Frimley Health NHS Foundation Trust

Acute presentation of cardiac arrhythmia

Key Points

- Management of arrhythmia requires an assessment of the patient as a whole and not just an assessment of the ECG
- Follow the ALS algorithm in emergency situations
- Use clinical judgement to decide on best course of action, a guideline cannot accommodate for all eventualities
- All atrial fibrillation patients should risk assessed for stroke an offered DOAC where appropriate

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Abbreviations

AF	Atrial Fibrillation
AV	Atrio-ventricular
CABG	Coronary Artery Bypass Graft
DCCV	Direct Current Cardioversion
DOAC	Direct Oral Anticoagulant
ECG	Electrocardiogram
ESC	European Society of Cardiology
ICD	Implantable Cardiac Defibrillator
IHD	Ischaemic Heart Disease
INR	International Normalised Ratio
IV	Intravenous
LAHB	Left Anterior Hemi-block
LBBB	Left Bundle Branch Block
LPHB	Left Posterior Hemi block
MI	Myocardial Infarction
OSA	Obstructive Sleep Apnoea
PR	PR interval measured in milliseconds
PTCA	Percutaneous Transluminal Coronary Angioplasty
QR	QR interval measured in milliseconds
QRS	QRS – ventricular depolarisation
QT	QT interval measured in milliseconds
QTc	Corrected QT interval, usually with Bazzet's correction – others are available
SVT	Supraventricular Tachycardia
VT	Ventricular Tachycardia
TTR	Time in Therapeutic Range
WPW	Wolff Parkinson White

Introduction and Scope

The following guidelines are intended to assist with the acute and inpatient management of cardiac arrhythmias and are aimed at both non-cardiologists and cardiologists. They are not a definitive pathway to be followed in every clinical circumstance. These guidelines are based on current ESC recommendations for atrial and ventricular arrhythmias, which can be accessed here: Guidelines

Use clinical judgment or contact the Cardiology team via the advice line if unsure.

Bradycardias

Sinus Bradycardia

Generally, investigation may be indicated, but no treatment is warranted unless symptoms develop. It usually presents as sick sinus syndrome with intermittent sinus node failure, sinoatrial block, and sinus pauses, which could lead to breathlessness or syncope.

Sinus bradycardia can occur through intrinsic (e.g., sinus node disease) or extrinsic (e.g., vagal/central) mechanisms.

The primary actions are:

1) Exclude and address other systemic causes, such as hypothyroidism, hypothermia, anorexia nervosa, intracranial events (including cerebral oedema or haemorrhage), OSA, myocarditis, and infections such as Lyme's disease, if no other cause is identified.
2) Identify side effects of non-cardiac medicines, for example, donepezil or lithium (see Appendix 1).
3) Stop or reduce the dose of cardiac medications, including beta-blockers, calcium channel blockers (verapamil/diltiazem), digoxin, flecainide, and amiodarone (see Appendix 1). Patients with a vagal origin of their bradycardia generally do not require pacing.

If treatment is deemed necessary, i.e. there is evidence of haemodynamic compromise, give atropine 600-1200 micrograms IV in the first instance and assess the response. Alternatively, isoprenaline can be given as an infusion (see Isoprenaline for Bradycardia guideline) in higher dependency areas with monitoring capabilities.

Persistent symptomatic bradycardia may require pacing (temporary or permanent). If temporary pacing is needed, transvenous pacing under x-ray guidance should be performed with a view to permanent pacing. Transcutaneous pacing is only to be used as a bridge to more definitive treatment in the cardiac catheter lab for transvenous pacing.

For advice, contact the cardiology advice line by phone. Out of hours, contact the interventional consultant cardiologist on call.

Chronotropic incompetence is a failure of the sinus node to increase the heart rate to meet the greater cardiac output demand of stress. It can present with SOB and lethargy. In the absence of presyncope or syncope, this can be managed as an outpatient.

Atrioventricular Block

This is typically split into four categories.

1) First-degree heart block, i.e. a prolonged PR interval beyond 200ms.
2) Mobitz Type 1 second-degree AV block. A progressively prolonged PR interval is seen, with a dropped QRS complex preceding a normal PR interval.
3) Mobitz Type 2 second-degree AV block. A fixed PR interval, where a proportion of atrial activations do not reach the ventricle, without prolongation of the PR interval beforehand.
4) Complete heart block. Complete AV dissociation is commonly described as p waves marching through the QRS complexes. The QRS rate will be regular because it is an escape rhythm; however, the QRS complex may be narrow or wide.

Management is based on the arrhythmia's symptoms and haemodynamic consequences.

