Management of Arrhythmias in the Intensive Care Unit

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Introduction

Cardiac arrhythmias in critically ill patients may result in significant haemodynamic compromise with potentially life threatening consequences. Increased hospital and one year mortality among surgical ICU patients who develop arrhythmias after non-cardiac and non-thoracic surgery has been clearly demonstrated.[1] Prevention of contributing factors and prompt recognition and treatment of the arrhythmia reduces morbidity, mortality and length of hospital stay.

Successful management should include correction of any reversible factors, in addition to the specific medical treatment aimed at the arrhythmia itself. Factors predisposing to the development of arrhythmias in the critically ill patient include:

- Sepsis and Systemic Inflammatory Response Syndrome (SIRS)
- Electrolyte abnormality (particularly potassium and magnesium)
- Hypoxaemia and hypercarbia
- Hypovolaemia
- Hyperthermia and hypothermia
- Drugs (e.g. inotropes)
- Vascular cannulation (pulmonary artery catheterisation and central line insertion)
- Structural or ischaemic heart disease

It is important to assess the patient (and not just the ECG), as the physiological insult and the urgency with which an arrhythmia should be treated will depend on the patient's cardiac physiology and function, as well as the ventricular response rate and duration.[2]

The first step in the evaluation of an arrhythmia is to assess the haemodynamic status of the patient. This includes assessment of pulse, blood pressure, presence of myocardial ischaemia and/or congestive cardiac failure (chest pain, breathlessness) and cerebral perfusion (confusion or reduced conscious level). In the presence of significant haemodynamic instability associated with a tachyarrhythmia (but not sinus tachycardia), prompt cardioversion is indicated and the Resuscitation Council Advanced Life Support guidelines should be followed. Stable patients with an arrhythmia should have a 12-lead ECG performed to diagnose the specific rhythm, which will guide selection of the most appropriate treatment.

The aim of arrhythmia treatment can be considered to be three-fold:

1) Ventricular rate control
2) Restoration of sinus rhythm
3) To minimise complications if sinus rhythm cannot be restored.

After managing any reversible causes, treatment options include synchronised DC cardioversion, anti-arrhythmic drug therapy and pacing. It should be noted that all anti-arrhythmic drugs are all potentially arrhythmogenic.

Arrhythmias can be broadly classified into *bradycardias* and *tachycardias*, which can be further divided into *broad complex* and *narrow complex*.

Bradycardia

Bradycardia is defined as a heart rate of less then 60 beats per minute (bpm), however in practice patients should be considered bradycardic if they have a pulse that is unusually slow for their haemodynamic state. In general, pulse rates above 50 bpm are usually well tolerated and in young, fit patients 40-50 bpm is often acceptable.

Figure 1: *Causes of bradycardia*

- Sinus bradycardia
- Sinus node dysfunction
- Heart block (Age-related fibrosis of the conducting system)
- Drugs – AV blocking drugs, metaraminol
- Hypothermia
- Hypothyroidism
- Head injury

Management of bradycardia

- Assess patient for adverse signs including heart rate less than 40 bpm, chest pain, heart failure or hypotension (systolic BP (SBP) <90mmHg).
    - If no adverse signs, the patient may be appropriate for observation and monitoring (in at least a level one setting). Consider use of glycopyrrolate 200 micrograms up to a maximum of 600 micrograms.
    - If adverse signs present or other concerns administer intravenous atropine 500 micrograms up to a maximum of 3mg.
- Consider the need for temporary pacing, which may initially be in the form of external pacing pads with mild sedation, however this should be succeeded by an intravenous temporary pacing wire at the earliest opportunity.
- Consider whether the patient has an indication for elective pacing to avert the risk of asystole.

