically intact patient with a spontaneous cardiac rhythm and an adequate output. The chances of achieving a successful resuscitation are greatly enhanced when:

- The arrest is witnessed.
- The underlying arrhythmia is VF or pulseless VT.
- Successful defibrillation is achieved in 2-3 minutes and not longer than 8 minutes.
- CPR is started and continued until the defibrillator arrives.

A comprehensive, structured, integrated, multidisciplinary system of post-resuscitation care after ROSC should be put in place.

Post Resuscitation Care

Clinical decisions need to be made during the early post resuscitation period include:

1. Airway and ventilator management

- Ensure that the airway is open, oxygenation and perfusion are adequate. The patient may require advanced airway support. Positioning the unintubated patient to prevent aspiration is essential.
- Maintain continuous quantitative waveform capnography to monitor ETT placement. Avoid hyperoxia as it may cause re-perfusion injury. Whether the patient is intubated or not, aim for SaO2 between 94-99%.

2. Haemodynamic management

- Assess the hemodynamic status of the patient. Vital signs should be monitored every 5 to 15 minutes and patient's progress reported at intermittent intervals. Report any deterioration or drastic changes immediately.
- 12 –lead ECG soon after ROSC and must be repeated to rule out acute coronary syndrome.
- IV fluid to optimize blood pressure, cardiac output and urine output. Maintain an accurate record of all fluids given.
- Drug infusions Anti-arrhythmics that has been effective during the resuscitation may need to be continued as infusions. Infusion pumps should be used to ensure accurate delivery.
- When resuscitation is prolonged, hypotension is common following return of spontaneous circulation. Maintain a mean arterial pressure of > 65 mm Hg or systolic BP > 90 mm Hg. Should hypotension persist despite fluid resuscitation, dopamine titrated to maintain a systolic blood pressure of 90 mmHg is the agent of choice.

3. Targeted temperature management

• Targeted temperature management improves outcomes of comatose patient after in-hospital or out-of-hospital cardiac arrest by reducing metabolism while vital

functions recover. Mild hypothermia is induced by cooling the patient to a temperature range of 33-36°C. This is maintained for 12-24 hours, followed by gradual re-warming (0.25-0.33°C/hours) to reach normal body temperature. There are various cooling methods that are currently used, such as cooling blankets.

4. Assess the neurological status of the patient

 The mental state of arousing from unconsciousness to a more alert state, ability to respond to verbal or painful stimuli. Check the pupils. Check the motor response to detect any motor deficit.

5. Blood glucose control

- Blood glucose of >10 mmol should be treated but hypoglycaemia must be avoided
- 6. Maintenance doses of anti-arrhythmics should be started when indicated.
- 7. Early assessment by cardiologist for percutaneous coronary intervention when acute coronary event is the underlying cause of the cardiac arrest.
- 8. Extra-corporeal CPR (ECPR) is indicated for selected patients with reversible etiology, for example drug overdose. This involves inserting catheters in the femoral vessels and using a bypass machine to take over the cardiopulmonary function.
- 9. Underlying abnormalities that may have led to the cardiac arrest should be actively sought and corrected. Common abnormalities that may require correction after the arrest include electrolyte imbalances, hypoxemia and acidosis.
- 10. Make immediate arrangement to **transfer the patient to ICU** if the patient's condition remains critical. Prior to transfer, ensure that the patient's condition is stabilized.

The NRC recognizes that not all hospitals are able to perform continuous quantitative waveform capnography and therapeutic hypothermia hence for now, they are highly recommended but not mandated.

Documentation of Resuscitation

The resuscitation record is an essential component of any resuscitation effort. The record allows us to reconstruct the sequence of events with correlation of interventions and responses during the resuscitation. It provides documentation of the life support procedures that were performed. Such documentation allows the evaluation of appropriateness of care and facilitates the prospective collection of data for measuring the outcome and effects of training. The record should include the following:

- 1. Patient demographics.
- 2. Times of arrest, initiation of CPR, defibrillation and initiation of ALS.
- 3. Doses and routes of administration of drugs.
- 4. Sequential cardiac monitoring with correlation with patient's status, therapy and responses.
- 5. Counter shocks delivered.
- 6. Special procedures.
- 7. Vital signs and patient's response.
- 8. Patient's status and disposition at the end of resuscitation.