1) First-degree heart block: This generally requires no treatment and does not require investigation unless there are symptoms suggestive of higher degrees of block, i.e., breathlessness, pre-syncope, or syncope.
    a) It can be managed as an outpatient with a routine cardiology referral if there are symptoms suggestive of a higher degree of block, i.e. dizziness, lethargy, syncope, which have not correlated with bradycardia during the acute presentation.

2) Mobitz type 1 second-degree AV block: This is typically due to increased vagal tone; however, if the underlying PR interval is prolonged, it may indicate more advanced AV nodal disease. Again, investigation depends on the patient's symptoms, age, and comorbidities, but the patient should have an outpatient referral to cardiology if there are associated symptoms. However, it does not require immediate treatment unless there is syncope correlating with AV block or if the patient is highly symptomatic.

3) Mobitz second-degree AV block 2: This requires discussion with the cardiology team with a view to admission, regardless of symptoms, due to prognostic implications. There is a high likelihood of developing complete heart block, which is associated with sudden death. Not all patients will require pacing, but this is a cardiology consultant's decision.

4) Complete Heart block -Admit under cardiology for consideration of a pacemaker insertion.

Any bradycardia causing haemodynamic compromise should be managed as an emergency and will require urgent discussion with cardiology and possibly intensive care to initiate:

1) Atropine
2) Isoprenaline infusion
3) Transcutaneous pacing
4) Temporary pacing wire
5) Permanent Pacing

Features of complete heart block which make these treatments more likely are:

1) Heart rate below 40bpm
2) A broad ventricular escape rhythm QRS >120ms
3) QTc Prolongation (>500ms)

4) Recurrent > 3s pauses associated with loss of cardiac output

If any of these features are present, then a discussion with the cardiologist team should be had, regardless of the time of day, to consider temporary pacing.

Bradycardia Pharmacology

Atropine

0.6-3mg in 0.6mg increments IV
Atropine can be helpful in emergencies involving high-degree AV block, but it generally has no effect if the AV node fails completely. Atropine abolishes all vagal tone and thus requires at least some AV function to be effective. It frequently leads to urinary retention and delirium in the elderly.

Isoprenaline

Is given as an infusion in high dependency areas with cardiac monitoring. See separate guideline on isoprenaline use, Isoprenaline for Bradycardia. It is recommended when there are features of shock caused by bradycardia.The prescription should not be based on heart rate alone.
If the patient fails to respond to atropine and isoprenaline, external pacing may be required before a Temporary Pacing wire or Permanent Pacemaker is inserted.

Transcutaneous Pacing

Tips;

1. Remember to attach the ECG monitoring electrodes as well as the defib pads
2. Adjust the rate and increase the energy output delivered until electrical capture is achieved (check that a QRS follows each pacing spike and that there is a central pulse – indicating mechanical capture)
3. Intravenous analgesia and/or sedation is required- contact an anaesthetist.

It is optimal for patients to be offered a permanent pacemaker as soon as possible; however, this is not always feasible. It is reasonable to place a transvenous wire via the femoral, axillary, or jugular vein until appropriate staff are available or investigations have been performed to determine the suitable device for implantation.

Specific Indications to Consider Emergency Temporary Pacing

Particularly, be vigilant of patients with STEMI or large NSTEMI with AV block or rhythm disturbance who may require temporary pacing around the time of emergency angioplasty.
Situations where a temporary pacing wire may be considered;

- Bilateral bundle branch block (alternating BBB or RBB with alternating LAHB/LPHB)
- 2nd or 3rd degree AV block after an acute anterior MI -usually transient but will require temporary pacing if the patient is compromised
- Any symptomatic bradycardia resistant to medication
- 2nd or 3rd-degree AV block with haemodynamic compromise or syncope at rest
- Asymptomatic AV block with severe bradycardia (<40bpm)+/- QRS>120ms +/- QTc prolongation >500ms)
- VT secondary to bradycardia, e.g. Torsades de Pointes
- Suppression of drug-resistant VT or SVT

- Drug overdose, e.g. digoxin, beta-blockers, verapamil
- Support for procedures that may promote bradycardia

  Contact the cardiology consultant for advice and with a view to temporary pacemaker.

