AlfredHealth

# Educational Resource Advanced Life Support (ALS) – Adult




**June 2012**

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# INTRODUCTION

The International Liaison Committee on Resuscitation (ILCOR) was founded on November 22, 1992. Its mission is to identify and review international science and knowledge relevant to cardiopulmonary resuscitation (CPR) and emergency cardiovascular care (ECC) and when there is consensus to offer treatment recommendations. Researchers from the ILCOR member councils evaluate resuscitation science in 5-year cycles. The most recent International Consensus Conference was held in Dallas in February 2010.

The Alfred Health Basic and Advanced Life Support Guidelines and Readings aim to provide a comprehensive and consistent approach to the management of cardio-pulmonary resuscitation, and has been developed utilising the evidence-based recommendations from ILCOR (2010), as well as the Australian Resuscitation Council (ARC) policy guideline statements.

## Chain of Survival

The actions linking the adult victim of sudden cardiac arrest with survival are called the adult Chain of Survival. The links in the Chain of Survival used by many resuscitation councils include prevention of the arrest, early recognition of the emergency and activation of the emergency medical services (EMS) system, early and high-quality cardiopulmonary resuscitation (CPR), early defibrillation, rapid advanced life support (ALS), and post-resuscitation care.



### Acknowledgements

A, Ballan, G Bingham, S Brack, E Burke, J Di Nunzio, S Dix, J Gasson, C Glatz, L Howard, M Logan, S Metcalf, S Musgrave, P Ross, G Robers, M Villella, J Watterson, J Williams

# Advanced Life Support (ALS)

ALS is basic life support (BLS) with the addition of defibrillation, intravenous insertion and medications, rhythm recognition and advanced airway management.

**ARC Advanced Life Support for Adults Flowchart (Figure 1)**

| **Shockable rhythms:** | **Non-shockable rhythms:** |
|---|---|
| - Ventricular Fibrillation (VF) | - Asystole |
| - Pulseless Ventricular Tachycardia (VT) | - Pulseless Electrical Activity (PEA) |

**Key points from 2010 ARC Advanced Life Support for Adults flowchart:**

- Attach defibrillator and identify cardiac rhythm. If shockable shock
- Minimise interruption to CPR during interventions
- Precordial thump is not part of the ARC flowchart
- All Alfred Health defibrillators have evidence to support use of 150 joule shocks *during an Emergency Defibrillation*
- CPR is continued during drug administration and is only interrupted briefly during shocking phase of defibrillation
- Attempts to secure airway should not delay CPR and should minimise interruptions to chest compressions
- IV access (large bore antecubital fossa preferably) and fluid line should be obtained as soon as possible

**For shockable rhythms**

- Adrenaline 1mg should be administered after the second shock then after every second cycle (one cycle is 2 minutes of compressions and ventilations)
- Amiodarone 300mg is administered after the third shock

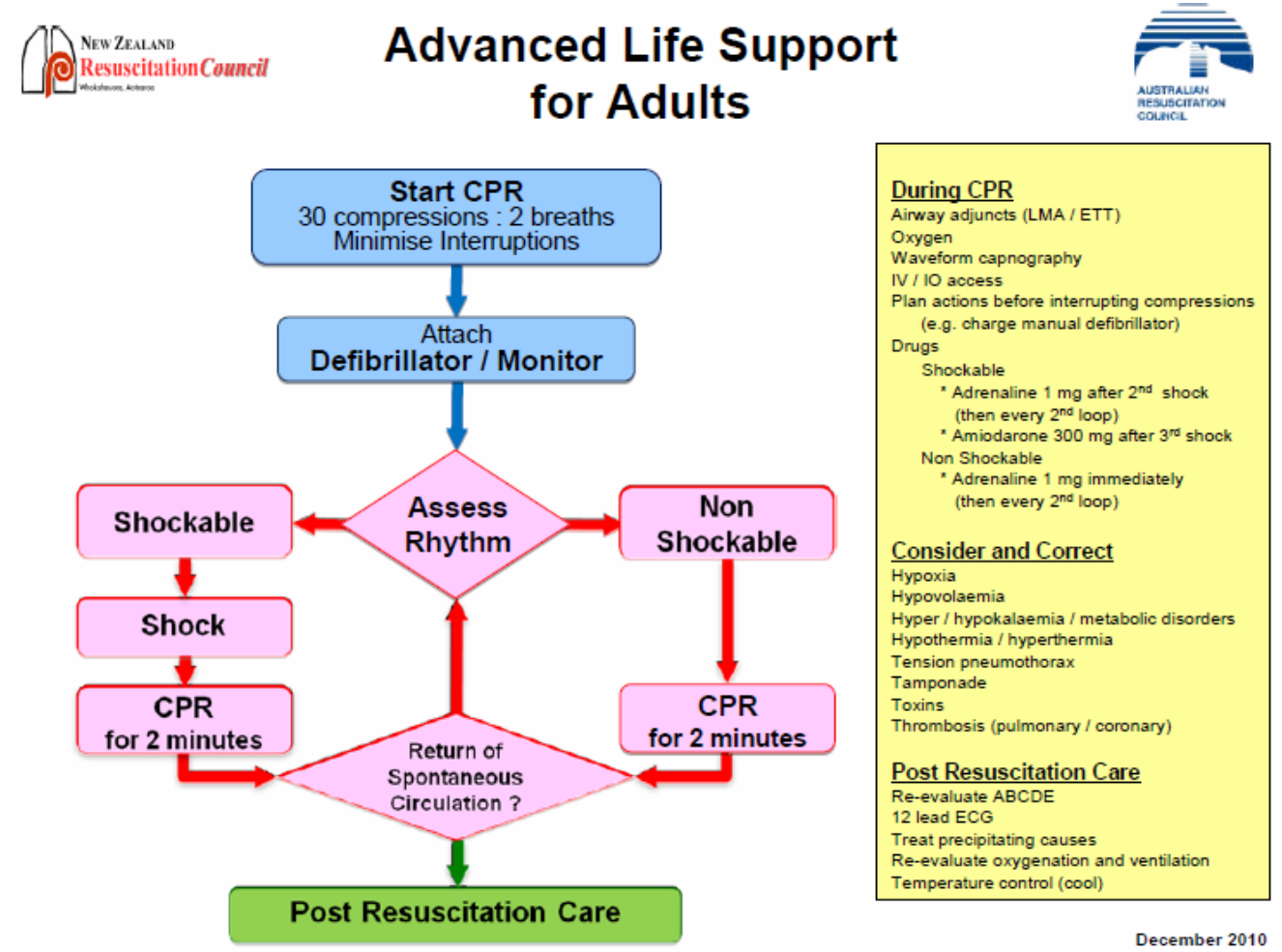
**For non-shockable rhythms**

- Adrenaline 1mg should be administered immediately and then after every second cycle
- Identify potentially reversible causes and conditions that may precipitate cardiac arrest or decrease the chance of successful resuscitation:

***"4** H's and **4** T's "*

| | |
|---|---|
| **H**ypovolaemia | **T**amponade, cardiac |
| **H**ypoxaemia | **T**ension pneumothorax |
| **H**ypothermia/**H**yperthermia | **T**oxins/poisons/drugs |
| **H**yper/hypokalaemia & metabolic disorders | **T**hrombosis, coronary/pulmonary |

**Figure 1 Adult Advanced Life Support Algorithm.**



# DEFIBRILLATION

Defibrillation is the delivery of direct current counter-shock through the heart to simultaneously depolarise the myocardial cells, with the aim of enabling a higher inherent pacemaker to regain organised control of the cardiac rhythm. Defibrillation as soon as possible provides the best chance of survival in victims with VF or pulseless VT (ARC Guideline 11.4, December 2010). It is imperative that the person performing defibrillation is familiar with the operation of the defibrillator being used and is fully aware of the safety aspects required to perform the procedure.

**Biphasic Defibrillation**

Biphasic defibrillation delivers the energy waveform in two phases: the current flows in one direction in the first phase of the shock and then reverses direction for the second phase. All new defibrillators deliver shocks using a variety of biphasic waveforms. Although it has not been demonstrated conclusively in randomised clinical studies that biphasic defibrillators save more lives than monophasic defibrillators, biphasic defibrillators achieve higher first-shock success rates.


A default biphasic energy level of 200J has been recommended as this falls within the range of published energy levels that have demonstrated efficacy for first and subsequent shocks (ILCOR, 2010). However other energy levels may be used providing there is relevant clinical data for a specific defibrillator that suggests that an alternative energy level provides adequate shock success (Successfully achieving an organised rhythm following first shock in over 90% of cases).

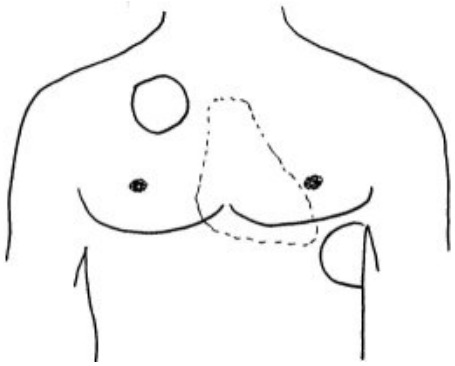
**All Alfred Health defibrillators have data meeting this for 150J shocks.**

**Indications**

- VF and Pulseless VT
- Fine VF masquerading as Asystole

**Procedure for Defibrillation**

- Place the pads on the chest
- One hands free pad is placed on the right subclavicular parasternal region over the 2nd intercostal space, whilst the other is placed on the mid-axilla line at the 6th intercostal space (cardiac apex).
- As it approaches the 2 minute cycle to assess rhythm, CPR must continue until the defibrillator has been charged.



**Placement of pads**

- Ensure proper skin preparation and that the pads are not in contact with anything else on the chest (ECG leads, dressings, internal devices, medication patches) as these can cause decreased conduction and burn the patient
- Dial the energy select button to 150 joules
- Inform all staff that the defibrillator is “charging” (call out loudly), CPR must continue until the defibrillator has reached 150J
- Stop CPR to assess rhythm
    - If non-shockable rhythm – “dump” charge and immediately continue CPR
    - If shockable rhythm -
- Ensure that the patient is not touching any metal.
- Ensure that personnel are not in contact with any fluids on the floor.
- Check that the correct energy setting has been selected
- Look around the bed area to ensure no-one is touching the patient, re-check the rhythm and shout “stand clear shocking”
- Push the shock button.
- Immediately recommence CPR

**Defibrillation Safety**

- The person performing the defibrillation is entirely responsible for the safety of the procedure and of all personnel in the vicinity.
- Be aware of electrical hazards (water, metal fixtures, oxygen and flammable substances).
- Ensure that pads are firmly in place (there must be good contact with the skin).
- If the patient has excessive chest hair, it may be necessary to clip or shave chest hair where the pads need to be located.

**Confirmation of Defibrillation**

- Check that the patient has a motor response to the defibrillation (if not therapeutically paralysed) indicating that a shock has been delivered.
- Evidence of shock delivery will also be seen on the paper print out.

**Failure to Deliver Shock**
If failure to deliver the shock occurs, troubleshoot the following possible causes:

- Defibrillator may be in “synchronised” mode.
- Ensure pads connector is connected to the defibrillator
- Check battery is charged.

## Emergency Synchronised Direct Current Reversion (DCR)

The delivery of the shock is “synchronised” to the R wave on the patient’s ECG delivering the shock in the non-vulnerable period of the myocardial action potential. This reduces the risk of causing R on T phenomenon and inducing VF/VT.

**Indications**
For haemodynamically compromised patients with the following arrhythmias:

- Supra Ventricular Tachycardia (SVT)
- Atrial Tachycardia
- Atrial Fibrillation with a rapid ventricular response (AF)
- Atrial Flutter
- Conscious Ventricular Tachycardia (VT).