Figure 2: *Indications for temporary pacing*

- Asystole
- Complete heart block
- Symptomatic bifasicular block (right bundle branch block and left axis deviation)
- Symptomatic trifasicular block (bifasicular block plus prolonged PR interval)
- Mobitz type II second degree heart block
- Symptomatic bradycardia refractive to pharmaceutical therapy

Consider pacing if the following are unresponsive to atropine:

- Mobitz type I second-degree heart block with hypotension
- Recurrent sinus pauses
- Sinus bradycardia

NB: Overdrive pacing can also be considered for refractory tachycardias

Tachycardias

Broad Complex Tachycardia

A broad complex tachycardia is defined as greater than 100 bpm with a QRS width of greater than 0.12 seconds (3 small squares on the ECG). Causes of a broad complex tachycardia include ventricular tachycardia (VT), torsade de pointes and ventricular fibrillation (VF). As VF is almost without exception pulseless, it should be treated according to ALS guidelines and is therefore not considered here. The management of broad complex tachycardias is summarised below:

Figure 3: *Management of broad complex tachycardia*

- Check airway and breathing. Administer high-flow oxygen.
- If in doubt assume all regular broad complex tachycardias are ventricular in origin.
- Assess for signs of haemodynamic collapse: SBP<90mmHg, chest pain, breathlessness or reduced conscious level. If present, proceed immediately to DC cardioversion (consider sedation).
- Correct electrolyte abnormalities and treat other reversible causes
- Management of torsades de pointes should include magnesium and pacing rather than pharmacological therapy.
- The preferred anti-arrhythmic for VT is amiodarone, 150mg over 10 minutes.

Monomorphic Ventricular Tachycardia

Ventricular tachycardia is defined by three or more consecutive ventricular beats. Sustained (>30 seconds) monomorphic VT is a re-entrant rhythm that is usually ventricular in origin. Causes of VT include ischaemic or structural heart disease, post myocardial infarction or cardiac surgery (due to ventricular scarring) and electrolyte disturbance in patients with a long QT interval.

Figure 4: *Causes of long QT interval*

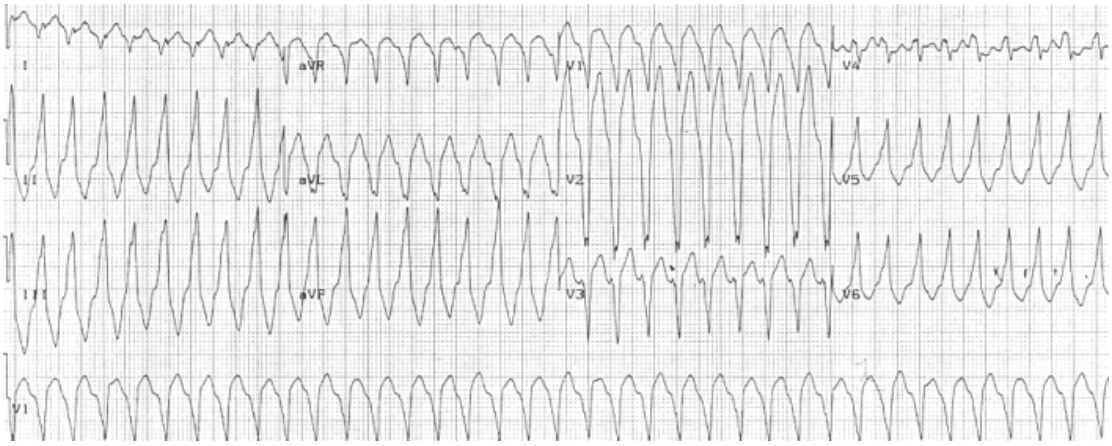
Congenital	Acquired
Roman-Ward Syndrome Lange-Jervell-Neilson Syndrome	Drugs: anti-arrhythmics, erythromycin, antihistamines, tricyclic antidepressants, phenothiazines

The differential diagnosis of VT is supraventricular tachycardia (SVT) with aberrant conduction. Differentiation is notoriously difficult and may be aided by a dose of adenosine - VT will be unaffected, whereas an SVT with aberrant conduction may be terminated or slowed allowing identification of the underlying atrial rhythm. If in doubt, assume that all regular broad complex tachycardias are ventricular in origin. Not only is this safer, but VT is also four times more common than SVT with aberrancy. If there is an irregular rate, it is likely to be atrial fibrillation with bundle branch block.

