Advanced Life Support Learning Package 2016
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ADVANCED LIFE SUPPORT

Introduction

Advanced Life Support (ALS) is Basic Life Support (BLS) with the addition of non-invasive or invasive interventions and pharmacological agents. Completion of Basic Life Support competency is mandatory to facilitate the progression to ALS competency.

This learning package provides ALS providers with information to assist in their preparation for ALS practice; to demonstrate competent knowledge and skills in the care and management of critically ill patients during life threatening emergencies. The Registered Nurse may be required to initiate and maintain ALS measures in the absence of a Medical Officer.

Objectives

On completion of the session, based on Epworth HealthCare Protocol, the ALS provider will be able to:

- Demonstrate competency in the management of life threatening situations, specifically:
  - Situational assessment
  - Patient assessment
  - Airway management
  - Ventilation techniques
  - Restoration and support of circulation
  - Post-arrest care
- Describe the occupational health and safety issues associated with ALS interventions
- Demonstrate the roles of ALS providers in relation to:
  - Advanced airway and ventilation maintenance
  - ECG monitoring
  - Arrhythmia analysis
  - Arrhythmia management
  - Management of resuscitation equipment
  - Occupational health and safety issues
  - Team leader decision making
  - Documentation

(Heinrich S. & Barry, M. 2014)
Roles and Responsibilities

The aim of ALS is to provide the best possible outcome for the patient. A coordinated approach to an emergency situation is the best way to achieve this outcome.

When faced with an emergency, remember some basic rules:

- Assess the situation
- Commence BLS
- Call for help
- TAKE CHARGE and delegate roles
- REMAIN IN CONTROL

Roles

- Team Leader - 1 person
- Airway – 1 person
- CPR – 1 person
- Scribe – 1 person
  - Drugs (dosage and time)
  - Procedures (intubation, cannulation)
  - Defibrillation (Joules and response (including BP))
  - Who was present at the emergency
  - Timekeeping
- Drugs - 1 person
- Intravenous (IV) cannulation /IO – 1 person
- Runner – 1 person

Remember

- Ascertain admission diagnosis / history / previous bloods
- Inform consultant
- Ascertain resuscitation status
- Inform relatives
- Organise social work/pastoral care (if required)
- A debrief is of vital importance – if for no other reason than to discuss what could be improved for next time
Post Resuscitation Care

One way or another, post resuscitation care will be required. It is important not to leave the area as the primary nurse may need help.

Issues to be considered

- Oxygen therapy/ventilation
- Ongoing monitoring and observations
- Drug infusions
- Preparation for transfer to ICU/Cardiac Unit/Theatre/Cath Lab
- Debrief with relatives and staff
GUIDELINES FOR MANAGEMENT

ACTIVATING CODE BLUE / MEDICAL EMERGENCY RESPONSE

A Code Blue should be called for any medical emergency where urgent medical assistance is required as per Epworth HealthCare Adult MET and Code Blue Criteria

Adult MET Criteria:

<table>
<thead>
<tr>
<th>Criteria</th>
<th>MET</th>
<th>Code Blue</th>
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<tbody>
<tr>
<td>Cardiovascular</td>
<td>SBP &lt; 90 or &gt; 200</td>
<td>Cardiac arrest: SBP &lt; 70</td>
</tr>
<tr>
<td></td>
<td>SBP fall &gt; 20 mmHg not due to medications</td>
<td></td>
</tr>
<tr>
<td></td>
<td>HR &lt; 40 or &gt; 130</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Uncontrolled bleeding</td>
<td>Respiratory arrest</td>
</tr>
<tr>
<td></td>
<td>Uncontrolled chest pain</td>
<td>Severe difficulty breathing</td>
</tr>
<tr>
<td>Respiratory</td>
<td>RR &lt; 8 or &gt; 28</td>
<td></td>
</tr>
<tr>
<td></td>
<td>SaO₂ &lt; 90% on 6L O₂ via Hudson mask</td>
<td></td>
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<tr>
<td></td>
<td>Stridor</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Severe difficulty breathing</td>
<td></td>
</tr>
<tr>
<td>Neurological</td>
<td>Decreased conscious state</td>
<td>Unconscious</td>
</tr>
<tr>
<td></td>
<td>Uncontrolled pain</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Seizures</td>
<td></td>
</tr>
<tr>
<td>Renal</td>
<td>Urine output &lt; 150mls in 6hrs</td>
<td></td>
</tr>
<tr>
<td></td>
<td>&gt; 500ml in 1hr</td>
<td></td>
</tr>
<tr>
<td>Endocrine</td>
<td>Glucose &lt; 3 or &gt; 20</td>
<td></td>
</tr>
<tr>
<td>Laboratory</td>
<td>Na &lt; 125 or &gt; 155</td>
<td></td>
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<tr>
<td></td>
<td>K+ &lt; 3 or &gt; 6</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Hb &lt; 70 or drop &gt; 20g/L</td>
<td></td>
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<tr>
<td></td>
<td>HCO₃⁻ &lt; 15mmol</td>
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This involves both of the following:

- Activating the “Code Blue” staff alert (call button)
- Dial local hospital site emergency number to confirm the medical emergency, stating “Code Blue”, and the location (ward/area and bed number)

The Security Department will electronically page the Medical Emergency Response Team, indicating “Code Blue” and the location.

The response team is site specific but may include:

- Intensive Care Fellow
- Intensive Care Liaison Nurse
- Intensive Care Nurse (ALS accredited)
- Cardiac Unit Nurse (ALS accredited)
• Emergency Controller
• Hospital Medical Fellow

Basic Life Support measures are to be commenced by staff on location and should continue upon arrival of the emergency response team and continue to assist as directed by the team.