**Procedure**
The technique for synchronised DCR is mostly the same as for non-synchronised defibrillation. The differences between the two are:

- Where possible, obtain consent from the patient
- Check the patient is adequately sedated with appropriately trained staff to manage the airway
- Consider the need to administer analgesia concurrently with sedation
- Engage the “SYNC” button
- Check the “SYNC” function has been engaged: marker is highlighted on the R wave, SYNC is displayed on the screen and the light is flashing.
- Select required energy level (25 – 50 Joules for biphasic defibrillator).

**Precordial Thump**
A precordial thump is not part of the ARC Advanced Life Support for Adults flowchart. It may however be considered for patients with monitored, pulseless ventricular tachycardia if a defibrillator is not immediately available. A precordial thump is a single sharp blow delivered to the patient’s chest. A precordial thump should not be used in patients with a recent sternotomy (eg. for coronary artery grafts or valve replacement), or recent chest trauma. It serves to deliver approximately 5 Joules of energy to the myocardium.

*Technique*
The fist is raised 25 – 30 cm above the patient’s chest and is brought down sharply at the level of mid-sternum using the ulna aspect of the fist.

# MEDICATIONS IN ADULT CARDIAC ARREST

No medication has been shown to improve long-term survival in humans after cardiac arrest. Priorities are defibrillation, oxygenation and ventilation together with cardiac compressions

**Intravenous (IV)**

- IV drug administration is preferable
- If there are no visible peripheral veins, the external jugular vein should be considered
- Avoid lower limb veins due to impairment of venous return below the diaphragm during cardiac arrest
- IV drug administration must be followed by a fluid flush of at least 20-30 ml and CPR

**Intraosseous (IO)**

- Is the preferred route if IV access is not available
- If IV access cannot be established, IO drug delivery will achieve adequate plasma concentrations

**Endotracheal**

- If IV/IO access can not be attained and an endotracheal tube (ETT) is present, some medication can be administered via the ETT
- Absorption is variable and plasma concentrations are substantially lower than when the same drug is delivered IV (increase in dose 3-10 times)
- Adrenaline, lignocaine and atropine are the only drugs that can be given via the ETT
- Dilution with water may achieve better drug absorption
- The frequency of administration should be the same as that used for intravenous administration of the above drugs.
- This route can not be used if a laryngeal mask is present.

## ADRENALINE

### Actions

- Naturally occurring catecholamine with alpha and beta effects.
- Administered during cardiac arrest to cause peripheral vasoconstriction via its alpha-adrenergic action, thus directing available cardiac output to the brain and myocardium.
- It may facilitate defibrillation by improving myocardial blood flow during CPR.

### Indications

- Ventricular fibrillation (VF) / Pulseless ventricular tachycardia (VT) after initial counter shocks have failed (after second shock then after every second cycle)
- Asystole and pulseless electrical activity (PEA) immediately (then every second cycle)

### Dosage

- 1 mg (1:10,000 or 1:1000) IV push
- May be given via ETT (3 times the dose diluted with 10-20 ml sterile $H_2O$).
- Higher doses have not been shown to improve long-term outcomes

### Adverse Effects

- Tachyarrhythmia's
- Hypertension post resuscitation
- Tissue necrosis at site of extravasation

## AMIODARONE

### Actions

- First line anti-arrhythmic for VF and pulseless VT
- Class III antiarrhythmic agent, having mainly potassium channel blocking properties. It prolongs the refractory period of atrial, nodal, and ventricular tissue by prolonging the action potential duration.
- It also effects sodium and calcium channels, and has some alpha and beta adrenergic blocking properties.
- Reduces the rate of impulse conduction through the AV node.
- Decreases sinus node automaticity.

### Indications

- VF/pulseless VT (after third shock)
- Consider administration for prophylaxis of recurrent VF/VT

**Dosage and administration**

- Initial dose is 300 mg IV slow push in the arrest situation
- Flush IV access line with 20 mL of 5% glucose prior to and following Amiodarone administration as Amiodarone is incompatible with Normal Saline
- For treatment of uncompromised tachyarrhythmias, amiodarone 300mg should be administered as an infusion over 20 to 30 minutes (diluted in 5% glucose)
- Additional 150mg IV slow push may be considered 3 – 5 minutes after first.
- May require infusion of 10-50mg/kg over 24 hours

**Adverse Effects**

- AV Blocks
- Prolonged QT interval
- Hypotension
- Bradycardia

## LIGNOCAINE

Lignocaine may be used in situations where amiodarone cannot be used or where ventricular ischaemic tissue is the possible cause of arrest.

**Actions**

- Class 1b sodium channel blocker.
- Shortens action potential duration.
- Suppresses automaticity of ventricular ectopic foci.
- Action is restricted to ischaemic ventricular myocardial cells.

**Indications**

- VF / Pulseless VT refractory to defibrillation
- May be used as prophylaxis for recurrent VF or VT

**Dosage and Administration**

- 1 – 1.5 mg/kg IV slow push.
- Additional dose of 0.5 - 0.75mg/kg may be considered.
- May be given via the ETT.
- It is not recommended to commence a lignocaine infusion until return of spontaneous circulation (ROSC)

**Adverse Effects**

- Coma
- Seizure activity
- Decreases effectiveness of defibrillation
- Hypotension

## CALCIUM

### Actions

- Calcium is essential for normal nerve and muscle activity
- Transiently increases myocardial contractility and excitability and systemic vascular resistance.

### Dosage and Administration

- Usual adult bolus dose is 5 to 10 ml of 10% calcium chloride
- Alternately 10ml of 10% calcium gluconate

### Adverse Effects

- Possible increase in myocardial and cerebral tissue injury by mediating cell death
- Tissue necrosis at site with extravasation

### Note

Routine administration of calcium is not recommended. Consider for Hyperkalaemia, Hypocalcaemia, Calcium channel blocker therapy or overdose

## MAGNESIUM

### Actions

- Magnesium is an electrolyte essential for maintaining membrane stability
- Hypomagnesaemia causes myocardial hyperexcitability, especially in the presence of hypokalaemia and digoxin toxicity

### Indications

- Magnesium should be given for hypomagnesaemia and torsades de pointes, but there is insufficient data to recommend for or against its routine use in cardiac arrest.

### Consider administration for:

- Cardiac arrest associated with digoxin toxicity
- VF/pulseless VT (when refractory to defibrillation and adrenaline)
- Documented hypokalaemia

### Dosage and Administration

- 5mmol IV slow push
- May be repeated once.
- Followed by an infusion of 20mmol $MgSO_4$ over 4 hours.

### Adverse Effects

- Rapid administration of Magnesium may cause asystole or significant clinical hypotension.
- Excessive use may cause respiratory failure and respiratory muscle weakness.

## POTASSIUM CHLORIDE

**Actions**

- Potassium is an electrolyte essential for maintaining membrane stability
- Hypokalaemia, especially in the setting of digoxin therapy and hypomagnesaemia, may lead to life threatening ventricular arrhythmias.

**Indications: *Consider for***

- Persistent VF due to documented or suspected hypokalaemia

**Dosage and Administration**

- Bolus of 5mmol IV slow push

**Adverse Effects**

- Excessive use will cause hyperkalaemia with bradycardia, hypotension and possible asystole.
- Extravasation may cause tissue necrosis.

## SODIUM BICARBONATE (NaHCO3)

**Actions**

- Sodium Bicarbonate is an alkalising solution used in situations of severe metabolic acidosis
- In most cardiac arrests, effective CPR and adequate ventilation negates the need for early use of $N_aHCO_3$.

**Indications**

- Treatment of documented metabolic acidosis
- Hyperkalaemia
- Tricyclic overdose
- Prolonged arrest (greater than 15 minutes)

**Dosage and Administration**

- 1 mmol per kg is initially given over 2 to 3 minutes, then as guided by arterial blood gases.

**Adverse Effects**

- Metabolic alkalosis
- Hypokalaemia
- Hypernatraemia
- Hyperosmolality
- Intracellular acidosis
- Sodium bicarbonate and adrenaline or calcium when mixed together may inactivate each other and precipitate and block the line

**Note**

Routine administration for the treatment of cardiac arrest is not recommended
NaHCO3 administration must be accompanied by adequate ventilation and CPR to prevent rebound intracellular acidosis caused by excessive $CO_2$ production.

## ATROPINE

**Actions**
Atropine is an anticholinergic agent. It blocks parasympathetic innervation, allowing the sympathetic nervous system to dominate.

**Indications**
Initial pharmacological treatment for symptomatic patients in:

- Severe bradycardia
- 2nd Degree AV block (type II)
- Complete heart block

**Dosage**

- Atropine is administered IV in doses of 500 to 600 mcg
- This dose can be repeated every 3 to 5 minutes up to a total of 3 mg

**Adverse Effects**

- Tachyarrhythmias
- Pupillary dilatation
- Increased intracranial pressure
- Urinary retention
- Excitement
- Delirium

**Note** There will be no response in heart transplantation patients (Consider Isoprenaline)

## ADENOSINE

### Actions

- Adenosine transiently blocks the conduction of impulses through the AV node interrupting re-entry pathways through the node
- It has an extremely short half-life of 0.6 to 10 seconds
- It should be given quickly through a large bore cannula in the antecubital fossa or central line as a rapid push followed by a rapid flush
- Adverse effects are usually transient and well tolerated because of the short half life

### Indications

- Haemodynamically stable Supraventricular Tachycardia (SVT)
- Paroxysmal SVT

### Contraindications

- Second and third degree AV blocks
- Sick Sinus Syndrome
- Will not revert AF or A/Flutter
- Acute Asthma (can precipitate bronchospasm)
- Long QT syndrome
- Decompensated heart failure

### Dosage and Administration

- Adenosine 6mg rapid IV push followed by 20 ml flush
- If there is no response to 6mg give a 12mg bolus followed by a 20 ml flush (which may be repeated)

### Adverse Effects

- Sinus arrest (2–10 seconds)
- AV Blocks
- Sinus bradycardia
- Hypotension
- Dyspnoea
- Facial flushing
- Feelings of impending doom
- Headache
- Chest pressure/pain

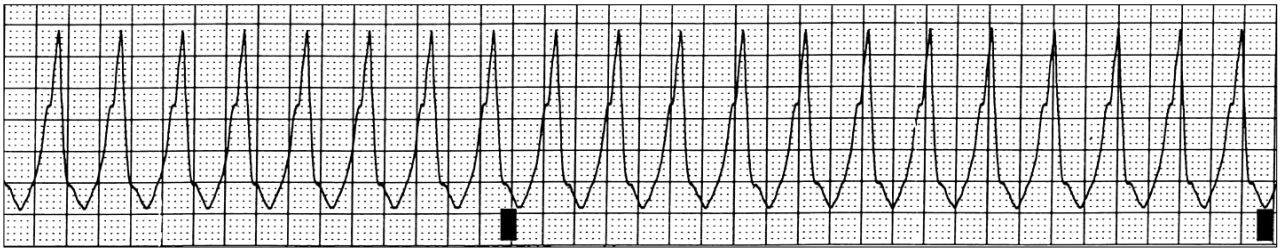
# VENTRICULAR TACHYCARDIA (VT)

**Characteristics**

- Ventricular Tachycardia is a wide complex regular tachycardia
- Rhythm originates in the ventricles
- Usually occurs as a result of an irritable ventricular focus
- Precipitating factors include: AMI, ischaemia, metabolic derangement, acid/base derangement, and cardiomyopathy

**ECG Characteristics**

Rate: 120 – 250 bpm
Rhythm: Regular
P Wave: If seen, dissociation is present
PR Interval: Not applicable
QRS: Wide, greater than 0.14 seconds