Figure 5: ECG features suggestive of VT rather than SVT with aberrant

conduction [3]
• Variable S wave in V1 (suggesting atrio-ventricular (AV) dissociation) • Independent P waves, fusion or capture beats (only present in 20-30% of VT) • Positive concordance in all precordial leads • Deep S wave in V6 • RSR pattern in V1 • QRS width greater than 0.14 seconds with right bundle branch block • QRS width greater than 0.16 seconds with left bundle branch block • Pronounced left axis deviation (-90 to 180°)

Figure 6: *ECG demonstrating monomorphic VT*



Initial management of the patient with VT depends on the rate, duration and the haemodynamic status of the patient. Hypotension (SBP<90mmHg), chest pain, heart failure, ventricular rate >150 bpm and a reduced conscious level are all indications for prompt, synchronised defibrillation. Patients who are currently stable and at minimal risk of impending circulatory collapse, can be treated pharmacologically with anti-arrhythmic therapy.

Management of ventricular tachycardia

If haemodynamic compromise is present:

- DC synchronised cardioversion with sedation.
- Correct hypokalaemia or hypomagnesaemia (60mmol KCL in 60ml normal saline over 2 hours minimum, 50% $MgSO_4$ 5ml over 30 minutes).
- If initial cardioversion unsuccessful, administer IV 150mg amiodarone over 10 minutes and consider repeated cardioversion. Bradycardia and hypotension can result from amiodarone administration, in which case the rate of infusion should be decreased.
- Second line pharmacological agents include lignocaine and sotalol (not if ejection fraction in known or suspected to be less than 40%).
- Consider overdrive pacing.
- Seek cardiological opinion if necessary.

In the absence of haemodynamic compromise:

- Correct hypokalaemia or hypomagnesaemia.
- Give 150mg of IV amiodarone over 10 minutes, or IV lignocaine 50mg over 2 minutes repeated to a maximum of 200mg.

- Consider synchronised DC cardioversion under sedation and/or repeated amiodarone administration.

Polymorphic Ventricular Tachycardia

Polymorphic VT with a normal QT interval typically degenerates into VF and is usually due to ischaemia. It virtually always causes significant haemodynamic compromise and therefore synchronised DC cardioversion is indicated. If the patient is stable, the same treatment algorithm to that of monomorphic VT should be followed.

Torsades de Pointes

Torsades de pointes ("twisting of the points") is a polymorphic form of VT with a prolonged QTc interval (< 460 msec).[2] It is recognised by beat-to-beat variation and a constantly changing axis.

Figure 7: *Rhythm strip showing torsades de pointes.*

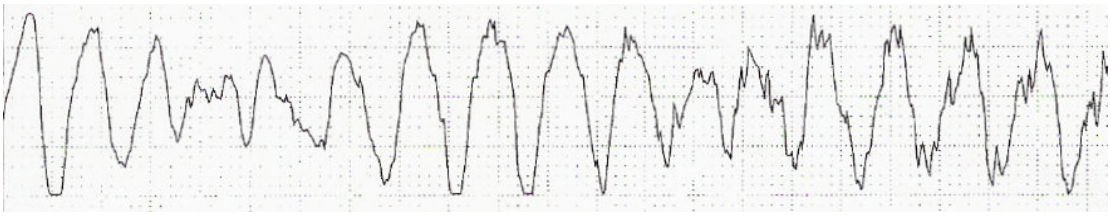


Figure 8: Causes of Torsades de Pointes

- Drugs
  - Anti-arrhythmics – predominantly class Ia and Ic drugs: procainamide, disopyramide, sotalol
  - Phenothiazines
  - Quinidine
  - Antibiotics: erythromycin, pentamidine, ketoconazole
  - Antihistamines: terfenadine, atemizole
  - Tricyclic antidepressants, e.g. clomipramine
- Congenital QT prolongation
  - Romano-Ward syndrome
  - Lang-Jervell-Neilson syndrome
- Electrolyte disturbance
  - Hypokalaemia
  - Hypomagnasaemia
  - Hypocalcaemia
- Profound bradycardia
- Myocardial infarction or ischaemia
- Neurological insults – subarachnoid haemorrhage, stroke, encephalitis
- Arsenic and insecticide poisoning

Management of torsades de pointes

- DC cardioversion if haemodynamic instability
- Give IV magnesium sulphate (regardless of magnesium levels) 5ml of 50% over 30 minutes.
- Correct other electrolyte abnormalities (particularly potassium).