Supraventricular Tachycardias

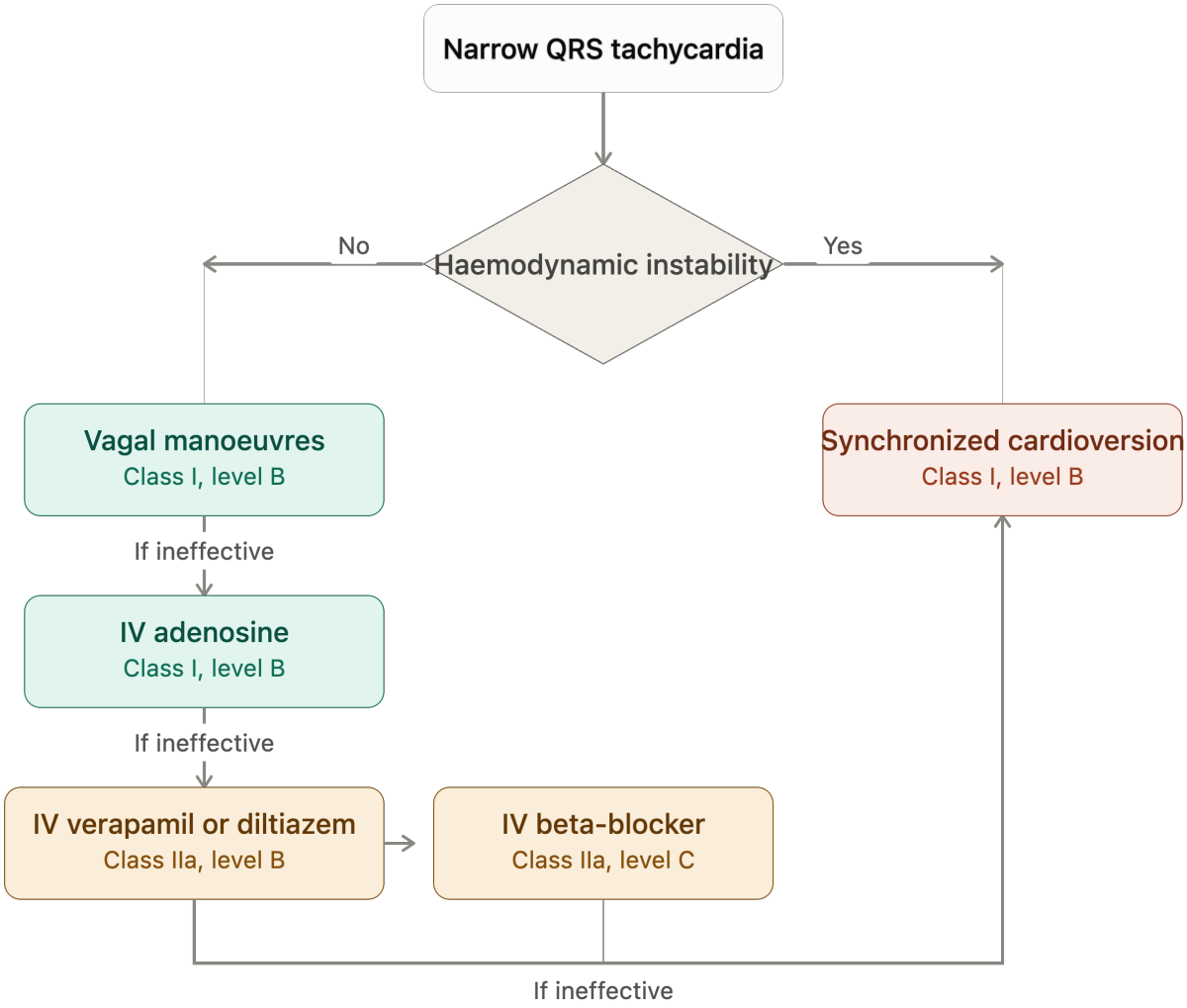
These include

- Atrial Fibrillation
- Atrial Flutter
- Atrial Tachycardia
- Atrioventricular Nodal Re-entrant Tachycardia (AVNRT - commonly called “SVT”)
- Atrioventricular re-entrant Tachycardia (AVRT - the tachycardia that patients with WPW have)

As with all arrhythmias, the 12-lead ECG is essential and must be obtained in all cases. Accurate diagnosis is essential, as therapy depends on the specific rhythm. Arrhythmias can be paroxysmal or persistent, and treatment should be tailored accordingly. Paroxysms should be terminated, and preventive treatment should be started. Chronic arrhythmias that cannot be terminated should be slowed if necessary.

Regular Narrow Complex Tachycardia

In the absence of an established diagnosis, the following flowchart can be used as a guide for the treatment of narrow QRS tachycardias which is based on the ESC algorithm .(ESC SVT guideline)



Vagal manoeuvres

Carotid Sinus Massage

- patient in a supine position with the neck extended and rotated to the opposite side
- Auscultate carotid arteries for bruits (carotid stenosis >70% is a contraindication)
- apply pressure on the carotid sinus for 5 to 10 s
- observe for changes in the tachycardia or termination.