Transfer to ICU

Once the airway, breathing and circulation are secured, safe transportation to the ICU/CCU should be arranged. Prior to transportation

- All IV lines should be secured.
- Patient should be connected to a portable monitor-defibrillator.
- A full oxygen cylinder should be connected to the breathing apparatus or ventilator.
- Atropine, epinephrine and lidocaine to be brought along.
- One doctor and one staff nurse to accompany the patient. Their responsibilities include
 - continuous assessment
 - maintenance of airway, breathing and circulation therapy
 - provision of necessary information to the ICU staff

Handling Distressed Relatives and Breaking Bad News

Handling distressed relatives is an under-emphasized part of the work. It is a time that the relatives will always remember and, if handled badly, will leave lasting scars. Providing genuine understanding and support is the key to their management. The following is suggested when speaking to relatives:

- **Empathize** sit, listen patiently and reflect the relatives' reactions rather than make assumptions and categorize them.
- **Enable** relatives to come to terms with the reality of the situation and to experience the pain.
- Encourage them to express their feelings and give them the emotional support.

Summary

After a successful resuscitation, it is crucial to

- 1. secure and stabilize airway, breathing and circulation.
- 2. monitor hemodynamic and neurological states are closely before, during and after transfer to ICU/CCU.
- 3. identify and treat acute coronary syndromes and other reversible causes
- 4. control temperature to optimize neurologic recovery
- 5. keep relatives informed of patient's condition.

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Chapter 10. CARDIAC ARREST IN SPECIAL CIRCUMSTANCES

1. Near drowning

This is an episode of asphyxiation in fluid, usually water. The main effect of submersion is respiratory arrest with cardiac arrest being secondary. Death must not be declared until basic and advanced life support has been continued for 45 minutes without success. This is particularly important in children who have a higher chance of survival. Rescuers must take no risk in attempting to reach or recover the patient. The patient should be removed from the water in a horizontal or head-down posture to reduce the chance of aspiration. In a diving accident, the cervical spine must be protected when moving the patient.

Airway and Breathing

Basic life support should not be performed in the water. Expired air ventilation may be attempted once the rescuer can stand or has reached a point of support. If a head or neck injury is suspected, the airway should be opened using the modified jaw thrust.

Circulation

Chest compression follows the standard algorithm; perform 5 cycles of CPR before activating the EMS for a lone rescuer. Once resuscitation is underway, a nasogastric tube should be inserted to decompress the stomach. When the patient has been stabilized, a chest X-ray, an arterial blood gas and a 12 lead ECG should be obtained. The patient should be hospitalized as secondary pulmonary edema may develop.

2. Pregnancy

The causes of cardiac arrest in pregnancy include hemorrhage, pulmonary embolism, amniotic fluid embolism, placenta abruption and eclampsia. It is important to remember that two lives are involved in the resuscitation of a pregnant patient.

Airway and Breathing

There is an increased risk of regurgitation and pulmonary aspiration in a pregnant patient. In the third trimester, the large uterus splints the diaphragm and high inflation pressures may be required. The landmark for a Heimlich maneuver in a choking pregnant patient is the mid-sternum.

Circulation

The pressure on the inferior vena cava from the gravid uterus needs to be relieved if venous return is to improve. Raising the mother's right hip or manually displacing the uterus to the left can do this. Chest compression is undertaken in the standard manner.

Maternal blood volume is large which can lead to fetal shock without the mother manifesting the signs of shock. An obstetrician and a pediatrician should be involved at an early stage.

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3. Poisoning

Rescuers must avoid being exposed to or coming into contact with any known toxic or hazardous material. Avoid mouth-to-mouth ventilation if the presence of toxic substances like cyanide, hydrogen sulphide, corrosives and organophosphates are suspected. It is also important to remember that many substances release vapors that are absorbed by the respiratory tract and skin; hence masks, eye shields, gloves and gowns are necessary protective gear.