Responsibilities

ICU Medical Fellow responsibilities:
• Coordinates the medical emergency response team
• The preferred person for intubation and airway management
• Manages the medical emergency until the admitting consultant arrives or instructs otherwise
• Liaise with relevant consultant and clinical staff

In the absence of Medical Officer, an ALS accredited Registered Nurse may, in accordance with protocols:
• Initiate defibrillation and pacing
• Administer Adrenaline and Amiodarone according to the ARC guidelines

Other roles of the ALS accredited nurse include:
• Facilitating ward staff participation – BLS, clinical assistance, information sharing and notification of appropriate persons
• Coordinating ongoing management strategies for patient management i.e. transferring the patient

IV access

• Preferred way of drug administration
• Peripheral IV cannula in a large peripheral vein
• External Jugular vein may be used if upper arms vein are not visible/accessible
• Lower limb veins should be avoided due to impairment of venous return below the diaphragm during cardiac arrest
• Each IV drug must be followed by a flush of at least 20-30mls of normal saline and 2 minutes of external cardiac compression
Intraosseous (IO) access

- Requires specific training on the methods and equipment required
- Preferred route if IV access cannot be obtained
- Attainable in all age groups
- Safe and effective for fluid resuscitation, drug administration and laboratory evaluation
- Please refer to Epworth HealthCare protocols

IO Medication and fluid administration

- The IO cannula should be flushed before and after each medication is administered. Fluids should be administered under pressure using an infusion pump, pressure bag, or manual injection through a syringe and 3 way tap. (Infusion rates equivalent to a 21 gauge peripheral intravenous catheter are typically achieved)
- The IO site and extremity should be monitored for infiltration during rapid fluid administration.

Application of LUCAS 2- External Chest Compression Device

- Please refer to Epworth HealthCare protocol for LUCAS2
- Please refer to Epworth HealthCare protocol for instructional video on LUCAS 2
Advanced Life Support for Adults

Start CPR
- 30 compressions: 2 breaths
- Minimise Interruptions

Attach
Defibrillator / Monitor

Assess Rhythm

Shockable

Shock

CPR for 2 minutes

Non Shockable

CPR for 2 minutes

Return of Spontaneous Circulation?

Post Resuscitation Care

During CPR
- Airway adjuncts (LMA / ETT)
- Oxygen
- Waveform capnography
- IV / IO access
- Plan actions before interrupting compressions (e.g. charge manual defibrillator)

Drugs
- **Shockable**
  - Adrenaline 1 mg after 2nd shock
  - Amdarone 300mg after 3 shocks
- **Non Shockable**
  - Adrenaline 1 mg immediately
  - (then every 2nd loop)

Consider and Correct
- Hypoxia
- Hypovolaemia
- Hyper / hypokalaemia / metabolic disorders
- Hypothermia / hyperthermia
- Tension pneumothorax
- Tamponade
- Toxins
- Thrombosis (pulmonary / coronary)

Post Resuscitation Care
- Re-evaluate ABCDE
- 12 lead ECG
- Treat precipitating causes
- Aim for: SpO2 94-98%, normocapnia and normoglycaemia
- Targeted temperature management

January 2016
Establish / Treat Causes as Early as Possible:

4 Hs and 4 Ts

Hypovolaemia (crystalloids / colloids, 20ml/kg)
Hypoxaemia (oxygen, ventilation)
Hypo/Hyperthermia
Hypo/Hyperkalaemia and metabolic disorders

Acidosis (sodium bicarbonate)

Thromboembolism Pulmonary /Coronary (Administer volume, and thrombolytics or anticoagulation)
Tension pneumothorax (needle decompression, then ICC placement)
Pericardial Tamponade (emergency pericardiocentesis, or prepare for resternotomy in post cardiac surgical patient)
Toxins / poisons / drugs (specific antidotes or supportive management)
NON SHOCKABLE RHYTHMS

Asystole

“Asystole is characterised by the absence of any cardiac electrical activity” (ARC Guideline 11.2 2010)

Pulseless Electrical Activity (PEA)

(Sometimes referred to as Electromechanical Dissociation EMD)

“PEA is the presence of a coordinated electrical rhythm without a detectable cardiac output” (ARC Guideline 11.2 2010)

Initiate Basic Life Support Measures

Verify rhythm

For Asystole

• Observe more than one lead, ensure leads connected
• Increase gain/amplitude

For Pulseless Electrical Activity (PEA)

• Verify rhythm

Management

• Continue CPR
• IV or IO access
• Adrenaline 1 mg IV/IO push immediately
• Repeat Adrenaline 1 mg IV/IO push, every 2nd cycle
• Check ABGs and Serum electrolytes
Establish and treat causes as early as possible

Consider

- Advanced airway management
- Calcium Chloride 10% 5-10mls, IV/IO push for
  - Hyperkalaemia
  - Hypocalcaemia
  - Overdose of Calcium Channel-blocking drugs
- Sodium Bicarbonate 8.4% (1mmol/mL) IV/IO 1mmol/kg over 2-3 minutes, via a dedicated line for
  - Hyperkalaemia
  - Treatment of documented metabolic acidosis
  - Overdose with tricyclics depressants
  - Protracted arrest (greater than 15 minutes)
- Fluid if suspicion of hypovolaemia (20mL/kg)
- Thrombolytics if proven or suspected PE
SHOCKABLE RHYTHMS

Ventricular Fibrillation

Unconscious, Pulseless Ventricular Tachycardia

“Ventricular fibrillation is asynchronous chaotic ventricular activity that produces no cardiac output” (ARC Guideline 11.2 2010)

“Pulseless ventricular tachycardia is a wide complex regular tachycardia associated with no clinically detectable cardiac output” (ARC Guideline 11.2 2010)

Initiate Basic Life Support measures – if there are no signs of life

Management

Immediate defibrillation

- 1st defibrillation, single shock of
  - 200 Joules for Biphasic defibrillator
  - 360 Joules for Monophasic defibrillator
- Continue CPR for 2 minutes
- IV or IO access
- 2nd defibrillation
- Adrenaline 1 mg IV/IO push (after 2nd defibrillation)
- Check ABGs and Serum electrolytes
- 3rd defibrillation
- Amiodarone 300mg IV/IO bolus,
- Repeat defibrillation every cycle, (2 minutes)
- Repeat Adrenaline 1 mg IV/IO push, every 2nd cycle (every 4 minutes)
Establish and treat causes as early as possible