- Stop precipitating medications.
- Consider overdrive pacing at 100bpm, increasing the rate if needed.
- Consider beta blocker administration e.g. esmolol.
- Consult a cardiologist if necessary.

Narrow complex tachycardias

Narrow complex tachycardias originate either from the atria or AV node (i.e. supra-ventricular). It is useful to classify them into regular and irregular rhythms as this assists with formulation of a differential diagnosis. Regular narrow complex tachycardias include sinus tachycardia, atrioventricular node re-entrant tachycardia (AVNRT), AV re-entrant tachycardia (AVRT); ectopic atrial tachycardia and atrial flutter with fixed conduction. Irregular narrow complex tachycardias include atrial fibrillation, atrial flutter with variable block and multi-focal atrial tachycardia. The management of narrow complex tachycardias is summarised below:

Figure 9: *Management of Narrow Complex Tachycardias*

- Check airway and breathing. Administer high-flow oxygen.
- Assess for signs of haemodynamic collapse. If present, proceed immediately to synchronised DC cardioversion. If initial three shocks are unsuccessful, 150mg amiodarone can be administered over 10 minutes before further shocks are attempted.
- If no haemodynamic compromise present, treatment options depend on the onset and duration of the arrhythmia. If the arhythmia is known to have a duration of less than 24 hours, heparin should be given and synchronised cardioversion attempted. Cardioversion of a un-anticoagulated patient of longer than 24 hours duration carries an unacceptably high risk of thromboembolism.
- Perform a trial of vagal manoeuvres which include a valsalva manoeuvre, application of cold water to the face, carotid sinus massage (exclude a carotid bruit before doing this).
- If there is no contraindication to adenosine (asthma – may precipitate bronchospasm, AV block or known/suspected WPW) administer 6mg rapidly into a large vain followed rapidly by a saline flush. This may slow the ventricular response rate to allow correct identification of the atrial rhythm and in some cases may even cardiovert the rhythm. The patient should be on a cardiac monitor and warned to expect a transient feeling of chest tightness, nausea or flushing. If this fails, three further doses of 12mg adenosine may be administered at 2 minute intervals.
- Other pharmacological therapies include amiodarone, esmolol, digoxin and verapamil. Verapamil should be avoided in the presence of WPW syndrome, recent beta blockade usage or signs of impaired left ventricular function exist. Caution should be exercised when using multiple pharmacological agents.
- If the arrhythmia persists despite the above measures, a cardiological opinion should be sought regarding the possible indication for overdrive pacing.

Regular narrow complex tachycardias

- **Sinus Tachycardia**

Sinus tachycardia is the commonest arrhythmia in critically ill patients and often reflects underlying systemic illness. It rarely requires drug therapy, and treatment should be aimed at identification and treatment of the underlying cause. Common causes include sympathetic stimulation (laryngoscopy, pain, anxiety), hypoxia, hypercarbia, drugs (e.g. iontropes or digoxin toxicity), hypovolaemia, thyrotoxicosis, infection, pulmonary embolism, pneumothorax, cardiac tamponade and drug/alcohol withdrawal.

- **Atrio-Ventricular Re-Entrant Tachycardia (AVRT)**

AVRT occurs when an accessory tract that bypasses the AV node exists, and has an incidence of 0.1 to 0.3%. As a result of the accessory pathway, the ventricles are activated prematurely as a normally conducted impulse would be slowed at the AV node. It is this premature excitation that produces the characteristic delta wave and prolonged PR interval (>0.12 seconds) seen on ECG.