Except in paraquat ingestion, high concentration oxygen should be given to counteract tissue hypoxia. The risk of pulmonary aspiration is increased after poisoning. Endotracheal intubation before gastric lavage is essential in the unconscious patient.

After resuscitation, the management of the poisoned patient includes removal of poison by gastric lavage, adsorption, catharsis or specific therapy depending on the poison.

4. Hypothermia

Hypothermic patients with a perfusing rhythm and without a preceding cardiac arrest, perform non-invasive re-warming e.g. external warming with heating blankets, forced air, and warmed infusion. For severely hypothermic patients in cardiac arrest they may benefit from invasive re-warming e.g. cardiopulmonary bypass or extracorporeal circulation.

Summary

The basic principles of resuscitation remain the same no matter under what circumstances cardiac arrest occurs. The main differences lie in the safety implications for patients and health care workers.

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ANNEX I. BRADYARRHYTHMIAS AND TACHYARRHYTHMIAS

Introduction

This chapter focuses on management of bradyarrhythmias and tachyarrhythmias. A review of basic cardiac electrophysiology and characteristics of normal sinus rhythm will enhance understanding of brady and tachyarrhythmias. It is important to remember that the ECG rhythm strip must be interpreted in the light of the patient's symptoms when making treatment decisions.

Electrical Activity of the Heart Heart Muscle Cells

The heart has 2 types of cells:

- 1. Working muscle cells that contract and relaxes resulting in the pumping action of the heart.
- 2. Electrical cells that form the conduction pathway. Pacemaker cells are electrical cells that can generate an electrical impulse, a property known as automaticity. Pacemaker cells are found in the: -
- sinoatrial node (SAN)
- atrial conduction pathways
- atrioventricular node (AVN)
 known as AV junction together
- bundle of His
- bundle branches
- ventricular Purkinje system

In the normal heart, the SAN is where electrical impulses originate. However, any of the above pacemaker cell is capable of generating electrical impulses giving rise to premature atrial or junctional or ventricular complexes. One important principle to remember is that each impulse originating from the same site will have the same morphology e.g. all P waves from the SAN will look the same but a P wave from the atrial conduction pathway will look different from a SAN-P wave.

Basic Electrophysiology

There are different concentrations of potassium, sodium and calcium ions inside and outside the cells of the heart. Movement of these ions in and out of the heart cells result in depolarization and repolarization of the cells, which in turn lead to the different wave forms (P, QRS, T waves) on the ECG.

At the start of depolarization, sodium ions enter the cells. When depolarization occurs in the atrial muscle mass, P wave is seen on the ECG. When depolarization occurs in the ventricular mass, QRS complex is seen on the ECG.

When calcium ions start entering the cells after sodium ions had stopped, this is represented by the ST segment of the ECG. Repolarization then follows in which potassium ions leave the cells. Repolarization is represented by T wave on the ECG. Soon after, depolarization will start again and this cycle of movement of ions results in the pumping and the electrical conduction of the heart.

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One of the important rules to remember about ECG interpretation is that **every QRS complex must be followed by a T wave** because together they represent the complete cycle of depolarization and repolarization of the ventricles of a normal beating heart.

However, it is **not necessary for every QRS to be preceded by a P wave** because atrial activity (represented by P wave) may not be transmitted to the ventricles (represented by QRS) e.g. in heart blocks, or ventricular activity is not initiated by atrial activity e.g. premature ventricular complex (previously known as ventricular ectopics).

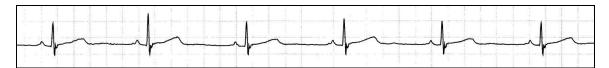
Rhythm Strip versus 12 Lead ECG

When using a cardiac monitor, electrodes placed on the chest or the limbs should leave the precordium unobstructed for cardiac compression and defibrillation. The lead that displays the most prominent P wave with sufficient QRS to trigger the rate meter should be selected as the monitoring lead. This is usually lead II because it follows the direction of the normal electrical impulse.