Consider

- Advanced airway management
- 2\textsuperscript{nd} dose of Amiodarone 150mg IV/IO bolus and infusion at 15mg/kg/24hours (max dose 1200mg)
- Potassium Chloride
  - Persistent VF due to documented or suspected hypokalaemia
  - Give 5mmol IV/IO bolus
- Magnesium Chloride
  - Torsade de points
  - Cardiac arrest associated with digoxin toxicity
  - VF and pulseless VT refractory to defibrillation and vasopressor
  - Documented hypokalaemia
  - Documented hypomagnesium
  - Give 5mmol IV/IO bolus
  - May be repeated once
  - Infusion of 20mmol over 4 hours
- Fluid if suspicion of hypovolaemia -20ml/kg
- Sodium Bicarbonate 8.4\% (1mmol/mL) IV/IO 1mmol/kg over 2-3 minutes, must not come in contact with other drugs, for
  - Hyperkalaemia
  - Treatment of documented metabolic acidosis
  - Overdose with tricyclics depressants
  - Protracted arrest (greater than 15 minutes)
- Thrombolytics if proven or suspected PE
- Lignocaine:
  - May be considered if Amiodarone cannot be used
  - Prophylaxis in the setting of recurrent VF or VT
  - 1mg/kg IV/IO bolus
  - May give an additional bolus of 0.5mg/kg IV/IO (Max dose of 300mg to be administered in 1 hour)
TACHYARRHYTHMIAS

A tachyarrhythmia may originate from the atria, for example Supraventricular Tachycardia (SVT), Sinus Tachycardia (ST) or Atrial Fibrillation (AF). Tachyarrhythmias may also originate from the ventricles, for example Ventricular Tachycardia (VT) or Ventricular Fibrillation (VF).

Whilst assessing patient consider the following

- Verify rhythm & vital signs
- Assess circulation, chest pain, and other symptoms, such as syncope, shortness of breath, dizziness, and palpitations
- Give O2 therapy via mask
- Ensure IV/IO access
- Obtain ECG (without delaying treatment)

The following adverse features will require immediate treatment

- Systolic BP < 90mmHg
- Heart rate > 150/min (if symptomatic)
- Chest pain
- Heart failure
- Drowsiness or confusion

Management

- Synchronised cardioversion with sedation
- If unsuccessful, Amiodarone 300mg IV over 10-20min in Glucose 5%
- Repeat synchronised cardioversion
- Amiodarone infusion 1000mg over 24 hours
**Broad Complex Tachycardia**

- Could be supraventricular tachycardia with aberrant conduction (ie. bundle branch block BBB)
- In peri-arrest setting assumption is of ventricular origin
- Regular broad complex tachycardia
  - In the absence of adverse features, treat with Amiodarone 300mg IV/IO, over 20-60min (or as directed by medical team)
  - Follow by Amiodarone infusion 1000mg in 100ml Glucose 5% over 24 hours
  - Continue to monitor and assess patient
  - Seek medical help and transfer to a monitored area
  - If unstable or adverse features develop - Synchronised Cardioversion
- Irregular broad complex tachycardia
  - Most likely Arial Flutter/Atrial Fibrillation with BBB
  - Other possible causes are AF with ventricular pre-excitation (eg Wolff-Parkinson-White Syndrome) or polymorphic VT (i.e Torsades de Points)
  - Seek medical advice
  - Synchronised cardioversion is the safest management
  - Avoid adenosine, digoxin, verapamil and diltiazem in pre-excited AF or Atrial Flutter (since these drugs block the AV node and may cause a relative increase in pre-excitation)
**Torsades De Pointes**

“Torsades de pointes is a specific type of ventricular tachyarrhythmia that occurs in the setting of acquired or congenital prolongation of the QT interval on the ECG” (ARC Guideline 11.2 2010)

- Self limiting or sustained can cause cardiac arrest (management according to VF or pulseless VT algorithm)
- Stop all drugs known to prolong QT
- Correct electrolytes abnormalities especially hypokalaemia and other causes i.e ischaemia
- Magnesium chloride 5mmol IV/IO over 10min, repeated once and then an infusion of 20mmol over 4 hours
- Emergency pacing or isoprenaline may be indicated:
  - For Heart block and symptomatic bradycardia presenting with Torsades de Pointes
  - To prevent relapse
- Avoid Amiodarone as it may make the situation worse
Narrow Complex Tachycardia

Regular narrow complex tachycardia

- Sinus Tachycardia
- AV nodal re-entry tachycardia (AVNRT, i.e SVT)
- AV re-entry tachycardia (AVRT, WPW Syndrome)
- Atrial flutter with regular AV conduction (usually 2:1) (ARC Guideline 11.9 2009)

Management

- If unstable with adverse features, synchronised cardioversion with sedation except for ST
- If stable
  - Vagal manoeuvres
  - Adenosine 6mg, IV bolus with 20ml flush
  - If ventricular rate slows, look for atrial activity
  - Adenosine may be repeated twice, at 12mg, IV rapid bolus
  - If Adenosine is contraindicated, consider Verapamil 2.5-5mg IV over 2min, or Diltiazem 15-20mg over 2min

Irregular narrow complex tachycardia

- Most common is AF and sometimes atrial flutter with variable AV conduction
- If unstable with adverse features, synchronised cardioversion with sedation
- If there are no adverse features
  - Rate control by drug therapy
    - IV or oral beta blockers
    - Digoxin 250-500mcg IV or oral
    - If onset < 48hours Amiodarone 300mg IV over 20-60min, then infusion 1000mg over 24 hours in divided dose
  - Rhythm control using drugs to encourage chemical cardioversion
  - Rhythm control by electrical cardioversion
  - Treat to prevent complications (eg. Anticoagulation)
BRADYARRHYTHMIAS

Bradyarrhythmia is defined as a heart rate of less than 60/min, which include: AV blocks, in particular 2\textsuperscript{nd} degree type 2 and 3\textsuperscript{rd} degree. The common symptoms with bradycardia include syncope, shortness of breath, dizziness or chest pain.