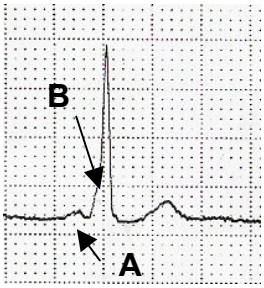


Figure 10: *Demonstrating the characteristic ECG findings of a patient with Wolff-Parkinson White (WPW) syndrome, with a short PR interval (A) and slurred upstroke of the QRS complex (B), known as the delta wave.*

The diagnosis of WPW is reserved for patients with pre-excitation *and* tachyarrythimias. AVRT can produce a narrow complex tachycardia if orthodromic conduction occurs, whereby conduction occurs down through the AV node and back up the re-entrant track. Conversely, a broad complex tachycardia is seen if the impulse travels down the re-entrant pathway and back up through the AV node (antidromic conduction).

If patients with AVRT develop atrial fibrillation (AF) it can result in the generation of a rapid ventricular response (due to bypassing of the rate limiting AV node), which can progress into VF. WPW patients in AF are often hypotensive with evidence of hypoperfusion and should therefore be considered for primary synchronised cardioversion. If stable, the physiological treatment aim is to prolong the anterograde refractory period of the accessory pathway relative to the AV node. This slows the rate of impulse transmission through the accessory pathway and slows the ventricular rate. This is in stark contrast to the treatment principles of AF in the non-WPW patient, where the aim is to slow the refractory period of the AV node. Therefore drugs that are conventionally used to treat AF could have disastrous consequences. Adenosine, verapamil and digoxin should therefore be used with caution in patients suspected of having WPW. Procainamide, ibulitide and flecainide are preferred agents

as they slow conduction through the bypass tract. Amiodarone is safe in WPW patients.

- **Atrio-Ventricular Nodal Re-Entrant Tachycardia (AVNRT)**

AVNRT (also known as junctional tachycardia) typically occurs with sudden onset of 140-180 bpm and is indicated by a lack of P waves. It is more common in females and those with structural heart disease. AVNRT involves dual AV nodal pathways and re-entry into the atrium. The principle aim is to block AV conduction, either with vagal manoeuvres or AV blocking drugs such as adenosine, beta-blockers and digoxin. Long-term preventative treatment includes beta-blockers or radio-ablative treatment.

- **Atrial Flutter**

Atrial flutter is a re-entrant tachycardia identified by flutter waves (P waves in a saw tooth fashion) in the inferior leads at a rate of approximately 300 bpm. Two-to-one conduction block is often present resulting in a ventricular response rate of 150 bpm, however this can change suddenly. Two-to-one block results in a regular tachycardia, but variable block will cause an irregular narrow complex tachycardia. Again, the unstable patient should be synchronised DC cardioverted, which has a success rate of 95-100% [4]. If this fails, treat in the same way as atrial fibrillation.

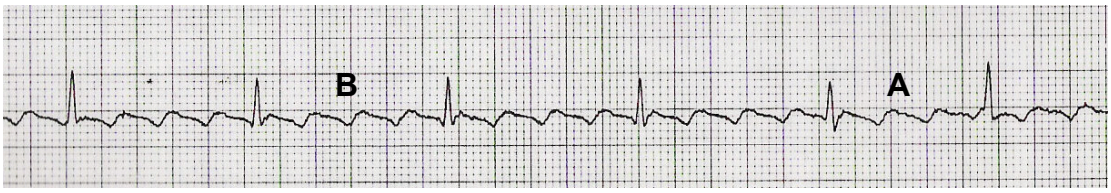


Figure 11: *Rhythm strip demonstrating the saw tooth pattern of P waves seen in atrial flutter. The block varies between 3:1 (A) and 4:1 (B).*

Irregular Narrow Complex Tachycardia

- **Atrial Fibrillation (AF)**

Atrial fibrillation is the most common arrhythmia in the ICU. The prevalence of AF in the general population increases exponentially with advancing age. Risk factors for the development of AF include structural heart disease (70% in Framingham study over 22 year follow up),[5] hypertension (50%),[5] valvular heart disease (34%)[6] and left ventricular hypertrophy.[2]