It is important to remember that display and print out from a cardiac monitor is suitable for rhythm recognition only and not for other analysis e.g. ST segment abnormalities. When indicated, a 12 lead ECG should be performed.

Normal Sinus Rhythm

An electrical impulse that originates from the SAN and is conducted normally through the rest of the heart is a normal sinus beat. Normal sinus rhythm has the following characteristics (Fig. I.1):



- 1. rate between 60 to 100 beats/minute
- 2. regular rhythm
- 3. the P wave is positive (i.e. upright) in lead II
- 4. the PR interval is constant and less than 0.2 second (5 small squares), whereby the PR interval is measured from the beginning of the P wave to the beginning of the QRS complex.
- 5. the QRS complex is narrow and less than 0.12 second (3 small squares)
- 6. each P wave is followed by a QRS complex

Sinus Bradycardia, Tachycardia, Arrhythmia

Sinus bradycardia has all of the above characteristics except that the rate is less than 60 beats / minute. Sinus tachycardia has a rate above 100 beats / minute. Sinus arrhythmia refers to the physiological slowing down and speeding up of the heart in response to respiration. It has all the characteristics of normal sinus rhythm except that it is slightly irregular.

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Bradyarrhythmia Fig. I.1

The various types of heart blocks contribute to some of the important bradyarrhythmias. Unlike tachyarrhythmias whereby an accurate diagnosis of the arrhythmia may be needed for treatment decisions, the management of patients with bradyarrhythmias is based less on an accurate ECG diagnosis but more on patients' symptoms. Algorithm I.1 summarizes the management of bradyarrhythmias.

Tachyarrhythmias

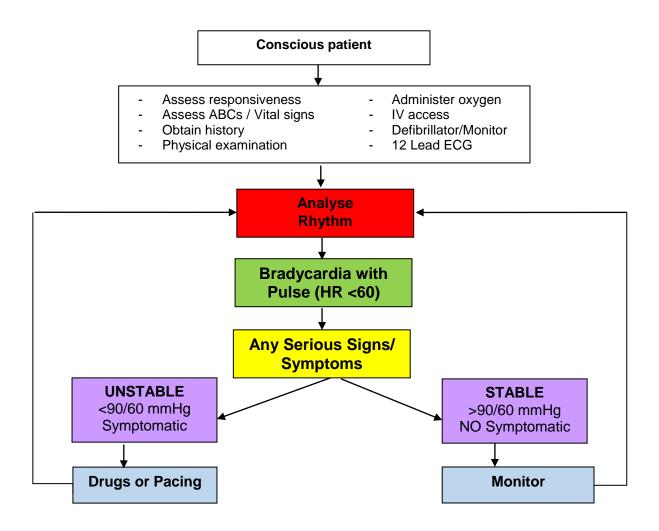
In managing tachyarrhythmias, one of the key differentiation is whether the QRS complex is wide or narrow. Equally important to remember is that the ECG rhythm must be interpreted in the light of patient's signs and symptoms. Algorithm I.2 summarizes the management of tachyarrhythmias.

Summary

When faced with an abnormal ECG, it is important to correlate it with the patient's signs and symptoms. Management decisions must be based on the patient's condition rather than on the ECG.

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Algorithm I-1 Management of Bradyarrhythmias



First line

- IV Atropine 0.6mg every 3-5mins (max 4 doses)
 May cause paradoxical bradycardia post heart transplant
- IV Dopamine infusion 5-20 mcg/kg/min
- IV Adrenalne infusion 2-10 mcg/min