When assessing patient consider the following

- Verify rhythm and vital signs
- Assess circulation, chest pain, and other symptoms, such as syncope, shortness of breath, dizziness, and palpitations
- Give 02 therapy via mask
- Ensure IV/IO access
- Obtain ECG (without delaying treatment)

The following adverse features require immediate treatment:

- Systolic BP < 90mmHg
- Heart rate < 40/min
- Ventricular arrhythmia
- Heart failure

Management of conscious/symptomatic bradyarrhythmia

- Atropine 500-600mcg IV bolus, repeated every 3-5 minutes to a total of 3mg
- Low dose adrenaline (bolus or infusion) at rate of 2-10mcg/min
- Isoprenaline 2-5mcg/minute
- Pacing is indicated if
  - Recent asystole
  - Mobitz II AV block
- Complete AV block
- Ventricular standstill of > 3sec

**To commence pacing**

- Turn the defibrillator ‘On’
- Increase rate to 70-90 beats/minute
- Start pacing
- Increase output until 100% capture
- Consider demand/non demand pacing (refer to section on pacing)

**Unconscious Bradycardia**

A pulseless (unconscious) bradycardia is managed as per non-shockable cardiac arrest algorithm.
**Bradycardia Algorithm**

*(includes rates inappropriately slow for haemodynamic state)*

If appropriate, give oxygen, cannulate a vein, and record a 12-lead ECG

If adverse signs are present:

- Systolic BP < 90 mmHg
- Heart rate < 40 beats min⁻¹
- Ventricular arrhythmias compromising BP
- Heart failure

1. **Atropine 500 mcg IV**
2. **Satisfactory response?**
   - **YES**
   - **NO**

   **Risk of asystole?**
   - Recent asystole
   - Mobitz II AV block
   - Complete heart block with broad QRS
   - Ventricular pause > 3s

3. **Interim measures:**
   - Atropine 500 mcg IV** repeat to maximum of 3 mg
   - Adrenaline 2-10 mcg min⁻¹
   - Alternative drugs * OR
   - Transcutaneous pacing

4. **Seek expert help**
   - **Arrange transvenous pacing**

* Alternatives include:
  - Aminophylline
  - Isoprenaline
  - Dopamine
  - Glucagon (if beta-blocker or calcium-channel blocker overdose)
  - Glycopyrrolate can be used instead of atropine

**500-600 mcg**

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**Australian Resuscitation Council**

**Guideline 11.9**

**November 2009**
**Tachycardia Algorithm (with pulse)**

**Unstable**

- Amiodarone 300mg IV over 10-20 min and repeat shock, followed by:
- Amiodarone 900mg over 24 h

**Stable**

- **Is Patient Stable?**
  - Signs of instability include:
    1. Reduced conscious level
    2. Chest pain
    3. Systolic BP < 90 mmHg
    4. Heart failure
  - Rate-related symptoms uncommon at less than 150 beats/min

**Is QRS narrow (< 0.12 sec)?**

- **Narrow**
  - **Regular**
    - Use vagal manoeuvres
    - Adenosine 6 mg rapid IV bolus; if unsuccessful give 12 mg; if unsuccessful give further 12 mg.
    - Monitor ECG continuously
  - **Irregular**
  - Irregular Narrow Complex Tachycardia
    - Probable atrial fibrillation
    - Control rate with:
      - β-Blocker IV or digoxin IV
      - If onset < 48 h consider
    - Amiodarone 300mg IV 20-60min; then 900mg over 24 h
  - Seek expert help

- **Broad**
  - **Seek expert help**

**Possibilities include:**
- AF with bundle branch block treat as for narrow complex
- Pre-excited AF consider amiodarone
- Polymorphic VT (e.g. torsade de pointes – give magnesium 2g over 10 min)

**If Ventricular Tachycardia (or uncertain rhythm):**
- Amiodarone 300 mg IV over 20-60 min; then 900 mg over 24 h
- If previously confirmed SVT with bundle branch block:
  - Give adenosine as for regular narrow complex tachycardia

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*Adapted from Australian Resuscitation Council Guideline 11.9 (November 2009)*

For correct dose of Magnesium see page 18 of this package

Amiodarone dose as per hospital policy
DEFIBRILLATION

Defibrillation is the therapeutic use of electrical current to depolarise the entire myocardium, permitting the dominant (sinus) pacemaker to resume control.

Indications

Defibrillation has been proven to be the definitive therapy for treatment of cardiac arrest secondary to VF and pulseless VT. International resuscitation guidelines clearly emphasise the importance of early BLS, especially compressions and early defibrillation for the successful treatment of such patients.

Early BLS and defibrillation is vital to survival from a cardiac arrest as:

- The most frequent initial rhythm with a witnessed sudden cardiac arrest is VF. The vast majority of survivors come from this group (ARC ALS guideline 11.2; 2010).
- The highest possible survival rate of cardiac arrest is achieved through early defibrillation (ARC ALS guideline 11.2; 2010).
- If patients with VF are defibrillated within one to two minutes of the onset of VF there is a 90% survival rate. However the chance of a successful defibrillation diminishes rapidly over time (Harrison, G. A., 1998; & Baskett, P, (Ed). 2000).

Energy, current and voltage

When delivering a shock, a large flow of electrons (current - measured in milliamperes) passes through the heart over a brief period.

The driving force of the current is referred to as the electrical potential or voltage.

If the energy and current are too low, the shock will not terminate the arrhythmia. If energy and current are too high, functional and morphological damage may result. There is no clear relationship between body size and energy requirements for adults. Studies have shown that the appropriate energy level is 200J for a biphasic defibrillator and 360J for a monophasic defibrillator. (ARC ALS guideline 11.2; 2010).

At Epworth HealthCare the majority of defibrillators are biphasic.