Valsalva Manoeuvre

- Whilst supine, the patient is asked to take a deep breath and blow out against a closed glottis for 10 to 15 seconds (consider asking the patient to blow into a 10 mL syringe until the plunger moves for 10 to 15 seconds)

Modified Valsalva Manoeuvre (has more than double the success rate of the standard VM)

- The patient blows against a closed glottis or syringe, initially in an upright sitting position, for 15 seconds, then is lowered into a supine position with the legs brought into either a knees-to-chest position or extended at a 45- to 90-degree angle to the torso. The patient

continues to blow outwards for 10 to 15 seconds, and the legs are held in position for 45 seconds to one minute.

Adenosine administration

Rapid bolus with immediate 20 ml sodium chloride 0.9% flush using a large, centrally located (e.g. antecubital) vein

Dosing should be as per trust guidelines: Initially, 6mg; if there is no response, repeat with 12mg; if there is no response, repeat with 18mg and then 24mg (unlicensed doses of 18mg & 24mg) Further doses should be discussed with a Cardiology Consultant.

The purpose of adenosine administration is to induce complete AV block, thereby terminating tachycardias that depend on the AV node to continue (i.e., AVNRT and AVRT).

If the adenosine dose is sufficient to cause AV block but does not terminate the tachycardia, then other therapies, such as AV nodal blockers, should be used to slow the rate, or other rhythm control strategies, such as flecainide, amiodarone, or DCCV, should be considered.

Side effects include transient dyspnoea, facial flushing, chest pain, AF, bronchoconstriction in severe obstructive airways disease, a metallic taste in the mouth, and a feeling of “impending doom”.

ALWAYS RECORD THE RHYTHM STRIP DURING ADENOSINE ADMINISTRATION; it will tell you the diagnosis.

Contraindications include asthma or COPD with evidence of bronchospasm. – use of IV Verapamil 2.5mg-5mg IV over 2 mins as an alternative.

AVNRT and AVRT

Once terminated, both can be managed as outpatients and do not require admission unless other patient factors require it. There is no need to check the troponin unless there are clear ischaemic symptoms.
Exceptions to this are patients with syncope due to Wolff-Parkinson-White (WPW) syndrome, atrial flutter with 1:1 conduction, or arrhythmia with associated new heart failure, who should be referred to the cardiology team for initial inpatient management.

Supraventricular arrhythmias may be treated by ablation as an outpatient; rarely, an inpatient EP study +/- ablation may be indicated.

DO NOT GIVE AV NODAL-BLOCKING DRUGS TO WPW PATIENTS WITH PRE-EXCITED ATRIAL FIBRILLATION.

Do not use amiodarone in this situation as it also has AV nodal slowing properties.
An ECG with Pre-excited AF will show an irregularly irregular rhythm, with varying QRS complex widths, typically broader at faster rates.

The management is with urgent DCCV. An in-hours cardiology consultation should be made, and an outpatient plan should be made before discharge. Do not discharge these patients without review by a cardiology consultant.

Synchronised DCCV energy for atrial arrhythmia can start at 200J and if this fails escalate to 360J

Follow up

All patients presenting with a symptomatic regular, narrow, complex tachycardia should be considered for referral to the arrhythmia clinic as a routine referral. Truly first-time events with no previous palpitations can be discharged without follow up, however any patient with recurrent symptoms and documented SVT should be seen in an arrhythmia clinic by either consultant or arrhythmia nurse.

If the arrhythmia is associated with syncope, or near syncope, or the heart rate is documented above 200bpm then the follow up should be urgent and inpatient advice sought, regardless of the presence of previous symptoms.

Atrial Fibrillation

AF management should be guided by its temporality and is defined as being either:

1) First-time AF: self-explanatory
2) Paroxysmal AF: episodes spontaneously resolving and lasting less than 7 days
3) Persistent AF: lasting longer than 7 days or requiring cardioversion
4) Long-standing persistent AF: lasting longer than 1 year
5) Permanent AF: shared decision-making that further rhythm control is not appropriate.

Clinical Scenarios

First time AF – as the main presenting complaint

A good rule of thumb is to rate-control the patient and offer outpatient cardioversion after discussing it with cardiology and administering appropriate anticoagulation.

A large proportion of patients will have spontaneously cardioverted by their appointment. The figure below from the ESC represents a reasonable first-line approach; however, the patient's individual clinical situations and preferences should be considered. Additionally, it is reasonable to offer inpatient rhythm control to newly diagnosed AF patients if they are highly symptomatic, unable to be discharged due to symptoms or cannot achieve rate control.