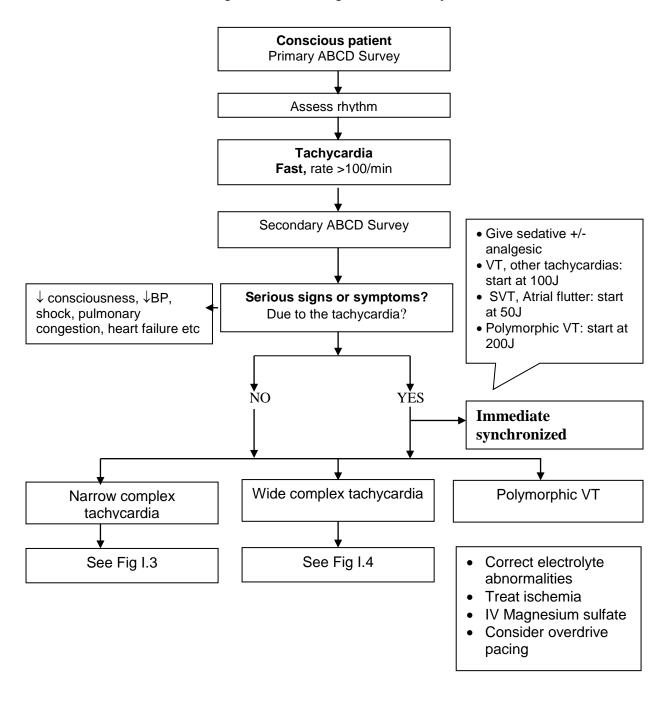
If drug therapy fails

- Begin transcutaneous pacing with analgesia and sedation
- · Transvenous pacing, if available

- No further treatment needed for the bradycardia
- If Type II 2nd degree heart block or 3rd degree heart block:
 - Rhythm + haemodynamic monitoring
 - Standby for transcutaneous pacing in event of deterioration

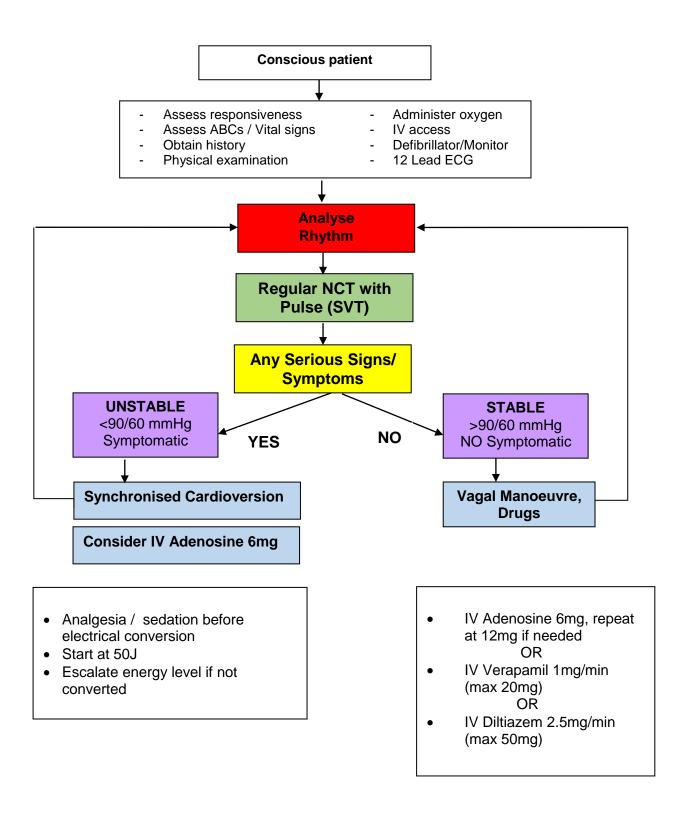
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Algorithm I-2 Management of Tachycardia