Factors influencing defibrillation outcomes
• Duration of VF / Pulseless VT
• Chest wall (transthoracic) impedance
• Current flow is determined by the energy chosen and the transthoracic impedance (measured in ohms). Many factors determine transthoracic impedance:
  • Energy delivered
  • Electrode skin coupling material
  • Number and time interval of previous shocks
  • Phase of ventilation
  • Distance between electrodes (size of chest)
  • Skin resistance factors such as hair, moisture and gel can also increase or decrease impedance
  • **Electrode size:** The larger the electrode, the less the impedance. Electrodes are usually about 8.5 cm by 12 cm (adult)
  • **Electrode Position:** The electrodes should be placed in a position that will maximise current flow through the myocardium. The recommended placement is on the apex and sternum. The sternal electrode is placed to the right of the upper part of the sternum below the clavicle over the 2\(^{nd}\) intercostal space and the apex electrode (the negative electrode) is placed anteriorly over the left of the nipple with the centre of the electrode over the mid-axillary line in the 5\(^{th}\)-6\(^{th}\) intercostal space. See next page.

If the patient has a permanent pacemaker (PPM) or implantable cardiac defibrillator (ICD) insitu, the recommended electrode position is either the conventional sternal / apical position or the anterior-posterior position. In the anterior – posterior position, the ‘apical’ electrode is placed mid-clavicular line 5\(^{th}\)-6\(^{th}\) intercostal space, and the ‘sternal’ electrode is placed in the back, sub-scapular area mid scapular line. Make sure the electrodes are placed 8cm away from PPM / ICD or other devices (ARC Electrical Therapy for Adult Life Support Guideline 11.4; page 2, 2010).
Defibrillation pad placement

Please note: PPM’s have a suppressor circuit which limit the amount of current that enters the pulse generator. Defibrillation can interfere indirectly as a result of myocardial injury lead fracture or displacement.

**Biphasic waveform defibrillation**

Most current defibrillators have a biphasic waveform due to improved effectiveness (82% 1st shock reversion compared with 66% of monophasic devices) and cause less myocardial tissue damage (AHA, 2000. p 110). The majority of biphasic devices are capable of compensating for varying levels of impedance and will adjust delivery and level of voltage to deliver set joules.

**The defibrillator**

The AC power charges the battery only. It does not supply energy for the shock, therefore the defibrillator does not have to be plugged in whilst in use. The battery (DC power source) supplies the energy for the shock therefore the defibrillator must be on AC charge when not in use.
Technique for performing defibrillation in emergency DCR

1. Identify the machine as biphasic or monophasic
2. Expose chest and check for lines, pacemakers, excessive hair, water, skin patches etc
3. Stick defibrillation pads on chest and connect cable
4. Select 200J (for biphasic) or 360J (for monophasic).
5. Press charge button Call loudly: “Charging” (Charging should take less than 10 seconds).
   Continue compressions during charging
6. Call loudly “stand clear” – visual check to ensure that all persons providing chest compressions and breathing stand clear. Remove oxygen.
7. Verify rhythm (shockable/ non-shockable).
8. If shockable -Press shock
9. If non-shockable press disarm button
10. Immediately commence 2 mins CPR
11. Repeat from step 5

If the defibrillator failed to discharge:

Troubleshoot and recharge immediately and try again. Do not interrupt chest compressions whilst troubleshooting.

Causes of failure to discharge

- Defibrillator not turned on
- Joules not dialled up
- Waited more than 30 seconds to deliver shock resulting in device disarming
- Cables not attached properly or fractured cables
- In synchronised mode and waiting for an ‘R’ wave
- Internal failure – battery not charged
- Attempt to shock before fully charged
- Poor contact with chest wall – short circuit of current

Failure to revert rhythm

- Incorrect electrode position
- Non-responsive myocardium
Adverse effects and safety precautions

Burns to patients may occasionally be experienced at the site of application of the electrodes as a result of excessive current passing through the area. This may be due to:

- Inadequate application of pads (change pads after 8 shocks)
- Passage of portion of current through ECG monitoring electrodes. In this case, the burn appears at the site of the electrodes.

Electrocution of patient and bystanders

Shock may be experienced by staff through one or more of the following:

- By contact with the electrodes while defibrillation is being performed
- By contact with the patient while defibrillation is being performed
- By contact with other conductive devices attached to the patient, including bed sides, bag valve mask and O2 connection and ECG leads

Issues of safety

- Avoid allowing oxygen from BVM to flow onto the person’s chest during the delivery of the shock
- During discharge, all personnel should be kept clear of the patient. Ensure CPR is paused and ventilation is paused prior to shock
- Avoid placing the patient in contact with metal fixtures, e.g. bedrails

Paper recorder

Summary provides strips of all rhythms pre and post defibrillation.
TRANSCUTANEOUS PACING

Transcutaneous pacing is an emergency procedure used to supplement the patient’s heart rate when bradycardia compromises cardiac output.

Transcutaneous pacing is more likely to be effective earlier in an arrest situation. Therefore it should be initiated at the same time that the first line treatments are being commenced.

Transcutaneous pacing is also known as external, non-invasive pacing. A defibrillator with pacing capability is attached to the patient using multifunction adhesive pads. The adhesive pads have a conductive gel that provides a low resistance interface between the machine and the patient’s skin. Energy is passed from the machine from one pad to the other. The pads are placed in such a way that the energy passes through the heart. If the energy is sufficient it will depolarise the myocardium to cause myocardial contraction.

To commence pacing

Pad placement and adhesion

The interface between the adhesive pads and the patient is one of the important parts of transcutaneous pacing.

- Expose the patient’s chest
- Clip excess hair from the patient’s chest. Hair can lift the pad off the chest and provides extra resistance to energy flow from the pad to the patient
- Dry the chest. Excess moisture on the patient’s chest can lead to the pads not adhering properly to the patient’s chest and run the risk of coming off mid therapy. Moisture can also provide a low resistance pathway for the pacing energy to pass over the chest wall rather than through it
- Attach the adhesive electrodes to the appropriate positions so the whole surface is in contact with the skin (i.e. no air pockets)

Electrode positioning

The pacing pads can be applied posterior-anteriorly or in the standard sternum/apex position.
Posterior-Anterior placement

The apex electrode (negative electrode) is placed anteriorly below the left of the nipple with the centre of the electrode over the mid-axillary line in the 5\textsuperscript{th}-6\textsuperscript{th} intercostal space. The positive electrode is placed posteriorly, opposite the anterior electrode, between the scapula and the spine (5\textsuperscript{th} to 6\textsuperscript{th} intercostal space).