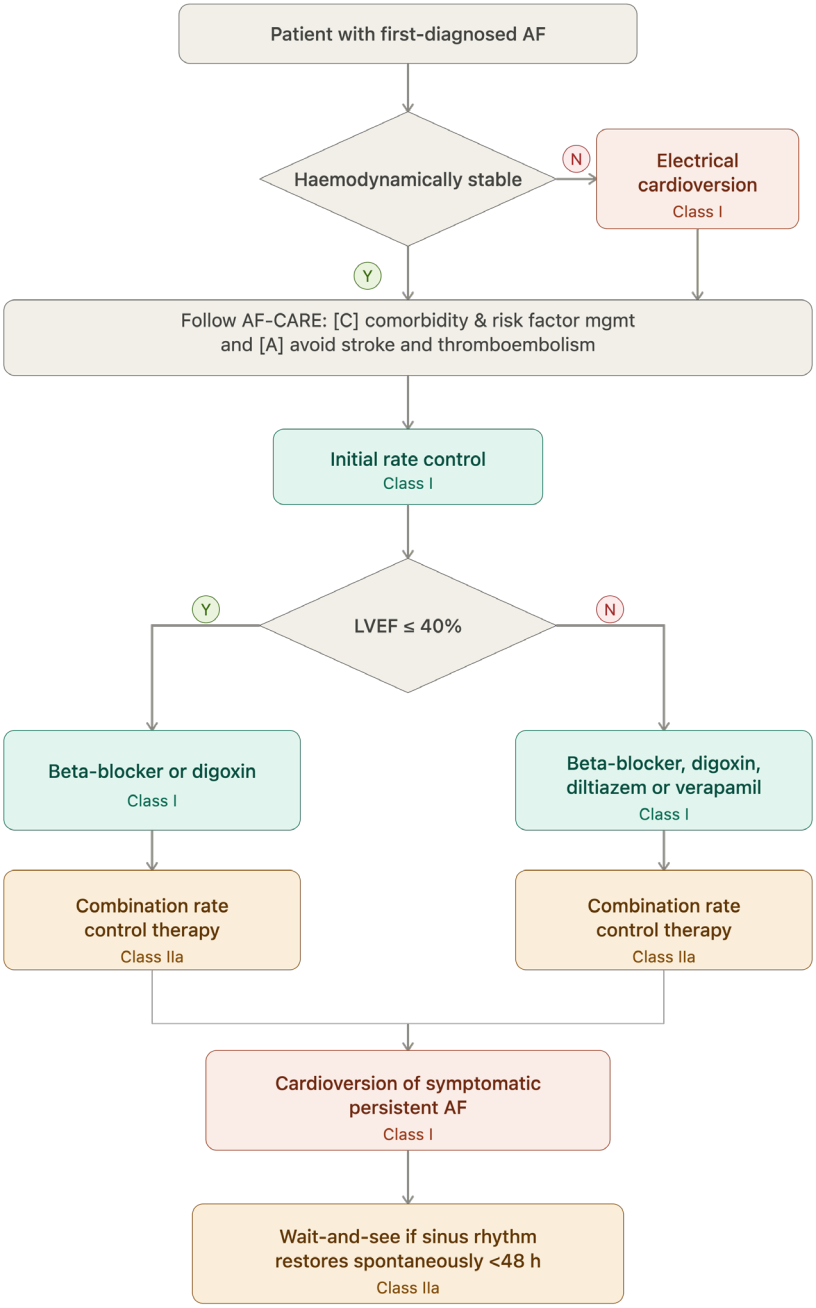
Patients with their first episode of AF leading to hospital admission should be referred to the arrhythmia nurse team to be seen as outpatients if they are candidates for rhythm control. Please discuss with cardiology if unsure if this is the case.

Generally, rhythm control candidates;

- Low frailty score
- Have had AF for less than 2 years.
- Alcohol consumption within recommended limits
- Normal thyroid function

There are no absolute rules to rhythm control and patient specific advice should be sought in grey areas.

Frail patients with incidental AF can be discharged to their GP with appropriate anticoagulation and rate control medicines as needed.



Exacerbation of paroxysmal AF

Patients experiencing recurrent episodes of AF should have a clear strategy to manage their symptoms. If this plan is ineffective, further rhythm-control options should be discussed in the acute setting in consultation with cardiology. Typically, patients use a pill-in-the-pocket strategy, with or without regular AV nodal blocking medications.

The maximum dose of flecainide in 24 hours is 300mg. IV flecainide (unlicensed medication) requires cardiac monitoring. Increasing doses of flecainide and sotalol necessitate ECG monitoring at initiation and after one week to assess for QT and QRS interval prolongation; the arrhythmia nurses will be able to facilitate this process.