A patient who has a rhythm of VT on the monitor can be:

- Conscious and haemodynamically stable (uncompromised)
- Conscious and haemodynamically unstable (compromised)
- Pulseless and unconscious

**Management**

*Conscious stable VT:*

- 12 lead ECG, continuous cardiac monitoring, oxygen and large bore IVC
- 300 mg IV amiodarone over 20 to 60 minutes followed by an infusion of 1g over 24 hours
- Frequent BP and assessment of GCS
- Place hands free defibrillation pads on patient's chest in case of decompensation

*Conscious Unstable VT:*

- Continuous cardiac monitoring, oxygen and large bore IVC (x2)
- Urgent 12 lead ECG
- Airway management
- Emergency synchronised cardioversion with sedation, +/-analgesia

*Pulseless*

- Management as per ARC flowchart shockable rhythm (see below: Management of VF)

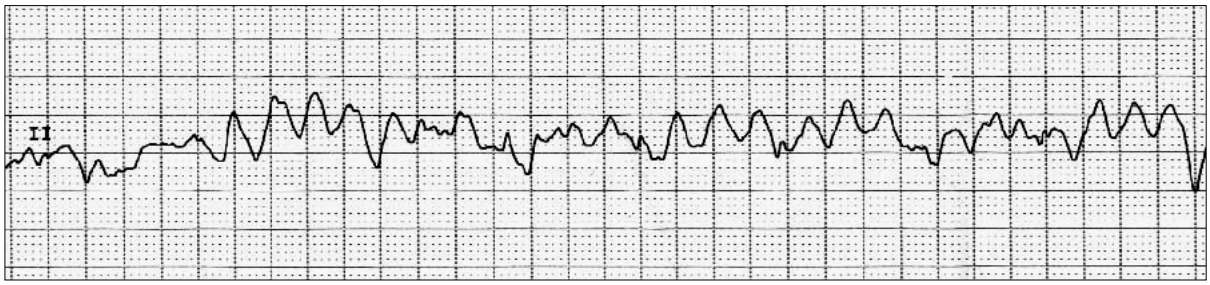
# VENTRICULAR FIBRILLATION (VF)

**Characteristics**

- Ventricular Fibrillation is chaotic ventricular activity that produces no cardiac output
- Irregular and disorganised electrical activity
- Rarely is this rhythm self-limiting
- Fine VF may masquerade as asystole
- Non-synchronous defibrillation has been shown to be the most effective method of terminating this rhythm and should be administered as soon as possible

**ECG Characteristics**

Rhythm: chaotic, irregular
Rate: unable to be measured as there are no P waves or QRS complexes
P wave: absent; wavy irregular deflections; varying size, shape and height; coarse or fine
PR interval: not measurable
QRS complex: absent




**Management of shockable rhythms (VF and VT)**

- Start CPR (30 compressions to 2 breaths)
- Attach defibrillator and confirm rhythm is shockable
- Shock at 150 joules (single shock)
- Immediately recommence CPR and obtain IV access
- Continue CPR for 2 minutes
- Reassess rhythm and administer 2nd shock if still in shockable rhythm
- Recommence CPR and administer Adrenaline 1mg IV
- Administer Adrenaline after every 2nd cycle (every 4 minutes)
- Reassess rhythm and administer 3rd shock if still in shockable rhythm
- Recommence CPR and administer Amiodarone 300 mg IV
- Continue CPR and reassess rhythm every 2 minutes
- Identify and correct potential causes: ***4H's and 4T's•***
- Consider NaHCO3 1mmol/kg IV slowly over 2 to 3 minutes (For prolonged arrests >15mins or when indicated by ABGs).
- Continue resuscitation until a perfusing rhythm is re-established or the decision is made to discontinue resuscitation.

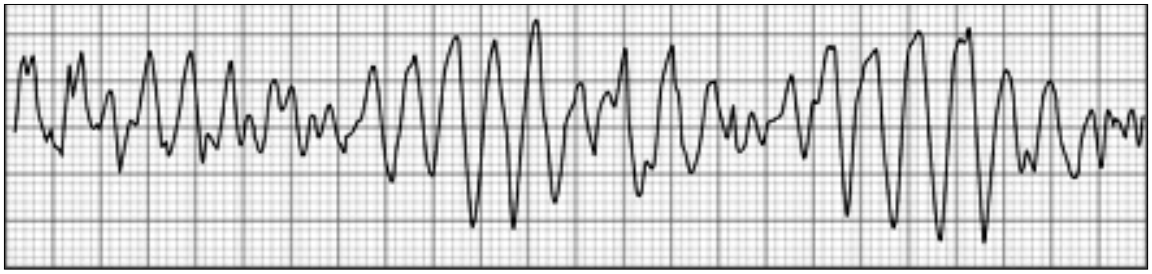
# TORSADE d POINTES

## Characteristics

- Polymorphic Ventricular Tachycardia (VT)
- “Twisting of the Points”
- QRS complexes appear to ‘twist” or undulate around the isoelectric line
- Associated with a prolonged QT interval
- Mechanism is as yet unknown
- May degenerate into Ventricular Fibrillation (VF)

## ECG Characteristics

| | |
|---|---|
| Rate: | 200 – 250 bpm |
| Rhythm: | May be regular or slightly irregular |
| P Wave: | Not observed |
| PR Interval: | Not applicable |
| QRS: | Wide, undulating |
| QT Interval: | Prolonged. Greater than 0.42 – 0.44 seconds |



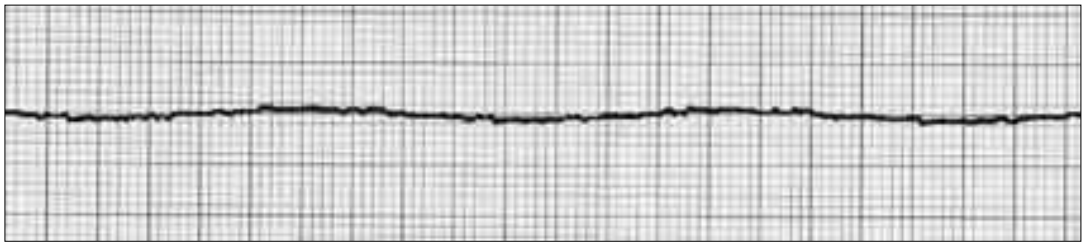
## Management

- Confirm rhythm
- Commence CPR if no output
- Remove any agent known to cause prolonged QT interval (eg Class Ia Antiarrhythmics, sotalol, amiodarone, phenothiazides, some antibiotics (erythromycin), cisapride, tricyclic antidepressants)
- Correct electrolyte abnormalities (especially hypokalaemia)
- Administer a bolus of 5 mmol of magnesium intravenously over 10 minutes
- May be repeated once and followed by an infusion of 20 mmol over four hours
- Torsades may also be precipitated by organophosphate poisoning
- Defibrillation is often transient in reverting Torsades
- Acute cardiac pacing is recommended for patients presenting with torsades de pointes due to heart block and symptomatic bradycardia
- If adverse signs develop: immediate synchronised cardioversion

# ASYSTOLE

**Characteristics**

- The absence of any electrical activity on the monitor
- There is also no mechanical activity (cardiac output) and the patient is unconscious
- There may be an occasional non-perfusing wide complex beat seen on the monitor
- Asystole has a much poorer prognosis than PEA and VF/VT rhythms
- Defibrillation is only indicated if there is reason to believe that the asystole may possibly be fine VF.



**Management**

- Confirm asystole in at least two leads and absence of cardiac output
- Commence CPR
- Increase amplitude (could be fine VF)
- Obtain IV access
- Administer Adrenaline 1mg IV push immediately
- Administer Adrenaline 1 mg after every second cycle (every 4 minutes)
- Defibrillate if there is a possibility the rhythm could be fine VF
- Identify and treat reversible causes: 4 H's and 4 T's

  - o **H**ypovolaemia
  - o **H**ypoxaemia
  - o **H**yper/hypokalaemia & metabolic disorders
  - o **H**ypothermia/Hyperthermia
  - o **T**amponade, cardiac
  - o **T**ension pneumothorax
  - o **T**oxins/poisons/drugs
  - o **T**hrombosis, coronary/ pulmonary

# PULSELESS ELECTRICAL ACTIVITY (PEA)

**Characteristics**

- Pulseless Electrical Activity is the presence of a normally perfusing electrical rhythm but without a detectable cardiac output.
- Caused by a reversible abnormality
- Electrical activity on the monitor is a far more favourable rhythm than asystole.

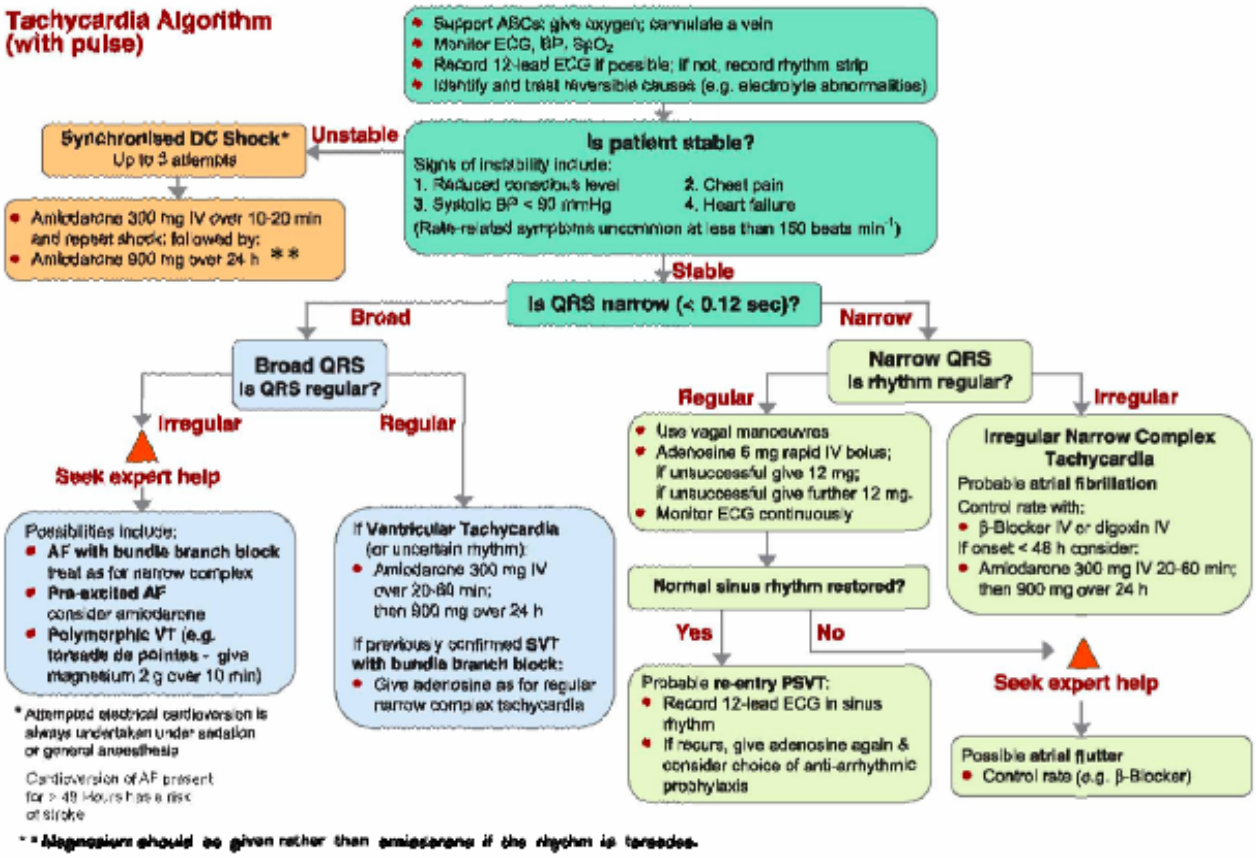
**Management**

- Confirm rhythm and absence of cardiac output
- Commence CPR
- Obtain IV access
- Administer Adrenaline 1mg IV push immediately
- Administer Adrenaline 1 mg after every second cycle (every 4 minutes)
- Identify and treat reversible causes: 4 H's and 4 T's

    - **H**ypovolaemia
    - **H**ypoxaemia
    - **H**yper/hypokalaemia & metabolic disorders
    - **H**ypothermia/Hyperthermia
    - **T**amponade, cardiac
    - **T**ension pneumothorax
    - **T**oxins/poisons/drugs
    - **T**hrombosis, coronary/ pulmonary

# TACHYARRHYTHMIAS

If the patient has developed a narrow complex tachyarrhythmia and has become symptomatic, there is a need for prompt intervention. If the patient is not haemodynamically compromised, pharmacological reversion or rate reduction of their arrhythmia may be appropriate. Emergency Synchronised Cardioversion is necessary for haemodynamically compromised patients. If the patient fails to maintain a cardiac output (no blood pressure or carotid pulse not palpable), then CPR must be instigated immediately.