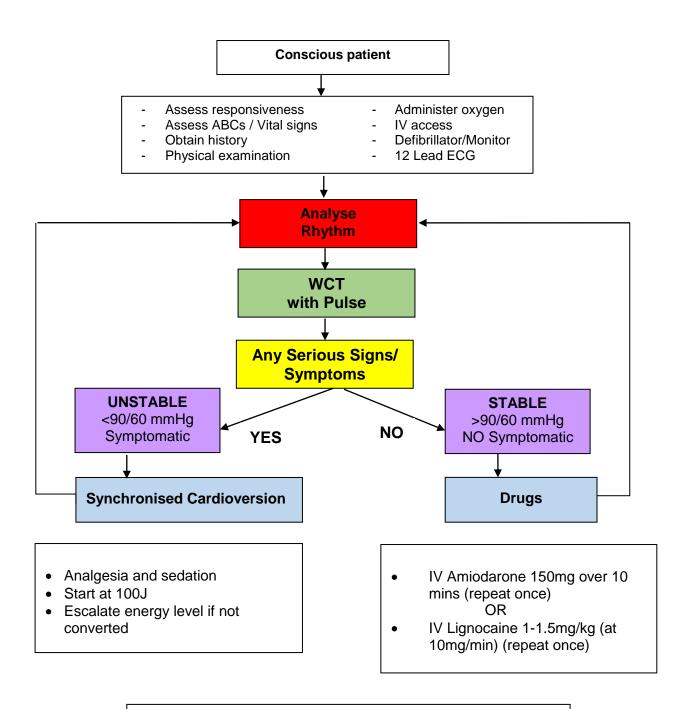


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Stable Narrow Complex Tachycardia Algorithm Fig. 1.3



Stable Wide Complex Tachycardia Algorithm Fig. 1.4



When converted

- IV Amiodarone 1mg/min x 6 hours, then 0.5mg/min x 18 hours OR
- IV Lignocaine 1-2mg/min x 24 hours
- Monitor ABCs, vital sings, 12 lead ECG
- Transfer as appropriate

Annex II. PEDIATRIC AND INFANT RESUSCITATION

Cardiac Arrest in Infants & Children

Introduction

Most arrests in infants and children are preventable; hence it is vital for healthcare providers to:

- Be aware of the precipitating factors
- Recognise infants and children in impending collapse
- Intervene before the patient collapse

Mechanism

The most common cause of arrest in infants and children is hypoxia or shock. The following are precipitating factors:

- Infection
- Hypovolemia, dehydration
- · Respiratory diseases e.g. pneumonia
- Trauma
- Cardiac disease
- Foreign body aspiration

Most infants and children response well to respiratory arrest when resuscitative measures are initiated early however they tend to perform badly in arrest of cardiac origin.

Signs of Impending Arrest				
Respiratory	Cardiac			
 Tachypnea or shallow breathing, apnea Accessory muscle use Chest retractions, severe – intercostal and subcostal Breath sounds decreased Restless or drowsy Cyanosis 	 Rapid and thready pulse, or bradycardia Rapid and deep breathing, or shallow breathing Cold and clammy Restless or drowsy Hypotensive 			

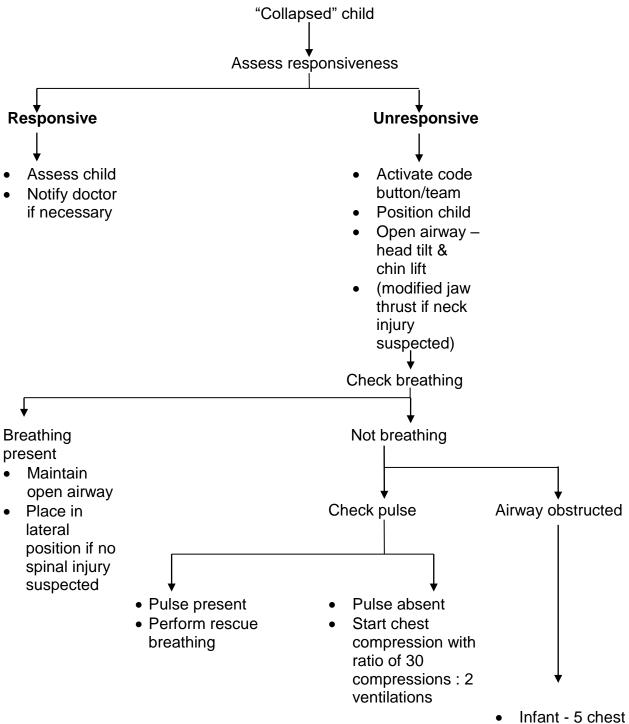
Vital Signs in Children

Age Group	Upper Limit of Respiratory Rate	Upper Limit of Pulse Rate
Infant	50	160
Toddler	35	140
School Age	25	120
Adolescent	20	110