Posterior-Anterior pad placement

The sternal/apex position

The same standard position for placement of defibrillator pads is used. Importantly, the negative electrode should be placed at the apex and the positive at the right sternal border, below the clavicle. This placement of the negative electrode at the apex ensures it is close to the heart and maximises current density to the myocardium, increasing the chance of capture.

Sternal/Apex pad placement
Safety considerations

- Do not place the pads over implanted pacemakers or AICDs (automatic internal cardiac defibrillators), this could lead to malfunction of the implanted device, or provide a low resistance pathway of concentrated energy to the heart via the pacing lead. Pads must be placed at least 8 cm from the implanted device.

- Do not place the pads over ECG electrodes, medication patches, jewellery or tapes, indwelling lines, as burning is likely to occur.

Connection of pads

Connect the electrodes to the pacer via the connecting cable.

ECG connection

Establish the ECG signal via the 3 lead ECG monitoring cable. The ECG signal is required on the pacer/defibrillator to commence demand pacing and is necessary for identifying electrical capture. To use the pacer mode on the defibrillator you need to have both the monitoring leads and pads attached to patient.

Establishing pacing

Individual devices have slightly different procedures that need to be followed, although all have the following steps to take to establish pacing and gain capture:

- Turn the machine to pacer (pacer on)
- Select demand mode (it usually defaults to demand mode)
- Set the heart rate you wish to achieve (usually 80bpm)
- Press start
- Increase the pacer current (output) until 100% myocardial capture is obtained
- Increase output by 10 milliamps as safety margin
- Once capture is obtained the patient’s pulse should be assessed to confirm cardiac output
- If electrical capture is attained and no cardiac output is apparent commence CPR

The ECG appearance of captured paced rhythm will show a pacing spike followed by a broad complex QRS complex. The ECG rhythm should be read via the pacemaker/defibrillator.
Intermittent failure to capture

Factors affecting capture

Factors that affect the effectiveness of transcutaneous pacing are

- Pad positioning - posterior - anterior is more likely to gain capture than sternal /apex positioning
- Level of energy delivered - the higher the output the greater the chance of capture
- Adhesion of the pad with the skin
- Bonding of the gel with the skin
- Size of the patient’s chest
- Size of the adhesive pads
- Dryness of the gel pads
- Phase of respiratory cycle
- Level of responsiveness of the heart

Length of therapy

Transcutaneous pacing is designed as a temporary measure to establish a reliable heart rhythm, providing time to institute a more invasive/reliable form of pacing.
Effort should be taken to ensure that transcutaneous pacing is required for the shortest time possible.

**Pacing modes**

Transcutaneous pacemakers have two modes of pacing: demand mode and fixed mode.

Demand mode is where the pacemaker will pace only when the patient’s heart rate drops below the set rate on the pacemaker. This differs from fixed mode where the pacemaker does not have “sensitivity” and will pace at its set rate despite the patient’s intrinsic heart rhythm. Risk of the fixed mode is pacing may occur on the T wave causing arrhythmias, therefore demand pacing is preferable and fixed pacing should only be used where demand pacing cannot be established.

The pacer in demand mode is reliant on an ECG monitoring lead to sense the underlying rhythm. The quality of the trace is important as interference can lead falsely inhibiting pacing.

**Other considerations**

Transcutaneous pacing evokes skeletal muscular contraction and varying degrees of pain and discomfort. The use of analgesia and sedation should be considered for all patients receiving transcutaneous pacing. The level of discomfort experienced by the patient is associated with the amount of energy used to achieve capture. Therefore using the least amount of energy to gain capture is desirable.

The patient can be touched during the use of transcutaneous pacing as only a small charge is passed through the patient whilst being paced. Carers should however avoid touching the pads during use, as a small (non dangerous) shock may be experienced.
RESUSCITATION MEDICATIONS

Please note:
In compliance with Victorian Legislation (Secretary Approval Cardiac Arrest, 13 July 2015, ratified by Epworth Standard 9 Working Party, Feb 2016) in the absence of a Doctor, Adrenaline and Amiodarone ONLY can be administered by a Registered Nurse with current ALS training and assessment (within previous 12 months). Medications must be administered in accordance with the ARC ALS guidelines.

- Adrenaline
- Amiodarone
- Calcium chloride
- Lignocaine
- Potassium chloride
- Magnesium chloride
- Sodium bicarbonate
- Atropine
- Isoprenaline
- Adenosine
ADRENALINE

Action

Adrenaline is a natural catecholamine with both alpha and beta adrenergic activity. It can produce the following cardiovascular responses:

- Increased systemic vascular resistance
- Increased systolic blood pressure
- Increased coronary and cerebral blood flow
- Increased strength of myocardial contraction
- Increased automaticity and heart rate
- It may aid defibrillation by increasing myocardial blood flow during CPR

Indications

- Shockable rhythms: ventricular fibrillation/pulseless ventricular tachycardia after initial defibrillations have failed (after the 2nd shock then every second cycle/4 minutes)
- Non shockable rhythm: asystole and pulseless electrical activity (PEA) give immediately (then every second cycle)
- Anaphylactic shock
- Bradycardia

Pharmacokinetics

- Onset: rapid
- Duration: 5 minutes
- Half life: approximately 3 - 5 minutes
- Metabolised by the liver and sympathetic nerve endings
- Excreted by the kidneys

Presentation

- Mini-jet/ pre-filled syringe – 10ml of 1:10000 solution = 1mg/10mL
- Ampoule – 10ml of 1:10000 solution = 1mg/10mL
- Ampoule – 1ml of 1:1000 solution = 1mg/1mL
Cardiac arrest dose

- Bolus = 1mg every 2nd cycle (every 4 minutes) during CPR, flushed with 20-30ml of IV fluid
- Infusion via CVC preferred) = 1 to 20mcg/min (6mg in 100ml of Glucose 5% or Sodium Chloride 0.9%)
- 1mL of prepared infusion solution = 60mcg/mL. When infused at 1mL/hr = 1mcg/min

Non-cardiac arrest dose

- Infusion = 2 to 10 mcg/min (6mg in 100ml of Glucose 5% or Sodium Chloride 0.9%)
- 1mL of prepared infusion solution = 60mcg/mL. When infused at 1mL/hr = 1mcg/min

Route

- IV
- IO (intraosseous)

Precautions

- May precipitate angina in the patient with ischaemic heart disease

Adverse Effects

- Tachyarrhythmia
- Severe hypertension after resuscitation
- Tissue necrosis if extravasation occurs
AMIODARONE

Action

It is a class III antiarrhythmic agent prolonging the duration of phase 3 and 4 action potential affecting the refractory period in atrial, nodal and ventricular tissues, therefore giving a very broad spectrum of activity.