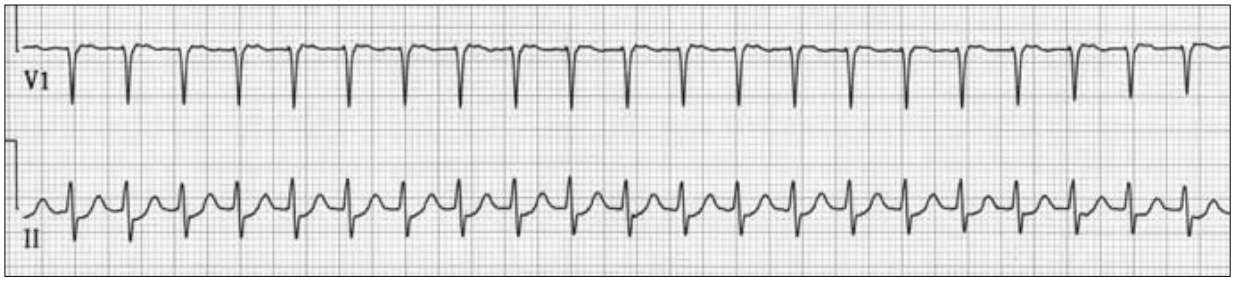
# SUPRAVENTRICULAR TACHYCARDIA (SVT)

**Characteristics**

- Accelerated rhythm originating above the ventricles
- Mechanism is often re-entry in nature
- Occurs due to pre-excitation syndromes (accessory AV pathways), often triggered by stimulants such as alcohol, caffeine and other drugs, or maybe due to ischaemic heart disease.
- Patient often experiences palpitations
- Patient may present haemodynamically stable or symptomatic with hypotension or syncope

**ECG Characteristics**

Rate: Atrial equals the ventricular rate: 140 – 240 bpm
Rhythm: Regular
P Wave: Usually not visible making the exact mechanism of the tachycardia uncertain
PR Interval: Unable to measure as P waves are hidden
QRS: Normal morphology, narrow complex




**Management**

- In the absence of adverse features, start with vagal manoeuvres
- Pharmacological intervention with Adenosine
- Adenosine 6mg rapid IV push followed by 20 ml flush, if there is no response to 6mg give a 12mg bolus followed by a 20 ml flush (which may be repeated)
- If Adenosine is contraindicated or fails to terminate SVT (without demonstrating it is atrial flutter), give a calcium channel blocker (eg Verapamil 2.5 to 5 mg IV over 2 minutes)
- **Emergency synchronised cardioversion if the patient is haemodynamically compromised**

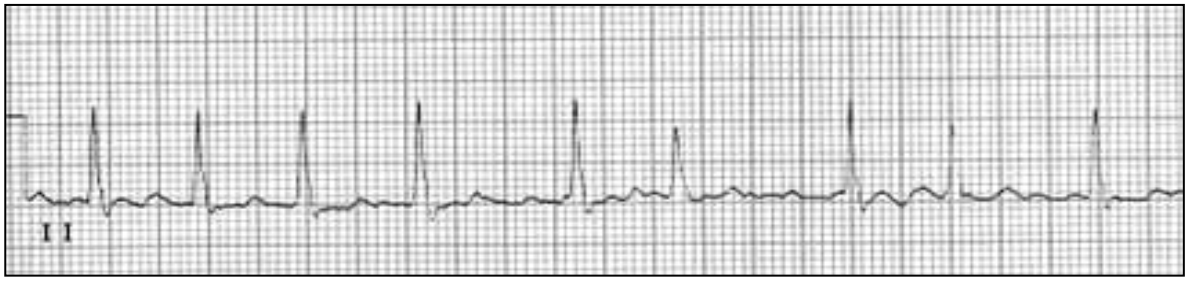
# ATRIAL FIBRILLATION (AF)

**Characteristics**

- Ectopic focus in the atria
- Atria are depolarised at rapid rates > 400 times per minute
- Chaotic, multitude of electrical sites generate impulses – uncoordinated activity causing the heart to quiver rather than contract
- Fibrillatory waves can be coarse (large) or fine (small) waves
- Atrial kick (loss of 30% of volume in the ventricles before systole) is lost due to the quivering
- Non-contracting atria also tend to pool blood within the chambers increasing the potential for thrombus formation
- AV node can not conduct impulses at such a fast rate, therefore only a percentage of the impulses pass through to the ventricles explaining the irregular ventricular rate
- Ventricular rate < 100 bpm - controlled atrial fibrillation
- Ventricular rate >100 bpm - atrial fibrillation with a rapid ventricular response
- In individuals with healthy hearts – usually temporary and may be associated with emotional stress, excessive alcohol or an acute illness (pneumonia, sepsis, PE and thyrotoxicosis).
- Chronic AF is commonly caused by valvular heart disease, hypertension, coronary heart disease and post cardiac surgery

**ECG Characteristics**

Rhythm: Irregular
Rate: Atrial rate > 400 bpm; ventricular rate dependant on the number of impulses conducted through to the ventricles
P waves: Fibrillatory waves replace P waves
PR interval: Nil
QRS complex: Normal




**Management**

- **Haemodynamically compromised patients : Emergency Synchronised Cardioversion is indicated**
- Pharmacological treatment for stable patients: Beta blockers, Digoxin, Amiodarone and Verapamil may be used for rate control. Choice of drug will depend on a number of factors including: persistent or permanent AF and presence or absence of heart failure.

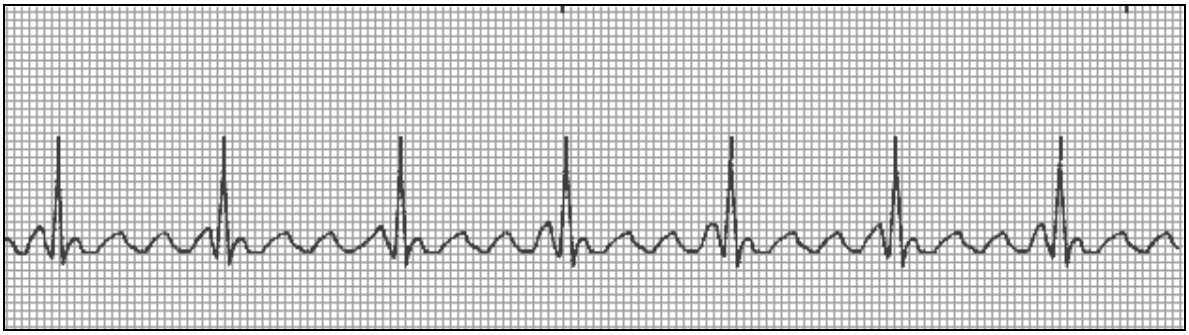
# ATRIAL FLUTTER

## Characteristics

- Atria are depolarised at a rate of approximately 300 times per minute
- Waveforms that resemble saw teeth – flutter waves
- AV node only conducts some of the impulses to protect the ventricles
- If the AV node allows every second impulse through the AV junction to the ventricles, then this is a 2:1 conduction (for every two flutter waves only one is followed by a QRS).
- Atrial flutter can also have a variable conduction block
- 2:1 conduction has a ventricular rate of 150 bpm, 3:1 conduction has a ventricular rate of 100 and 4:1 conduction has a ventricular rate of 75
- Often associated with cardiac disease, cardiomyopathy, ischaemia, congestive cardiac failure

## ECG Characteristics

Rhythm: Usually regular ventricular rate, but a variable AV block will produce an irregular rhythm
Rate: Atrial rate approximately 300 bpm
Ventricular rate may vary depending on the degree of AV block
P Wave: Flutter waves
PR Interval: Not measurable
QRS complex: Normal morphology and duration



## Management

- **Haemodynamically compromised patients : Emergency Synchronised Cardioversion is indicated**
- Pharmacological treatment for the stable patient may include: Amiodarone, Digoxin, Beta Blockers

# BRADYARRHYTHMIAS

The definition of a bradyarrhythmia is a heart rate less then 60 beats per minute; however for some people a heart rate of less than 60 beats per minutes is normal. The most common symptoms of bradycardia include:

- Syncope
- Shortness of breath
- Dizziness
- Chest pain

The following adverse signs suggest a need for immediate treatment:

- Systolic BP less than 90 mmHg
- Heart rate less than 40 beats per minute and symptomatic
- Ventricular arrhythmia
- Heart failure
- Heart rate less than 60 bpm with signs of poor perfusion