Lower limit of systolic blood pressure = 70 + (Age x 2) mmHg Lower limit of systolic blood pressure in infants = 60 mmHg

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Initial Resuscitation of Pediatric Patient



- Infant 5 chest thrusts and 5 back blows
- Child 30 chest compressions
- Perform finger sweep if foreign body visible in oral cavity

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Advance Life Support in Children

1 Immobilise Cervical Spine

- Manual
- Stiff collar
- Sand bag and tape
- Padding of approximately 2.5 cm beneath shoulders

2 Maintain Patent Airway

- Principles of airway control
 - Unconscious child is turned to the side after immobilising neck
 - Conscious child is allowed to assume optimal position
 - One parent is allowed to remain with the child
 - Equipment to open the airway is introduced gradually

Nasopharyngeal airway

Indication - semi-conscious patient

nasopharyngeal obstruction

Size -12 F for full term to 36F

- measure from tip to nose to angle of jaw

Oropharyngeal airway

Indication - unconscious patient

bite block after intubation

Size 000, 00, 0, 1, 2, 3, 4

measure from middle of incisors to angle of jaw

□ Suctioning apparatus & suctioning

Indication - secretion, blood, vomitus in airway

Catheter size 5F to 12F suction catheters

Technique observe sterile technique if suctioning ETT

insert catheter into ETT then apply suction pressure apply suction pressure only on withdrawing the catheter

suctioning should not exceed 5 seconds withdraw catheter in a rotating movement

hyperventilate before and after suctioning with 100% oxygen

3 Breathing

Principles use humidified oxygen

use the highest concentration of oxygen available

□ Oxygen Supplement

Nasal Cannula

Indication - modest oxygen supplementation, adequate ventilation O₂ flow rate- 0.5 to 6 l/min Maximum FiO₂- variable

Face Mask

Indication - moderate oxygen supplementation, adequate ventilation

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O₂ flow rate- 6 to 10 l/min Maximum FiO₂- 50%

Non re-breathing face mask with oxygen reservoir bag

Indication- moderate oxygen supplementation, adequate ventilation

O₂ flow rate- 10 to 15 l/min Maximum FiO₂- up to 85%

Airway Management for Inadequate Ventilation

Bag and Mask (self inflating)

Indication- High oxygen supplementation, inadequate ventilation

Size of bag- 240 ml for infant

500 ml for child 1600 ml for adult

Size of mask- should cover the bridge of nose to area just under the lips

circular 01, 1, 2 for infant

shaped to nose 2, 3 for child (shaped to nose 4, 5 for adult)

O2 flow rate - 5 to 10 l/min for infant

- 10 to 15 l/min for child

Maximum FiO2- up to 85%

Caution: routine use of cricoid pressure during intubation attempt is not recommended as it may occlude the trachea.

Caution: Hyperventilation is harmful during cardiac arrest. The ideal tidal volume should achieve modest chest wall rise without causing gastric distension or pneumothorax.

Endotracheal Intubation

Indications - apnea, moderate to severe respiratory distress, absent gag

reflex, severe head injury, severe facial injury or burns and oral

burns

Size of ETT - age + 16

4

- use 3 mm (internal diameter) for full term infant

- tubes are uncuffed from size 2.5 mm to 5.5 mm

- size 5 mm tube may be cuffed or uncuffed

A cuffed tracheal tube may be useful in cases of poor lung compliance, high airway resistance or large glottic leak. The cuff inflation pressure should be monitored regularly and must remain below 20cm H2O.

Size of adapter should be appropriate

Size of Laryngoscope

- straight blade size 0 for full term infant

- straight blade size 1 for 1 to 2 years old

- straight and curved blades size 2 for 2 to 8 years old

- curved blade size 3 for more than 8 years old

Technique - lubricate ETT

- check patency of tube

- check cuff

- use with or without stylet

- insert ETT to a depth of about :-

10 cm for full term infant

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12 cm for 6 months old infant (12+ age/2) cm for children more than 1 year old

- secure with cloth or adhesive tape

Larvngeal Mask Airway

The laryngeal mask airway is an acceptable initial airway device for providers experienced in its use.