Ensure patients have follow-up appointments with their cardiology team to review their treatment plan, as admission to A&E indicates a failure of the current strategy. If no follow-up is in place, request an arrhythmia nurse clinic appointment.

Generally, these patients do not require admission, but the severity of symptoms and any possible concurrent issues should guide the decision.

Recurrence of persistent AF

If the patient has experienced recurrent AF after previous cardioversion, the goal of treatment is to provide rate control and haemodynamic stability before presenting options for long-term rhythm control.

The decision to offer further cardioversion or ablation is complex and should be made by the arrhythmia team. Again, the ability to control the heart rate and the severity of symptoms should determine whether the patient requires inpatient rhythm control.

Early outpatient cardioversion is usually a reasonable course of action, but may require additional pharmacotherapy to be effective.

"Fast AF" in the context of other illnesses

Unless the patient is haemodynamically compromised, treatment of the underlying cause of the other illness and rate control should be the mainstays of therapy. If AF is new, i.e., first-time or paroxysmal, consider urgent cardioversion if there is haemodynamic instability, myocardial ischaemia, or heart failure.

Principles of Treatment - the 4 Pillars – to consider for all AF patients

Rhythm control, rate control, anticoagulation, risk factor modification

Rhythm Control:
DC cardioversion should be considered when there is haemodynamic compromise. In the absence of haemodynamic compromise, inpatient cardioversion is rarely required (mostly for fast AF/atrial flutter resistant to rate control); otherwise, DC cardioversions routinely occur as outpatients after at least 30 days of uninterrupted anticoagulation.

The decision to use a pill-in-the-pocket strategy for patients with paroxysmal atrial fibrillation should be made after consultation with the cardiology team.

If the patient is highly symptomatic but not haemodynamically unstable, AF of clearly very recent onset (<24 hours) can be terminated by IV flecainide (1-2mg/kg over 10 min; maximum dose 150mg) as an alternative to DCCV.

In the presence of heart failure or acute ischaemia, amiodarone should be used (300mg bolus via a small cannula in a large vein inserted as far down the arm as possible but not directly over the wrist joint, or centrally, then a 900mg infusion over 23 hours).
Review and treat the patient for underlying pathologies such as hyperthyroidism, sepsis, and electrolyte imbalance.

Rate Control:
General considerations

- Target a HR of <110 bpm unless there are ongoing symptoms or suspicion of tachycardia-induced cardiomyopathy.
- Adding digoxin to a beta-blocker is indicated if adequate rate control cannot be achieved with a single agent, but combining a beta-blocker with a calcium channel blocker or a triple combination with digoxin is not advised.

1. Beta-blockers
   Contraindicated in severe asthma, use with caution in tachycardia/bradycardia syndrome, acute heart failure/cardiogenic shock.

2. Calcium channel blockers (rate-limiting – diltiazem or verapamil)
   Contraindicated if LVEF ≤40%

3. Digoxin
   Oral loading dose - Preferred over IV route

Cardiac side-effects of digoxin are increased by acidosis, hypokalaemia, hypomagnesaemia, hypercalcaemia, hypoxia and hypothyroidism.

For rapid rate control prefer the use of intravenous beta blockade, i.e. metoprolol 2.5mg IV, as a first line agent, with oral agents used for longer term rate control.

Anticoagulation:
Unless otherwise contraindicated, patients with AF or atrial flutter should be anticoagulated if they have a CHA2DS2VA score of 1 or above, preferably with a novel oral anticoagulant (note that sex is no longer recognised as a point-scoring risk factor) or, if not appropriate, Warfarin (e.g., valvular AF) if they are at risk of developing a cardiogenic embolism.

Please refer to the appropriate guidance in the BNF for prescribing the correct DOAC doses based on patient weight and renal function, or to the specific Frimley Health guidelines on the intranet.

Do not withhold anticoagulation solely because the patient is at risk of falling. However, frail patients with frequent falls and subsequent injuries may not derive the same benefit from anticoagulation. Withholding anticoagulation should be discussed between a senior member of the medical team and the patient or next of kin.

Risk Factor Modification
Successful chronic care of patients with AF is heavily dependent on risk factor modification. Patients should be informed of the roles of diabetes, obesity, hypertension, sleep apnoea and alcohol use, all of which increase the likelihood of recurrent episodes and hospitalisations. Similarly, optimisation of heart failure treatment should be initiated if necessary.

Discharge Criteria for patients who have AF

1. Heart rate at rest <110bpm
2. Completed CHADS2VA score and DOAC offered if appropriate
3. Patient symptoms are adequately controlled for managing at home whilst awaiting OP appointment or cardioversion IF needed or appropriate advice is given to the GP is OP appointment is not necessary.
4. Thorough workup completed for investigation of causes and reversible factors, including sepsis, anaemia, metabolic derangement and thyrotoxicosis

Ventricular Tachycardia

Ventricular Tachycardia (VT) can vary from a critical life-threatening condition to a routine finding on a Holter monitor with minimal consequences.