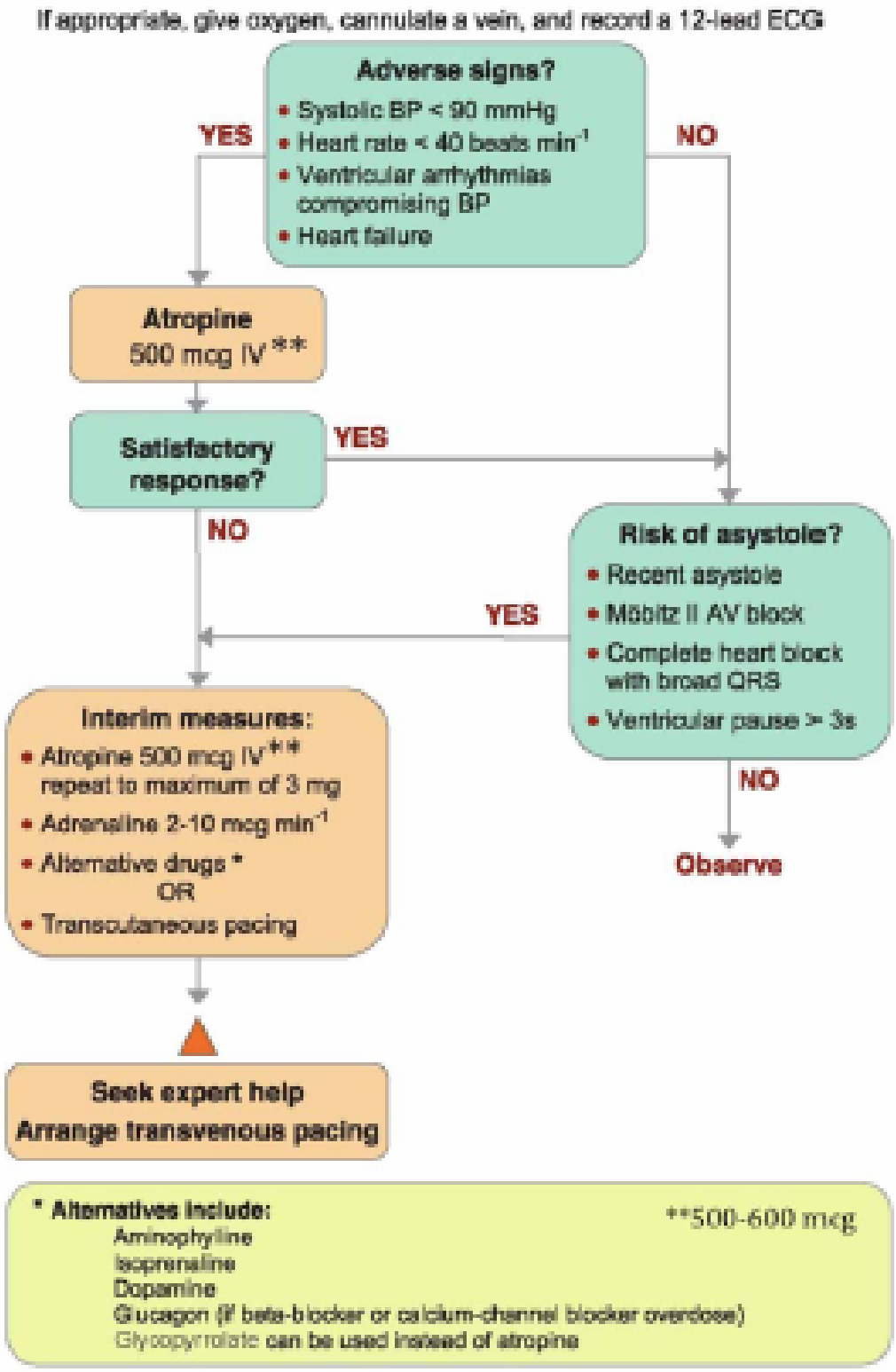
A pulseless patient with a bradyarrhythmia (PEA) requires CPR and management according to the ARC Advanced Life Support for Adults flowchart.

**Rhythms include**

- Sinus Bradycardia
- 1st Degree AV Block
- 2nd Degree Type I and Type II AV Block
- 2:1 AV Block
- 3rd Degree AV Block
- Idioventricular Rhythm

## Bradycardia Algorithm

(includes rates inappropriately slow for haemodynamic state)



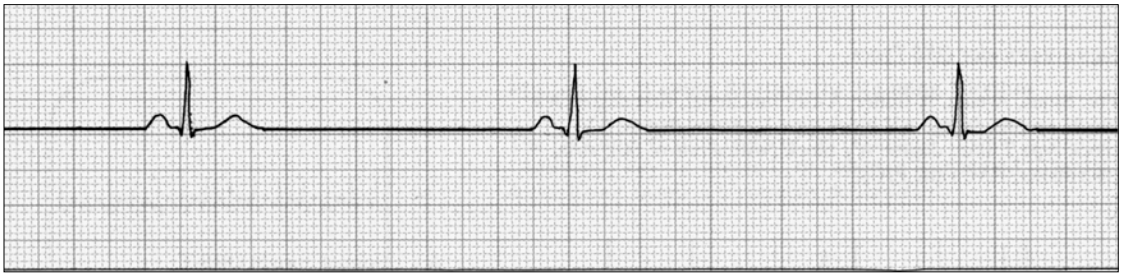
# SINUS BRADYCARDIA

**Characteristics**

- Caused by increased vagal stimulation or decreased sympathetic tone
- Increased ventricular filling time due to prolonged diastole
- Relatively benign rhythm
- Common in very fit athletic people
- Significant bradycardia may cause syncope

**ECG Characteristics**

Rate: Rate is less than 60 bpm
Atrial rate equals the ventricular rate
Rhythm: Regular, unless sinus arrhythmia present
P Wave: P wave present before every QRS complex
PR Interval: PR interval is between 0.12 - 0.20 seconds
QRS: Present after every P wave and of normal interval of 0.08 – 0.12 seconds



**Management**

- Initial pharmacological treatment for symptomatic patients is Atropine
- Atropine is administered IV in doses of 500 to 600 mcg
- This dose can be repeated every 3 to 5 minutes up to a total of 3 mg
- If Atropine is unsuccessful, adrenaline is the second line agent
- Adrenaline is administered at a rate of 2 – 10 mcg/minute to maintain a satisfactory heart rate and blood pressure

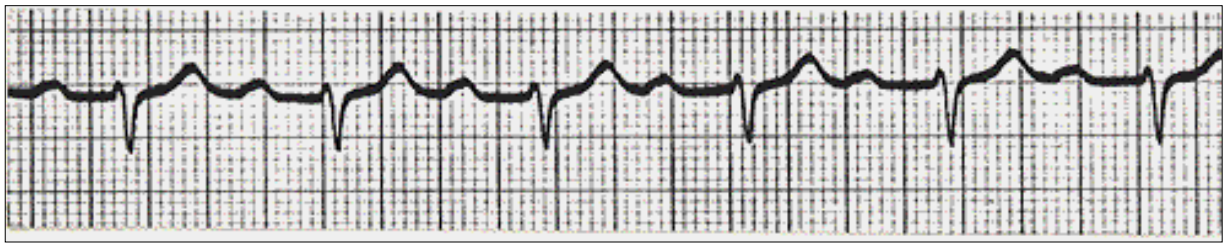
# 1ST DEGREE ATRIOVENTRICULAR (AV) BLOCK

**Characteristics**

- Sinus Rhythm
- Electrical impulses are delayed in the AV node longer than normal (reflected in the longer than normal PR interval)
- All impulses are conducted to the ventricles, but with a delayed conduction time

**ECG Characteristics**

Rate: Usually between 60 – 100 bpm
Rhythm: Usually Regular
P Wave: P wave present before every QRS complex
PR Interval: PR interval is greater than 0.20 seconds
QRS: Present after every P wave and of normal interval of 0.08 – 0.12 seconds



**Management**

- May be a normal finding in some individuals
- May occur in the setting of Inferior MI
- May be associated with Digitalis toxicity
- Usually requires no medical intervention

If the patient is bradycardiac and symptomatic:

- Atropine is administered IV in doses of 500 to 600 mcg
- This dose can be repeated every 3 to 5 minutes up to a total of 3 mg

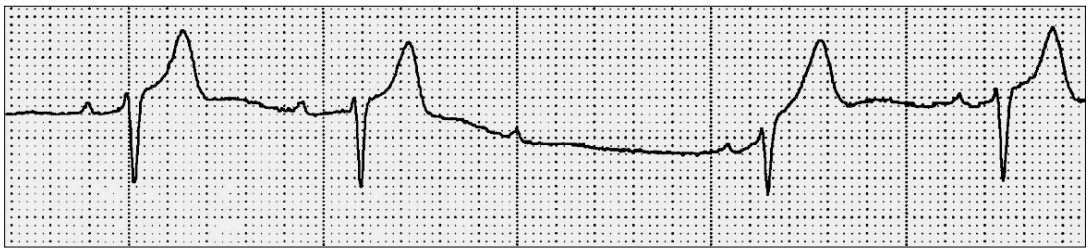
# 2ND DEGREE TYPE I AV BLOCK (WENCKEBACH) (MOBITZ TYPE I)

## Characteristics

- Not all atrial impulses are conducted to the ventricles
- Mechanism involves the progressive lengthening of the PR interval as a result of AV refractoriness until there is eventual failure to conduct an impulse to the ventricles
- After the non-conducted P wave, the sequence begins again
- Often transient and rarely progresses to Type II
- Often associated with Inferior and Right Ventricular MI, Digitalis toxicity, Post-op cardiothoracic surgery
- May occur in very fit, athletic people

## ECG Characteristics

Rate: Atrial rate is greater than the ventricular rate
Rhythm: Regularly Irregular
P Wave: Normal morphology, regular
PR Interval: Progressively lengthens until a QRS is dropped
QRS: Normal Interval; does not follow every P wave



## Management

- Monitor patient's cardiac rhythm and cardiac output
- Usually transient and does not require medical intervention
- Identify and treat any underlying cause(s)

If the patient is bradycardic and symptomatic:

- Initial pharmacological treatment for symptomatic patients is Atropine
- Atropine is administered IV in doses of 500 to 600 mcg
- This dose can be repeated every 3 to 5 minutes up to a total of 3 mg

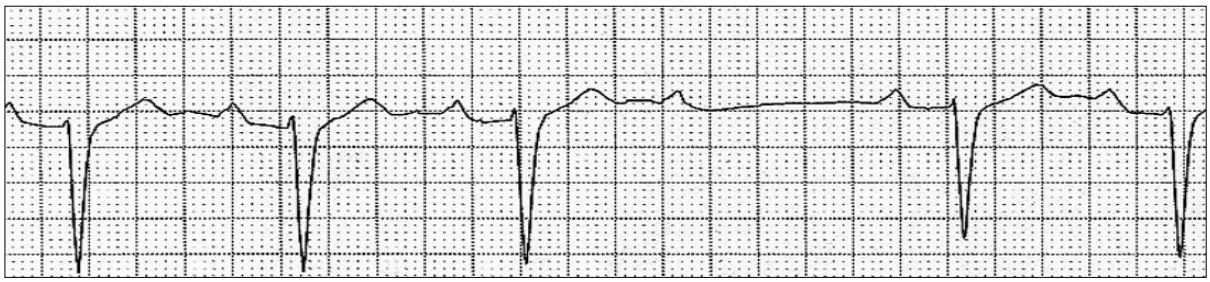
# 2ND DEGREE TYPE II AV BLOCK (MOBITZ II)

**Characteristics**

- Not all atrial impulses are conducted through to the ventricles
- Occurs in the Bundle of His or bundle branches
- Manifests in two ways: sporadic or regular non-conduction of P waves (irregular ventricular rhythm) or every second P wave not conducted to the ventricles (2:1 regular ventricular rhythm – discussed later)
- Less common but more serious than Second Degree Type I
- Can deteriorate rapidly to third degree AV block or ventricular standstill
- Associated with anterior septal MI, conduction system disease, digitalis toxicity

**ECG Characteristics**

| | |
|---|---|
| Rhythm: | Atrial rate regular. Ventricular rate usually regular but may be irregular if AV conduction ratios vary |
| Rate: | Atrial rate greater than the ventricular rate, can occur at a sinus rate Ventricular rate will depend on number of impulses conducted to ventricles |
| P wave: | Present, normal morphology, periodically non-conducted, P – P interval is regular |
| PR interval: | Normal and constant for conducted beats |
| QRS: | May be narrow or wide |



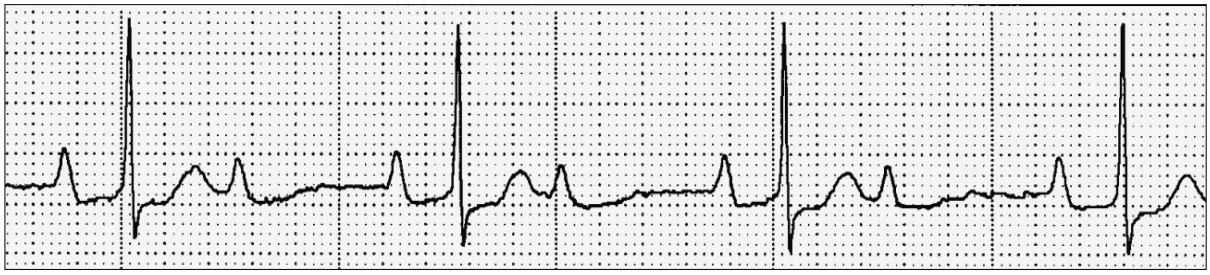
**Management**

- If the patient is asymptomatic: IV access, continuous cardiac monitoring, frequent observations of BP and GCS and have Atropine and pacing accessible
- Initial pharmacological treatment for symptomatic patients is Atropine
- Atropine is administered IV in doses of 500 to 600 mcg
- This dose can be repeated every 3 to 5 minutes up to a total of 3 mg
- If Atropine is unsuccessful, Adrenaline is the second line agent
- Adrenaline is administered at a rate of 2 – 10 mcg/minute to maintain a satisfactory heart rate and blood pressure
- Patients who fail to respond to pharmacotherapy may require electrical pacing