- Sinus node - decreases sinus automaticity
- Atrioventricular node - slows the speed of conduction and increases the refractory period in the AV node
- His-Purkinje system - lengthens the refractory period but does not modify the speed of conduction of the system

Indications

- Shockable rhythm after the 3rd defibrillation
- Prophylaxis of recurrent VF/VT (conscious/unconscious)

Pharmacokinetics

- Onset: Erratic, generally prompt
- Peak: 30 minutes
- Duration: 10 - 150 days
- Half life: 25 - 110 days
- Metabolised in the liver
- Excreted by the lacrimal glands, the skin and the biliary tract

Presentation

- Ampoule - 150mg/3mL

Dose

- 300mg IV/IO bolus **Must always be diluted in 10 – 20 mL** Glucose 5%
- Subsequent dose = 150mg IV/IO bolus
- Infusion = 15mg/kg over 24 hours (maximum 1200mg)
Infusion preparation

- 1000mg in 100ml of **Glucose 5%**
- Must be delivered by a volumetric infusion pump

Route

- IV
- IO

Precautions

- Peripheral route only in cardiac arrest situation
- Central line is very strongly recommended in non cardiac arrest situations
- Not compatible with any other drug
- **Not compatible with Sodium Chloride 0.9%**

Side Effects

- Hypotension
- Bradycardia
- Heart Block
CALCIUM CHLORIDE

Action
Calcium is essential for the function of both the nervous and muscular systems. Calcium ions increase myocardial excitability, contractility and peripheral resistance.

Indications
- Hyperkalaemia
- Hypocalcaemia
- Overdose of calcium-channel blocking drugs

Pharmacokinetics
- Onset: immediately
- Peak: immediately
- Duration: 0.5 – 2 hours
- Half life: unknown
- Metabolised by the liver
- Excretion is mainly in the faeces

Presentation
- Glass Vial- Calcium chloride 10% (1g in 10mL)

Dose
- IV bolus 0.5g - 1g (5-10mL of 10% solution)

Route
- IV
- IO

Precautions
- Tissue necrosis with extravasation
- Strictly contraindicated in patients treated with digoxin (risk of toxicity)
Adverse Effects

- Possible increase in myocardial and cerebral injury by mediating cell death
- Arrhythmias
- Syncope
- Hypercalcaemia
LIGNOCAINE

Action
A class 1B antiarrhythmic that depresses phase 0 of the action potential. It shortens the action potential and acts as a sodium channel blocker. Lignocaine has membrane stabilising effects therefore:

- Suppresses ventricular arrhythmias
- Blocks both activated and inactivated sodium channels

Indications

- Shockable rhythm when amiodarone cannot be used
- Prophylaxis in the setting of recurrent VF or VT

Pharmacokinetics

- Onset: immediate
- Duration: 10 – 20 minutes
- Elimination half life: 100 minutes
- Metabolised by the liver
- Excreted by the kidneys (NB: Dose modification if liver/renal dysfunction)

Presentation

- Ampoule 1% 50mg/5mL
- Pre-mixed Lignocaine Infusion – 0.4% Lignocaine (2 grams) in 500mls 5% Glucose

Dose

- Initial = 1mg/kg IV bolus
- An additional bolus dose of 0.5 mg/kg maybe given

Route

- IV
- IO
Adverse Effects

- Hypotension
- Bradycardia
- Heart block
- Asystole
- Slurred speech
- Altered consciousness
- Muscle twitching
- Seizures
POTASSIUM CHLORIDE

Action

Potassium is an electrolyte essential for membrane stability. It has several roles and influences on cardiac function by;

- Regulating transmembrane potential and nervous and muscle impulses
- Regulating automaticity, excitability and conduction velocity
- Hypokalaemia increases atrial and ventricular irritability, and may cause atrial and ventricular ectopy or tachyarrhythmias.

Indications

- Persistent VF
- Cardiac arrest (when hypokalaemia is suspected or confirmed with ABGs
- Hypokalaemia suspected or documented

Presentation

- 10mmol in 100mL of Sodium Chloride 0.9%
- Ampoule - 10mmol/10mL **Dose**
- 5mmol IV/IO bolus or as guided by ABGs

Route

- IV
- IO

Precautions

- Extravasation may lead to tissue necrosis

Adverse Effects

- Bradycardia
- Hypotension
- Possible asystole
MAGNESIUM CHLORIDE

Action

Magnesium is an electrolyte essential for membrane stability. It has similar properties to class III antiarrhythmic drugs as it;

- Delays the conduction time through the AV node
- Lengthens the refractory time in the atria and ventricles, decreasing ectopic firing
- Protects against proarrhythmic influence of long QT interval

Indications

- Torsades de Pointes
- Cardiac arrest associated with digoxin toxicity
- VF/pulseless VT (usually administered when refractory to defibrillation and adrenaline)
- Documented hypokalaemia
- Documented hypomagnesiaemia

Pharmacokinetics

- Onset: immediate
- Duration: 30 minutes
- Half life: 5-7 minutes
- Excreted by the kidneys

Presentation

- Ampoule – 480mg/5mL = 5mmol magnesium ions and 10mmol chloride ions

Cardiac arrest dose

- 5mmol Diluted in 10 to 20ml Glucose 5% /Sodium chloride 0.9%
- Dose may be repeated once

Non-cardiac arrest dose

- Infusion 20mmol over 4 hours
Route

- IV
- IO

Adverse Effects

- Muscle weakness
- Respiratory failure
SODIUM BICARBONATE

Action
Sodium bicarbonate is an alkalising solution, which combines with hydrogen ions to form a weak carbonic acid. This breaks down to produce CO₂ and H₂O. In most cardiac arrests, early efficient CPR and adequate ventilation negate the need for any NaHCO₃.