Important Differentiating Features to establish at the presentation

- Sustained vs Non-sustained: Non-sustained VT is more than three beats of ventricular origin and lasts less than 30 seconds. The risk of death is far higher in sustained VT, and therefore, the management is very different.
- Symptomatic - not all VT yields symptoms
- Haemodynamic Compromise - not all VT causes low blood pressure
- Underlying heart disease - VT in normal hearts can, in some cases, be "benign" - i.e. not associated with mortality, but probably with morbidity.

The presentation of sustained monomorphic ventricular tachycardia, is suggestive of the presence of significant structural heart disease with some exceptions and requires inpatient investigations

Monomorphic

The commonest causes include acute infarction/ischaemia and chronic left ventricular scarring after infarction.
First, correctly diagnose the rhythm by examining the 12-lead ECG. If this cannot be obtained because of haemodynamic collapse and urgent DC shock is required, at least record the rhythm strip during cardioversion.
Most instances of VT can be correctly diagnosed through ECG interpretation, but if in doubt, **treat broad complex tachycardia as VT**.
Features of VT include:

- Wide QRS complexes (more than 0.14 sec or 3.5 small squares)
- AV dissociation sometimes with capture and fusion beats
- A leftward axis shift compared to sinus rhythm
- Any previous history of IHD (MI, PTCA, CABG)

Treatment:

As with all rhythms, this depends on the clinical situation. ALS guidelines should be followed.
If the patient is hypotensive, in cardiac failure or has symptoms of cardiac ischaemia, DC cardioversion should be undertaken urgently.
Call 2222 for anaesthetic support and place defib pads ready to shock should the patient lose consciousness.
If no high-risk features are present, initial treatment should be amiodarone (300mg bolus via a small-bore cannula in a large vein or centrally, then a 900mg 23-hour infusion) **or** an intravenous beta-blocker such as metoprolol (2.5 mg stat; the dose may be repeated after 5 minutes).
This should be done in a level 1 care area i.e. CCU, MADU, with defib pads on the patient as patients in VT may drop their blood pressure at any time and require urgent DCCV.
Despite recent ALS guidance, do not seek a procainamide prescription as the drug is not licensed for use in the UK and cardiologists have very little experience with its use.

Do not give more than one additional drug – polypharmacy can be dangerous.

If drug therapy fails, or the patient has poor cardiac function, direct current cardioversion (200-360J) under sedation is the best therapy
Whatever method is used, full resuscitation facilities should be on hand. Inpatient cardiological assessment is mandatory in all cases of sustained VT.

Remember to check electrolyte levels. When the arrhythmia is refractory, magnesium, at an initial dose of 8 mmol (4 mL of 50%), may help, though the evidence is patchy.

NB: DO NOT TREAT A POSSIBLE VT WITH VERAPAMIL

Some patients presenting with ventricular arrhythmias who have an ICD implant may have received shocks from the device. The presence of an ICD does not prevent the use of emergency defibrillation or cardioversion in the event of a cardiac arrest or compromising VT that has not responded to ICD therapy.

Follow the UK Resuscitation Council Guidelines (2025) by defibrillating away from the device (usually left infraclavicular site). Haemodynamically stable VT or successfully treated patients can benefit from immediate ICD reprogramming.
Contact the pacing clinic 9-5 pm Monday- Friday for assistance.

Refractory VT, i.e. recurrent sustained VT despite amiodarone infusion, should be treated as above, with the addition of a lidocaine infusion after discussion with the cardiology team**, including out-of-hours**. If this fails, the final step in the acute pathway is intubation and ventilation to remove the adrenergic drive.

Polymorphic VT
This is less common and usually causes presyncope, syncope, or cardiac arrest, depending on the duration of the arrhythmia. It may be associated with QT prolongation (Torsade de Pointes), bradycardia, or myocardial ischaemia.

Documented polymorphic VT requires early discussion with the cardiology team and inpatient admission to CCU.

Treatment includes

- Temporary pacing
- Beta-blockers
- Potassium and magnesium replacement
- Removal of QT-prolonging drugs
- Urgent assessment for cardiac catheterisation will be necessary in cases where cardiac ischaemia is considered a cause.