# 2:1 AV BLOCK

**Characteristics**

- Conduction ratio is 2:1 (number of P waves to QRS complexes)
- May involve the AV Node or Sub-Nodal tissue
- May be associated with Inferior MI (AV Node) or Anterior MI (Sub-Nodal)
- Twice as many P waves than QRS complexes
- Unable to determine whether 2$^{nd}$ degree Type I or Type II



**Management**

- Monitor cardiac rhythm and cardiac output

If the patient is symptomatic:

- Initial pharmacological treatment for symptomatic patients is Atropine
- Atropine is administered IV in doses of 500 to 600 mcg
- This dose can be repeated every 3 to 5 minutes up to a total of 3 mg
- If Atropine is unsuccessful, Adrenaline is the second line agent
- Adrenaline is administered at a rate of 2 – 10 mcg/minute to maintain a satisfactory heart rate and blood pressure
- Patients who fail to respond to pharmacotherapy may require cardiac pacing

# 3RD DEGREE AV BLOCK (COMPLETE HEART BLOCK)

**Characteristics**

- No atrial impulses are transmitted to the ventricles
- Atria and ventricles beat independently of each other - AV dissociation
- Absolutely no relationship between the atrial and ventricular activity
- Atria paced by the sinus node at a sinus rate, ventricles are either paced by an escape pacemaker located in the AV junction at a rate of 40 – 60 bpm, or in the ventricles at a rate of 30 – 40 bpm (sometimes less)
- Patients may be asymptomatic or symptomatic if the ventricular rate is too slow severely decreasing cardiac output resulting in poor organ perfusion
- May be temporary or permanent
- May result from inferior MI, ischaemic heart disease, drug effects (digitalis and amiodarone), hyperkalaemia or chronic degenerative changes in the conduction system.

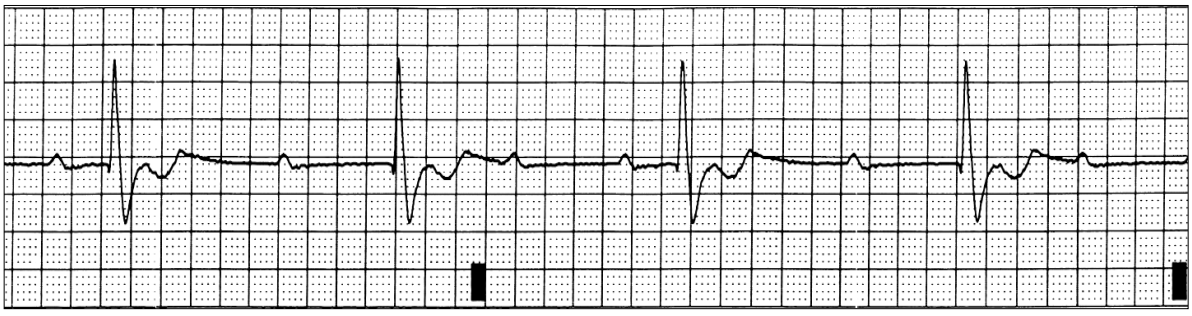
**ECG Characteristics**

Rhythm: Atrial and ventricular rate are regular

Rate: Atrial rate is usually normal
Ventricular rate less than atrial rate: rate of 40 – 60 bpm (if paced by AV junction), rate of 30 – 40 bpm or less (if paced by ventricles)

P wave: Sinus P waves with no constant relationship to the QRS, can be found hidden in the QRS complexes and T wave

PR interval: Variable QRS complex: Normal if block is located at level of AV node or bundle of His, wide if block located at level of bundle branches



**Management**

- Continuous cardiac monitoring and BP and If the patient is symptomatic
- Initial pharmacological treatment for symptomatic patients is Atropine
- Atropine is administered IV in doses of 500 to 600 mcg
- This dose can be repeated every 3 to 5 minutes up to a total of 3 mg
- If Atropine is unsuccessful, Adrenaline is the second line agent
- Adrenaline is administered at a rate of 2 – 10 mcg/minute to maintain a satisfactory heart rate and blood pressure
- Patients who fail to respond to pharmacotherapy may require temporary pacing

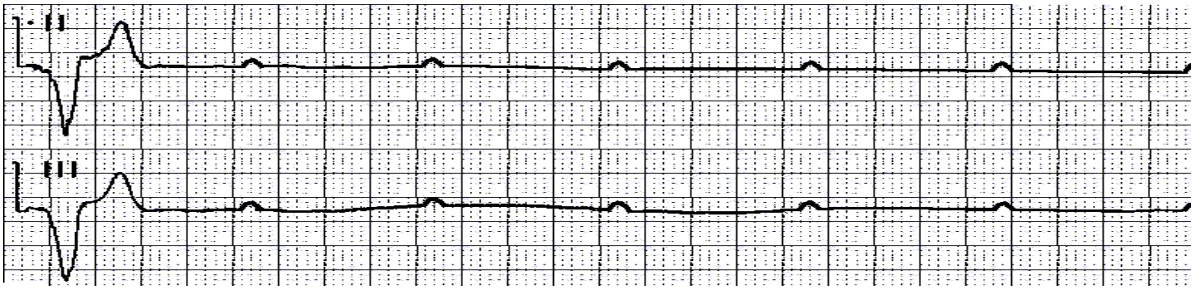
# VENTRICULAR STANDSTILL

## Characteristics

- Absence of electrical activity in the ventricles
- When the escape pacemaker fails in heart block
- There is normal atrial activity with no ventricular activity
- There is no cardiac output, pulse or blood pressure and the patient becomes unconscious immediately

## ECG characteristics

Rhythm: No ventricular rhythm
Regular atrial rhythm
Rate: No ventricular rate
Atrial rate is usually normal
P waves: Regular, sinus P waves
PR interval: Nil
QRS complex: Nil




## Management

- Commence CPR
- Administer Adrenaline 1mg IV push immediately
- Administer Adrenaline 1 mg after every second cycle (every 4 minutes)
- Identify and treat reversible causes: 4 H's and 4 T's
- External pacing

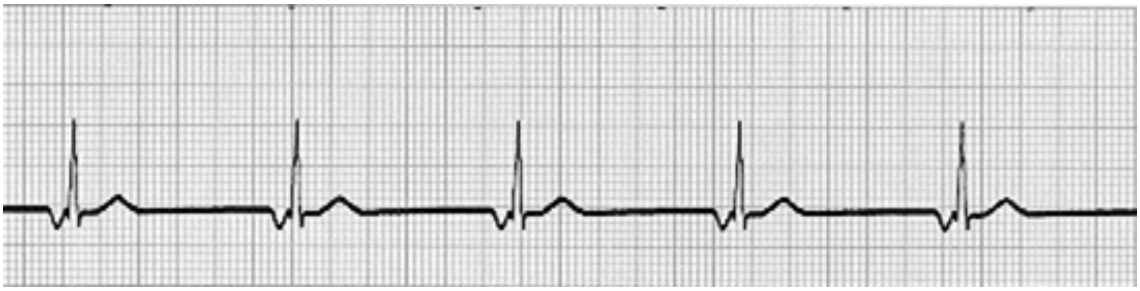
# JUNCTIONAL RHYTHM

**Characteristics**

- Delayed heartbeat originating from an ectopic focus in the AV junction
- Occurs when the rate of depolarisation of the SA node falls below the rate of the AV node
- May also occur due to SA or AV block
- May be secondary to digoxin toxicity, cardiac surgery or an inferior MI
- May be accompanied by symptoms such as palpitations, fatigue, dyspnoea, presyncope or may be entirely asymptomatic

**ECG Characteristics**

Rhythm: Regular
Rate: 40 to 60 bpm. May also present as accelerated > 60 bpm
P wave: Absent or retrograde presentation
PR interval: Not measurable
QRS complex: Normal < 0.12 seconds



**Management**

- Continuous cardiac monitoring and BP if the patient is symptomatic
- Investigation of the cause
- Specific suppressive treatment is rarely required as this rhythm serves as a protective mechanism
- In patients with complete AV block, high-grade AV block, or symptomatic sick sinus syndrome, a permanent pacemaker may be needed

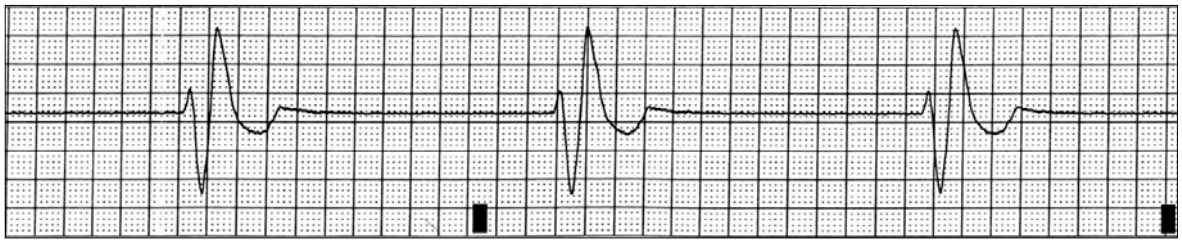
# IDIOVENTRICULAR RHYTHM

**Characteristics**

- Arrhythmia originating in a secondary pacemaker site in the ventricle
- Escape or rescue rhythm, protective mechanism, impulse initiated when higher pacemaker sites have failed
- Lasts for three or more consecutive beats
- Usually transient or intermittent
- Rate may be increased – accelerated idioventricular rhythm
- Often seen post myocardial infarction – thought to be a reperfusion arrhythmia

**ECG Characteristics**

Rhythm: Regular
Rate: 30 to 40 bpm (sometimes less)
P wave: Absent
PR interval: Not measurable
QRS complex: Wide > 0.12 seconds



**Management**

- Monitor rhythm and cardiac output
- Identify cause and treat
- Usually requires no therapy, unless patient becomes haemodynamically compromised

# EMERGENCY TRANSCUTANEOUS PACING

The concept of cardiac pacing is simple: the heart is a muscle that will contract when stimulated by an electrical impulse regardless of the origin of the impulse.

Emergency transcutaneous pacing is indicated for those patients that are haemodynamically unstable due to poor cardiac output secondary to an abnormally slow or absent heartbeat and have not responded to medication therapy. Transcutaneous pacing is most effective in patients who have a primary rate related problem and whose myocardial contractibility is effective and intact, for example, 3rd degree heart block.

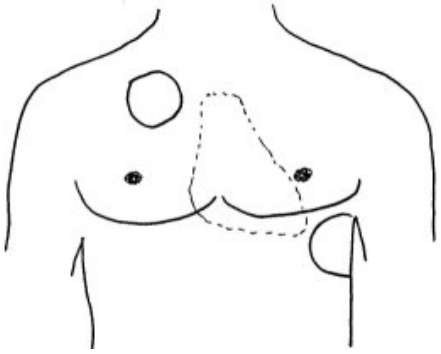
**Transcutaneous (External) Pacing**

Many of the newer defibrillators have built-in external cardiac pacing abilities. It is important to fully understand how to access the functions of and perform the procedure of emergency transcutaneous cardiac pacing. Further details of the machines available at Alfred Health are in Appendix I and II.