Indications
- Treatment of documented metabolic acidosis
- Overdose with tricyclics antidepressants
- Protracted arrest (greater than 15 minutes)
- Hyperkalaemia

Pharmacokinetics
- Onset: immediate
- Peak: rapid
- Duration: unknown
- Half life: unknown

Presentation
- Glass vial – 8.4% 100ml 1mmol/1ml

Dose
- 1mmol/kg of 8.4% solution given over 2-3 minutes followed by a 20-30mL flush
- Then as guided by Arterial Blood Gasses

Route
- IV
- IO

Precautions
- Sodium Bicarbonate when mixed along with adrenaline or calcium may inactivate each other or precipitate and block the IV line
Adverse Effects

- Metabolic alkalosis
- Hypokalaemia
- Hypernatraemia
- Hyperosmolarity
- Intracellular acidosis may develop or worsen when the CO2 liberated from NaHCO3 freely enters the cells
ATROPINE

Action

An anticholinergic, it inhibits acetylcholine at the parasympathetic neuro-effector junction therefore it;

- Blocks the vagal effects on the sinoatrial node and AV node
- Increases sinus rate and enhances conduction through the AV node
- May overcome vaso-vagal mediated vasodilation

Indications

- Symptomatic bradycardias and AV block

Pharmacokinetics

- Onset: 2 - 4 minutes
- Duration: 4 – 6 hours
- Half life: 2 – 3 hours
- Metabolised by the liver
- Excreted by the kidneys, small amounts in expired air as carbon dioxide and in faeces

Presentation

- Ampoule – 600 mcg in 1ml

Dose

- Bolus 500-600mcg every 3-5 minutes, up to the maximum dose of 3 mg

Route

- IV
- IO

Precautions

- Dilation of pupils may mask neurological status
- May cause confusion and hallucinations
- Low doses of the drug may worsen AV block and possibly decrease heart rate
• Cardiac transplant patients will not respond to vagal blockade as the heart is denervated

**Adverse Effects**

• Tachycardia
• Dry mouth
• Papillary dilation and blurred vision
• Urinary retention
ISOPRENALINE

Action
Isoprenaline is a beta 1 & 2 stimulant. It tends to maintain or increase systolic BP and decreases diastolic BP by lowering peripheral vascular resistance. It increases automaticity and atrioventricular nodal conduction and usually improves coronary blood flow.

Indications
- Symptomatic bradyarrhythmias

Pharmacokinetics
- Onset: Rapid 20 – 40 seconds
- Duration: effects persist for a few minutes after IV administration
- Half life: approximately 2 minutes

Presentation
Ampoule - 1mg in 5mL or 200mcg in 1mL

Dose
- IV/IO infusion 2-5 mcg/min (Titrated to heart rate)
- Infusion: 6 mg in 100mLs Glucose 5% (1mL/hr = 1 mcg/min)

Route
- IV
- IO

Precautions
- Ischaemic heart disease
- Tachyarrhythmias

Side Effects
- Hypertension
- Tachyarrhythmias
- Angina
- Sweating/facial flushing
- Mild tremor/nervousness/headache
- Hyperglycaemia
**ADENOSINE**

**Action**
Depresses the sinus node activity and slows conduction through the atrioventricular node.

**Indications**
- Initial drug management of Supraventricular Tachycardia

**Pharmacokinetics**
- Half life < 10 seconds
- Removed rapidly from circulation and not affected by renal or hepatic impairment

**Presentation**
- Ampoule- 6mg in 2mL

**Dose**
- **IV rapid bolus** 6-12 mg
- Followed by minimum 20mL Sodium Chloride 0.9% flush
- Administer via 3 way tap, large bore cannula (18g, 16g) in Cubital fossa or via CVC line
- If no response to 6mg then a further 12mg dose can be administered (up to 24 mg) if required

**Route**
- IV
- IO

**Precautions**
- Can produce bronchoconstriction in people with Asthma
- May produce transient arrhythmias i.e. ectopic beats, SB, ST, missed beats and AV block
- Should not be used in people with existing AV block, or Sick Sinus Syndrome (unless supported by a permanent pacemaker)
**Side Effects**

Because of the extremely short half life, side effects are usually transient.

- Facial flushing
- Bronchoconstriction
- Bradycardia (including sinus pause)
- Hypotension
- Feeling of impending doom (inform patient)
Other drugs contained on the resuscitation trolley are outlined below.

For further information on the medications, refer to eMIMs or IV injectable handbooks located on the wards.

**Diazepam**
Benzodiazepine, used for short term relief of acute anxiety

**Flumazenil**
Benzodiazepine antagonist, used for reversal of central sedative effects of benzodiazepines, particularly midazolam

**Metaraminol Tartrate (Aramine)**
Potent sympathomimetic, alpha specific, and has potent vasopressor effects and minor positive inotropic effects used to treat hypotension

**Metoprolol**
Beta Blocker, used for hypertension, angina pectoris or arrhythmias

**Midazolam**
Short acting Benzodiazepine, provides conscious sedation for short procedures

**Naloxone**
Opiate antagonist, used to reverse the effects of opiates particularly respiratory effects

**Procainamide**
Class 1A antiarrythmic, used in atrial and ventricular arrhythmias

**Propofol**
Short acting general anaesthetic, used to sedate patients

**Sotalol**
Combined class 2 and 3 antiarrythmic, used for refractory ventricular arrhythmias

**Rocuronium**
Nondepolarising neuromuscular blocker, used as a muscle relaxant when intubating a patient
REFERENCES


Roberts, P.R., Clinical Procedures in Emergency Medicine, 4th Ed, Saunders 2006.


Instruction Manuals

- Philips Heartstart XL
- Hewlett Packard Codemaster
- Cardiac Science Powerheart