4 Support Circulation

For delivery of drug and fluids

Endotracheal route

- Only for delivery of drugs, not for large volume of fluids
- Delivery via suction catheter passed deep into ETT
- Hyperventilate with 100% oxygen
- Delivery of epinephrine, atropine, lidocaine, nalaxone
- Dosage is higher than the intravenous route

□ Intravenous route

- Preferred route
- Difficult to establish sometimes
- Blood should be taken for GXM, FBC, U/E, Hypocount

□ Intraosseus route

Indication failed intravenous access Equipment intraosseous needle

> 3-way adaptor extension tubing

10 ml syringe for flushing

Monitor for extravasation of fluid

Defibrillation

Ventricular tachycardia or fibrillation is in infants and children, it is more common to see hypoxia induced sinus bradycardia degenerating into asystole.

Size of paddles 4.5 cm diameter for infants

8 cm diameter for child

Paddle position as for adults

alternatively, anterior paddle over the chest and

posterior paddle over the back

Energy level 4 J/kg (synchronised or unsynchronised)/

> (biphasic or monophasic waveform) should be used for the first and

subsequent shocks.

Pulseless ventricular tachycardia or fibrillation should be treated with a single shock followed by immediate resumption of CPR (30 compressions to 2 ventilations). Do not reassess the rhythm or feel for pulse. After 2mins of CPR, check the rhythm and give another shock if indicated.

AED may be used in children above one year of age. Attenuators of the electrical output are recommended between 1-8 years of age.

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Intravenous Fluids

Crystalloid (e.g. normal saline) and colloids (e.g. 5% albumin) are used as 10 – 20 ml/kg boluses in hypovolemia

□ Intravenous Drugs

Please refer to chapter 7 on Drugs

□ Temperature Control

Treat fever aggressively after a cardiac arrest.

In some studies, a child who regains spontaneous circulation but remains comatose after a cardiac arrest may benefit from being cooled to a core temperature of 32-34°C

For 12-24hrs. After a period of hypothermia, the child should be rewarmed slowly to 0.25-0.5°C

5 Neonatal Resuscitation

Advanced preparation

Find out if there is

- Meconium stain
- Premature delivery
- Twins

Prepare

- Radiant warmer
- Clean, dry towels

On delivery

- Clean and dry newborn baby
- For premature baby, cover head and body with face expose with plastic wrapping. No need to dry the baby beforehand.
- Put in Trendelenberg position
- Tactile stimulation and suctioning if required
- Apgar score the baby at 1 and 5 minutes

Apgar Score

Sign	0	1	2
Heart rate	Absent	Slow (< 100/min)	> 100/min
Respiration	Absent	Slow, irregular	Good, crying
Muscle Tone	Limp	Some flexion	Active motion
Reflex irritability (catheter in nares)	No response	Grimace	Cough or sneeze
Colour	Blue or pale	Pink body with blue extremities	Completely pink

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- Ventilation: an initial inflation for 2-3 seconds must be given for the first few breaths to help lung expansion.
- Do not bag and mask if there is still meconium in the mouth. Meconium should be suctioned away before bag and mask.
- Suctioning meconium from the baby's nose and mouth before delivery of the baby's chest (intrapartum suctioning) is not useful and no longer recommended.
- Chest compression if heart rate persistently less than 60/min
- Intubation and resuscitative drug via 5 F umbilical vein catheter may be needed.
- Administering of adrenaline via tracheal route is not recommended. If the tracheal route is the only option available, then a dose of 100mcg kg must be given.
- Standard resuscitation in delivery room should be 100% oxygen however, lower concentrations are acceptable.

Summary

Cardiorespiratory arrest in infants and children may be prevented by

- recognising the signs of impending arrest and
- intervening with appropriate measures.

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References

- Basic Cardiac Life Support Course Provider Manual 2017 Edition, National Resuscitation Council Singapore.
- 2. Advanced Cardiac Life Support Provider Manual Updated 2016, National Resuscitation Council Singapore.
- 2015 American Heart Association Guidelines Update for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care.