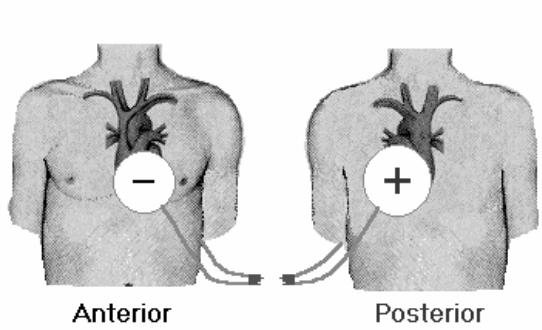
**Procedure for Transcutaneous Pacing**

- Commence basic life support (BLS) to provide oxygenation, ventilation and circulatory support
- Attach the hands free electrode pads to the patient's chest placing them in the anterior – apex position (right of sternum, sub-clavicular and 6th intercostal region over the apex of the heart)
- Alternatively the pads may be placed in the anterior – posterior position (anterior pad placed to left of sternum at 5th intercostal space, with posterior pad placed directly between the inferior border of the scapula and spine)
- Ensure good contact between pads and skin
- Turn dial on defibrillator to pacer
- Set pace maker to desired rate (approximately 80 bpm)
- Start pacing
- Decrease amplitude to 30 mA
- Slowly increase amplitude until capture is achieved (a pacing spike before every QRS complex)
- Monitor haemodynamic responsiveness to pacing (BP, GCS, Pulse)
- Monitor for consistent capture
- Consider sedation and/or analgesia if patient becomes responsive or is awake.

**Anterior pad placement**



**Anterior- posterior pad placement**



# INTUBATION IN THE SETTING OF CARDIAC ARREST

There is insufficient evidence to support or refute the use of any specific technique to maintain an airway and provide ventilation in adults with cardiopulmonary arrest. Either bag valve mask (BVM) alone or in combination with tracheal intubation is acceptable ventilation during CPR. Rescuers must weigh the risks and benefits of intubation versus the need to provide effective chest compressions. The intubation attempt will require interruption of chest compressions, however once an advanced airway is in place, ventilation will not require interruption of chest compressions. An attempt at intubation must not interrupt cardiac compressions for more than 20 seconds. Intubation may be deferred until ROSC.

## OTHER INDICATIONS FOR INTUBATION

- Loss of gag/cough reflex; that is, the ability to protect the airway (for example, head injury with GCS <8) to prevent aspiration
- Airway obstruction: acute laryngeal oedema, for example, inhalation burns, epiglottitis
- Anticipated loss of control of airway, for example, neck trauma, acute stridor
- Worsening respiratory distress

## PREPARATION FOR INTUBATION

- Connect an adult air viva with appropriate size mask to oxygen and place at the head of the bed for pre-oxygenation of patient prior to intubation
- Select appropriate size ETT (male size 8 to 9 mm and female size 7 to 8 mm)
- Open tube and inflate the balloon with a 10 ml syringe to check for patency and no air leak
- Deflate cuff
- If introducer is requested by person performing intubation, insert adult introducer (stylet) into lumen of the ETT (end of stylet should just sit flush with tip of the ETT bevel)
- Apply lubrication to tube
- Check laryngoscope handle is securely attached to the blade and light source is adequate
- Trache tape to secure ETT
- Bougie for difficult intubations
- $ETCO^2$ monitor
- Yankauer sucker attached to suction and turned on and at head of bed
- Set up cannulation pack if patient not already cannulated (preferably 2 large bore IV cannulae)
- 500 ml N/Saline primed for drug and flush line (pump IV line)

- Draw up anaesthetic drugs and label syringes
- Check portable ventilator – connections and settings
- Suggested initial settings – SIMV (+/- Pressure Support), PEEP 5, Frequency (RR) 10 to 12, FiO2 100%, Tidal Volume 5 to 7 mls/kg

## CRICOID PRESSURE

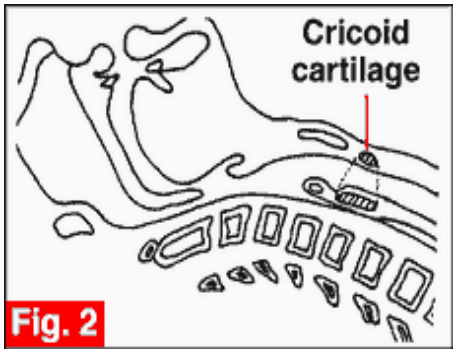
Cricoid pressure compresses the upper oesophagus decreasing the likelihood of passive regurgitation in the unconscious patient.

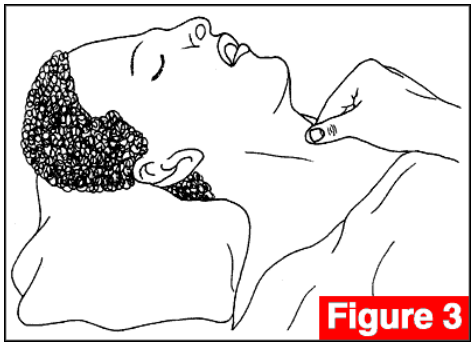
Cricoid pressure should not be used if there is swelling of the front of the neck from recent trauma or if the anatomy is difficult to define. It should not be performed at any time, if there are active vomiting attempts by the victim.

Particularly in the setting of trauma, remember to protect the cervical spine. If feasible, the patient would be in a position that allowed intubation. This may require support under the back of the head.

**Note:** The **routine use** of cricoid pressure to prevent aspiration in cardiac arrest is not recommended. If cricoid pressure is used during cardiac arrest, the pressure should be adjusted, relaxed, or released if it impedes ventilation or placement of an advanced airway.

Identify the cricoid and thyroid cartilages (fig 2).




Upon advice from the person responsible for the airway, place the thumb and middle finger on either side of the cricoid cartilage, with the fingers together and the index finger above, in the space between the cricoid and thyroid cartilages (figure 3). Apply pressure directly backwards and maintain until advised to release.

# MEDICATIONS FOR INTUBATION

Rapid Sequence Induction (RSI) is otherwise known as the technique for “crash intubation”. It is usually performed in an emergency situation and the drugs used include induction agents (sedatives) and paralysing agents. Note: RSI is not used in the cardiac arrest situation.

**Induction Agents (Sedatives)**

**Sodium Thiopentone** is a rapidly acting barbiturate

- An intravenous injection of sodium thiopentone causes loss of consciousness within 15 to 30 seconds and lasts for 5 to 10 minutes
- Sodium Thiopentone (500 mgs) is prepared by dissolving powder in sterile water and diluted up to 20 ml (25 mg/ml)

Recommended dosage is approximately 3 to 7 mg/kg for adults and children.

Sodium Thiopentone:

- Decreases contractility
- Increases heart rate, coronary blood flow and oxygen demand of the heart
- Decreases venous tone causing pooling of blood in the peripheral veins (can cause hypotension in hypovolemic patients)

**Propofol** is a short acting anaesthetic agent suitable for induction and maintenance of anaesthesia in adults.

- Rapid onset of action, approximately 30 seconds
- Propofol can cause hypotension and decreased heart rate

Recommended dosage is 1.0 to 2.5mg/kg for induction by slow intravenous injection. Repeated boluses of 25 mg to 50 mg may be given if required. For maintenance, a continuous infusion of 4 to 12 mg/kg/hr should be commenced.

**Fentanyl** is an opioid analgesic.

- Peak analgesic effect occurs within several minutes and duration of action is 30 to 60 minutes

Recommended dosage for induction of anaesthesia is 50 to 100 mcg intravenously and repeated at 2 to 3 minutely intervals.

Fentanyl can cause bradycardia and muscle rigidity.

**Midazolam** is a short acting sedative used for induction of anaesthesia and for conscious sedation. Midazolam has amnestic properties.

Contraindications: myesthesia gravis and hypersensitivity to benzodiazepines.

Recommended dosage is 3 to 10 mg by slow IV injection.

**Ketamine** is a rapid acting general anaesthetic agent used prior to administration of other general anaesthetic agents

- Onset of action is rapid; a 2mg/kg intravenous injection produces anaesthesia within 30 seconds and lasts for 5 to 10 minutes
- Elevation of BP begins shortly after injection, reaches a maximum within a few minutes and returns to pre-anaesthetic values within 15 minutes.
- Consider use in haemodynamically unstable patients and asthmatics

Contraindications: any condition in which a significant elevation in BP would be hazardous.

**Paralysing Agents**

**Suxamethonium** is a short acting depolarising neuromuscular blocking agent. Onset of action is 30 to 60 seconds post IV injection and duration of action is approximately 3 to 7 minutes

Recommended dosage is 1 to 2 mg/kg.

Contraindications: recent burns, increased potassium levels, severe muscle trauma or allergy.

Suxamethonium:

- Can cause bradycardia especially with second dose (more common in children), which may be prevented by prior administration of atropine
- Increased serum potassium levels by 1mmol/L with greater increases in patients with recent burns, paraplegia and severe muscle trauma
- Increased intracranial and intraocular pressure

**Vecuronium** is an intermediate acting non-neuromuscular blocking agent.

- Vecuronium is prepared by dissolving the powder in water immediately prior to use.

Recommended dosage: 0.08 to 0.1 mg/kg (2 to 8 mg prn) and will produce relaxation for 15 to 60 minutes.

**Rocuronium**: is an intermediate acting non-depolarising neuromuscular blocking agent

- Rocuronium has a relatively fast onset of action, but variable often within 60 seconds post-intravenous injection of 0.6mg/kg and has duration of 30 to 40 minutes.

Maintenance dosage: 0.15mg/kg bodyweight.

# ASSISTING WITH INTUBATION

Following pre-oxygenation, sedation and paralysis:

- Head positioning – neck slightly flexed and head extended with pillow under the head and neck, not under the shoulder (if spinal injury is suspected, spinal immobilisation must be maintained throughout procedure)
- Apply cricoid pressure (as previously discussed)
- Assist with insertion of ETT by passing the laryngoscope, then the Yankauer sucker to clear airway prior to insertion of tube, then the ETT
- Once the ETT is inserted, inflate cuff with 10 ml syringe
- Connect $CO^2$ monitor and air viva to ETT
- Check capnograph waveform on monitor
- Auscultate lungs for equal air entry
- Auscultate neck to listen for air leak around cuff
- Secure ETT with trache tape
- Attach ventilator to ETT
- Document size of ETT, length of ETT to lips, ventilator settings and baseline observations
- Ensure post ETT insertion CXR is attended and if ETT is advanced or pulled back that the new position of tube is documented.

# POST-RESUSCITATION CARE

After the return of spontaneous circulation, the resuscitation of the patient does not stop. It is essential that cardiac monitoring and constant maintenance of airway, breathing, and circulation continues. Interventions in the post-resuscitation period are likely to significantly influence the final outcome. Hypoxic brain injury, myocardial injury or subsequent organ failure are the predominant causes of morbidity and mortality after cardiac arrest.

The aims of therapy after initial resuscitation are to:

- Continue respiratory support
- Maintain cerebral perfusion
- Treat and prevent cardiac arrhythmias
- Determine and treat the cause of the arrest

**Treatment Recommendations**

**Blood pressure**

- Aim for a BP equal to the patient's usual BP or at least a systolic pressure greater than 100mmHg

**Oxygenation**

- Once ROSC has been established, oxygen should be titrated to achieve a SaO2 of 94 – 98% (confirmed by arterial blood gas analysis).