Post-operative AF is common, particularly after cardiac surgery when the incidence is 25-40% with a peak onset at day two. If there are signs of cardiovascular instability, heparin should be administered and the patient cardioverted. However if stable, recent studies (AFFIRM, RACE) indicate that rhythm control does not prevent morbidity and mortality from cardiovascular causes when compared with rate control[7]. In addition, post-operative AF in this setting often runs a self-correcting course, with restoration of sinus rhythm in 90% of patients by 6-8 weeks. Anti-coagulation will be required for this period, as the stroke risk for a patient without anticoagulation in AF is quoted at 2% per year.

Management of AF

- If haemodynamic compromise exists, proceed immediately to synchronised DC cardioversion under sedation. If three initial shocks are unsuccessful, 150mg amiodarone can be administered over 10 minutes before further shocks are attempted. Electrical cardioversion may be more effective if the defibrillator pads are placed in the anterior/posterior position to increase the current flowing through the atria. If electrical cardioversion is unsuccessful IV 300mg amiodarone can be administered over one hour and repeated as necessary. Amiodarone has a conversion rate of upto 80% in AF, however extreme caution should be exercised when administering amiodarone to the critically ill patient on account of the risk of serious acute pulmonary toxicity.[8] In contrast, ibutilide represents a relatively new anti-arrhythmic agent that is reported to have high conversion rates and lower pulmonary toxicity.[7]
- If no haemodynamic compromise is present, treatment options depend on the onset of the AF. If the AF has a duration of less than 24 hours, heparin should be given and the patient cardioverted. Eliciting the onset of arrhythmia is often difficult in practise and is only possible if he patient has been symptomatic or on a cardiac monitor. Cardioversion of a patient with AF of longer than 24 hours duration carries an unacceptably high risk of thromboembolism.
- Perform a trial of vagal manoeuvres which include a valsalva manoeuvre, application of cold water to the face, carotid sinus massage (exclude a carotid bruit before doing this).
- Correct any precipitating factors such as electrolyte imbalance and treat underlying problems such as sepsis, cardiac failure and thyrotoxicosis.
- If there is no contraindication to adenosine (asthma – may precipitate bronchospasm, AV block or known/suspected WPW) administer 6mg rapidly into a large vain followed rapidly by a saline flush. The patient should be on a cardiac monitor, ideally with a continually printed rhythm strip, which is found on most defibrillators. Warn the patient to expect a transient feeling of chest tightness, nausea or flushing. This may slow the ventricular response rate to allow confirmation of the atrial rhythm and occasionally may cardiovert the rhythm. If this fails, repeat with 12mg up to a maximum of 36mg. The effects of adenosine are potentiated by dipyridamole and antagonised by theophylline.
- Other pharmacological therapy includes amiodarone, esmolol, digoxin, diltiazem and verapamil. Avoid verapamil in the presence of WPW syndrome, recent beta blockade usage or signs of impaired left ventricular function. Caution should be exercised when using multiple pharmacological agents.
- If the arrhythmia persists, a cardiological opinion should be sought regarding the possible indication for atrial overdrive pacing.
- Commence formal anticoagulation with heparin if the AF persists for longer than 24 hours.
- DC cardioversion should be attempted 4 weeks after anti-coagulation in patients without significant mitral valve disease or left atrial impairment. If cardioversion is successful, continue anticoagulation for a further 4 weeks as patients continue to be at risk from embolic phenomena. Patients in whom cardioversion is unsuccessful or inappropriate should be maintained on digoxin and warfarin (if appropriate).

- **Multifocal atrial tachycardia**

Multifocal atrial tachycardia is an irregular atrial tachycardia diagnosed by the existence of 3 or more P wave morphologies and PR intervals. Hypoxia is the

commonest cause of this abnormality, however it may also be associated with theophylline, metabolic derangement and end-stage cardiomyopathy. Treatment consists of treating the underlying cause. AV blockers may be useful to stabilise the patient in the interim.

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