**Control of arterial carbon dioxide**

- Maintain normocarbia
- Hyperventilation may be detrimental and should be avoided
- Arterial blood gas measurements should be used to titrate ventilation in the immediate post-resuscitation period, rather than end tidal $CO^2$ levels.

**Blood glucose control**

- Several human studies have documented a strong association between high blood glucose after resuscitation from cardiac arrest and poor neurologic outcome
- Monitor blood glucose frequently and treat hyperglycaemia (>10mmol/L) with insulin but avoid hypoglycaemia

**Prophylactic antiarrhythmics**

- May be reasonable to continue an infusion of an antiarrhythmic that successfully restored a stable rhythm during resuscitation
- If no antiarrhythmic drug was used during resuscitation from a shockable rhythm, an antiarrhythmic drug may be considered to prevent recurrent VF

**Temperature control and Induced Hypothermia**

- Induced hypothermia has been shown to be beneficial in some patients still comatose after ROSC

- Comatose adult patients with return of spontaneous circulation after an out-of-hospital VF arrest should be cooled to 32 to 34 ºC for 12 to 24 hours.
- Induced hypothermia may also benefit comatose adult patients with return of spontaneous circulation after an out-of-hospital cardiac arrest from a non-shockable rhythm or cardiac arrest in hospital
- Rapid infusion of ice-cold intravenous fluid 30ml/kg is a safe, feasible and simple method of initially lowering core temperature by up to 1.5ºC, as are application of ice packs
- Hyperthermia should be avoided

**Monitoring and Investigations**

Central Nervous System

- Ensure adequate oxygenation (titrate FiO2 according to SaO2)
- Maintain perfusion pressure (MAP)
- GCS

Cardiovascular System

- Avoid persistent hypotension and optimise organ perfusion with inotropic support
- Blood for biochemical analysis: U&E, FBE, Troponin I and CK, Magnesium, LFT's, Calcium
- Perform 12-lead ECG
- Continuous cardiac monitoring
- Consider invasive monitoring (Arterial line and CVC line)
- Identify complications of CPR (tension pneumothorax and tamponade)

Respiratory System

- Chest XRAY (check ETT and line position, assess for complications of CPR - pneumothorax, fractured ribs and aspiration)
- Assess ABG's and titrate FiO2 accordingly
- Mechanical Ventilation, $ETCO^2$ and $PaCO^2$ monitoring

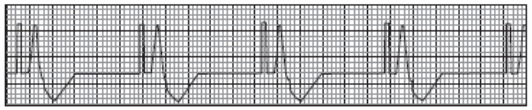
Renal System

- Consider indwelling catheter for hourly urine measures
- Maintain accurate fluid balance
- Check Urea &. Creatinine

Other considerations

- Notify and inform the patient's Parent Unit and the patient's relatives of what has occurred
- Inform and reassure the patient
- Organise transfer and paperwork
- Consider and organise a debriefing for staff involved in the resuscitation and care of the patien

# APPENDIX I PHILLIPS HEARTSTREAM TRANSCUTANEOUS PACING



## Transcutaneous Pacing

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PHILIPS

Printed in the Netherlands
4522 977 00341/861 * DEC 2004


## Performing Transcutaneous Pacing

**Prepare the patient**
Make sure the patient understands the procedure. Sedation may be necessary to improve tolerance of transcutaneous pacing.

**Clean and dry skin**
If hair prevents good pad contact with the skin, shave the area before applying pads. Be sure to avoid breaking the skin. Follow the manufacturer's directions on the pad pouch.

**Apply pads**
Apply pads to the patient, preferably using anterior-posterior placement. Do not reverse the pads. Connect the pads to the defibrillator/monitor.

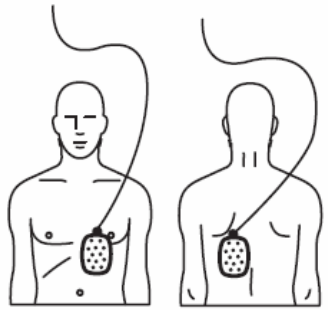

**Turn the pacer functionality on**
Activate pacing functionality. Each QRS complex should have a dot marker associated with it. If not, adjust the ECG size or select another lead. Leads aVL or $V_1$ are recommended with anterior-apex pad placement.


**Select mode (demand or fixed)**
Demand pacing is recommended. The ECG monitor must use a minimum of a 3-wire system for demand pacing. Adjust the rate to the desired number of paced pulses per minute (ppm).

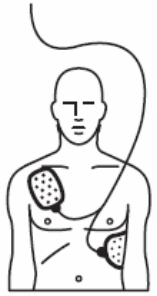

**Capture the heart rate**
Start the pacer. Increase the output (mA) until pacer spikes are visible in front of each QRS complex and capture has occurred. Then, decrease the output to the lowest level that still maintains capture.

**Evaluate the patient**
Did the patient improve with capture? Evaluate BP, $Sp0_2$, and pulse rate. If hemodynamic monitoring is available, evaluate stroke volume and $SvO_2$.
NOTE: Capture alone does *not* guarantee the cardiac output has improved.

## Anterior-Posterior Placement for Pacing (Standard)



## Anterior-Apex Placement for Pacing (Optional)



## Pacing Modes

**Demand (synchronous) mode** is the preferred means of pacing as it paces only when the patient's heart rate falls below a level set by the clinician. This mode avoids problems such as a pacer impulse landing on a T-wave and possibly causing a ventricular dysrhythmia.

**Fixed (asynchronous) mode** paces at the rate set by the clinician regardless of the patient's heart rate. This mode is not the preferred means of pacing and is usually reserved for when (1) the pacer cannot sense the heart rate or (2) when motion artifact prevents the pacer from sensing the heart rate.

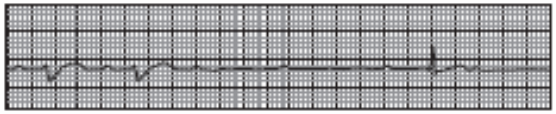
# Transcutaneous Pacing

## About Transcutaneous Pacing

Transcutaneous pacing is the technique of electrically stimulating the heart externally by using a set of electrode pads.

This technique is not as efficient as transvenous pacing because the electrical stimulus (the pads) is not in direct contact with the heart muscle. Transcutaneous pacing is a good temporary solution in an emergency situation to improve a slow heart rate resulting in inadequate cardiac output.

## Emergency Indications

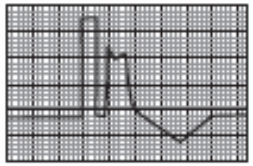


1. Slow heart rates (bradycardias) that produce unstable hemodynamics (e.g., low blood pressure, stroke volume, $SvO_2$)
2. Slow heart rates (particularly escape rhythms) that do not respond to drug therapy
3. Any condition as a temporary measure in preparation for a transvenous pacemaker
4. Non-emergent indications
   a. Overdrive pacing (when drugs and electrical cardioversion have failed)
   b. Heart blocks in the presence of myocardial infarction

## Key Points

### Obtaining capture

Increase the output (mA) until the pacer spike is seen in front of the QRS complex. The amount of mA used varies per patient. Increasing the mA slightly above where capture is obtained may help prevent the loss of capture.



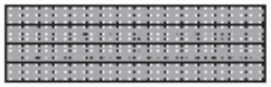
The QRS from the pacemaker will appear wide (like a PVC or ectopic beat), which is normal for a transcutaneous pacemaker.

The muscle under the pads will contract as the pacemaker discharges. However, this muscle contraction does not mean the pacemaker is producing good cardiac output.

Effective capture of the cardiac muscle is seen by improving hemodynamics.

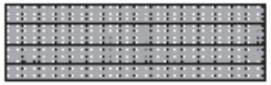
### Troubleshooting

If the pacemaker spike is not in front of each QRS complex, then one of two problems may exist.



**1) Failure to capture**

Failure to obtain capture occurs in demand and fixed mode. Increasing the output (mA) may obtain capture. Be sure the pads have good skin contact. Check for correct pad placement.



**2) Failure to sense**

This problem occurs in demand mode only and is seen when the pacemaker discharges immediately after the patient's own QRS complex (the discharge occurs in the refractory period of the heart). In this case, the pads are not sensing the patient's heartbeat. Select a different monitoring lead or reposition the pads. Fixed pacing may be indicated.

## Patient Preparation

| | |
|---|---|
| **Psychological** | • Educate patient about possible discomfort with pacing.<br>• Instruct patient and family that muscles will twitch with each pacemaker beat. |
| **Sedation** | • If sedation is needed (a normal occurrence), be prepared to initiate when pacemaker is activated.<br>• Discomfort may not be noticed until higher mAs are used.<br>• Sedation should be for a targeted level (e.g., Ramsey of 2-4). |
| **Skin** | • Prepare skin for pad placement (cleaning and shaving, if necessary). Be sure to avoid breaking the skin while shaving.<br>• Check skin routinely to avoid severe skin irritation.<br>• If patient's condition allows, move pad placement as necessary to protect skin. |

## Patient Care

| Patient Oriented | Pacemaker Oriented |
|---|---|
| Evaluate pads for comfort. Pads should be checked often. Normally they are checked every 30 minutes (to avoid severe skin irritation). They should not be left in the same place for more than a few hours.<br><br>Ensure there is a pulse with each QRS complex. Otherwise, electromechanical dissociation may exist. | Routinely ensure that the pacemaker is capturing the heart rate with each discharge (check for a pulse with each pacer spike).<br><br>Identify the length of time the pacemaker is to be used. Keep in mind that transcutaneous pacing is only temporary, usually less than a few hours. |

# APPENDIX II PHILLIPS HEARTSTART MRX TRANSCUTANEOUS PACING

## PHILIPS NONINVASIVE PACING
### (TRANSCUTANEOUS PACING)

**Demand Mode Pacing** (*Requires ECG leads and pads*)

1. Turn the Therapy Knob to **Pacer**.
2. Press the Lead Select button to select the desired lead.
3. Verify white R-wave markers appear above or on the ECG waveform.
4. Press the ***Pacer Rate*** soft key, select the desired number of paced pulses per minute, and press the Menu Select button.
5. If needed, press the ***Pacer Output*** soft key, use the Navigation buttons to adjust the initial pacer output, and press the Menu Select button.
6. Press the ***Start Pacing*** soft key.
7. Verify white pacing markers appear in front of every paced QRS.
8. If cardiac capture is not obtained, press the ***Pacer Output*** soft key, increase the output until capture occurs, decrease the output to the lowest level that still maintains capture, and press the Menu Select button.
9. Verify the presence of a peripheral pulse.

Demand Mode Capture

**Fixed Mode Pacing** (*Can be done with pads only. If you want to see ECG waveforms you must also have electrodes on the patient.)*

1. Turn the Therapy Knob to **Pacer**.
2. Set the pacer mode to Fixed.
   - Select **Pacer Mode** from the Main Menu.
   - Select **Fixed** and press the Menu Select button.
3. If using leads, press the Lead Select button to select the desired lead.
4. Press the ***Pacer Rate*** soft key, select the desired number of paced pulses per minute, and press the Menu Select button.
5. If needed, press the ***Pacer Output*** soft key, use the Navigation buttons to adjust the initial pacer output, and press the Menu Select button.
6. Press the ***Start Pacing*** soft key.
7. Verify cardiac capture. If it is not obtained, press the ***Pacer Output*** soft key, increase the output until capture occurs, decrease the output to the lowest level that still maintains capture, and press the Menu Select button.
8. Verify the presence of a peripheral pulse.

***NOTE: In Fixed and Demand modes, if monitoring $SpO_2$ when pacing, activate the Pulse alarms to assess for peripheral perfusion through the $SpO_2$ transducer.***

453564042671
Edition 3
July 2009

HEARTSTART MRx
MONITOR/DEFIBRILLATOR

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