Ventricular Ectopic Beats

These are ubiquitous and do not require treatment unless they are causing symptoms such as palpitations or dizziness, when the patient should be referred for investigation and management. The urgency of this or the need for inpatient investigation will depend on the severity of symptoms, e.g., syncope requires inpatient assessment. Frequent ectopy, whether symptomatic or not, may indicate underlying structural heart disease and referral for non–urgent investigation as a minimum requirement is appropriate.

Consider offering low-dose beta-blockers (such as bisoprolol 1.25 mg OD or 2.5 mg OD) to highly symptomatic patients with frequent ventricular ectopic. See Appendix 2 for a more general

overview of how to manage palpitations Those within the low risk (Green box) generally do not require further investigation or follow-up.

Appendix 1: Drugs that may cause bradycardia or conduction disorders

Table 6 Drugs that may cause bradycardia or conduction disorders

	Sinus node bradycardia	AVB
Beta-blockers	+	+
Antihypertensives		
Non-dihydropyridine calcium channel blockers	+	+
Methyldopa	+	-
Clonidine	+	-
Antiarrhythmics		
Amiodarone	+	+
Dronedarone	+	+
Sotalol	+	+
Flecainide	+	+
Propafenone	+	+
Procainamide	-	+
Disopyramide	+	+
Adenosine	+	+
Digoxin	+	+
Ivabradine	+	-
Psychoactive and neuroactive drugs		
Donepezil	+	+
Lithium	+	+
Opioid analgesics	+	-
Phenothiazine	+	+
Phenytoin	+	+
Selective serotonin reuptake inhibitors	-	+
Tricyclic antidepressants	-	+
Carbamazepine	+	+

Continued

Table 6 *Continued*

	Sinus node bradycardia	AVB
Others		
Muscle relaxants	+	-
Cannabis	+	-
Propofol	+	-
Ticagrelor	+	+
High-dose corticosteroids	+	-
Chloroquine	-	+
H_2 antagonists	+	+
Proton pump inhibitors	+	-
Chemotherapy		
Arsenic trioxide	+	+
Bortezomib	+	+
Capecitabine	+	-
Cisplatin	+	-
Cyclophosphamide	+	+
Doxorubicin	+	-
Epirubicin	+	-
5-fluorouracil	+	+
Ifosfamide	+	-
Interleukin-2	+	-
Methotrexate	+	-
Mitroxantrone	+	+
Paclitaxel	+	-
Rituximab	+	+
Thalidomide	+	+
Anthracycline	-	+
Taxane	-	+

AVB = atrioventricular block.

Appendix 2 – Palpitations risk assessment

Back to pathway

Risk Assessment

High risk	Moderate risk	Low risk

• High risk structural heart disease
• Family history of sudden cardiac death under 40 years old
• Family history of inherited heart disease
• 2nd or 3rd degree atrioventricular block
• A history of palpitations with syncope or near syncope
• Palpitations precipitated by exercise
	• History suggests recurrent tachyarrhythmia, AF or flutter
	• Palpitations with associated symptoms including chest pain and light-headedness
	• Abnormal ECG other than 2nd or 3rd AV block
	• History or physical symptoms of structural heart disease, heart failure or hypertension
	• History of symptoms clearly consistent with paroxysmal supraventricular tachycardia (sudden onset and offset of a fast regular heartbeat, with multiple uneventful ambulatory rhythm monitor recordings)
	• Ventricular ectopic:
	  • If underlying heart disease is suspected from clinical assessment and/or ECG, or
	  • If ectopic are frequent or if ventricular tachycardia is suspected
		• Skipped beats OR
		• Thumping beats OR
		• Short fluttering OR
		• Slow pounding AND
		• Normal ECG AND
		• No family history AND
		• No associated symptoms AND
		• Not provoked by exercise AND
		• No structural heart disease/HTN/Heart failure

DVLA regulations state that:

-For Group 1 entitlement, driving must cease if an arrhythmia has caused or is likely to cause incapacity. Driving may be permitted when the underlying cause has been identified and controlled for at least 4 weeks.

-For Group 2 entitlement, the driver is disqualified from driving if an arrhythmia has caused or is likely to cause incapacity. Driving may be permitted when the underlying cause has been identified and the arrhythmia is controlled for at least 3 months.

-People with certain occupations, for example those working at height or with potentially dangerous machinery, will need to stop work until a diagnosis is confirmed or the underlying condition is treated.

References

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National Institute for Health and Care Excellence (2021) *Atrial fibrillation: diagnosis and management.* Available at: https://www.nice.org.uk/guidance/ng196 (Accessed 08.03.23)

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Full version control record

Version:	3
Guidelines Lead(s):	Dr Iain Sim, Consultant Cardiologist and Cardiac Electrophysiologist
Contributor(s):	Dr Iain Sim, Dr Arvind Singhal, Dr Paresh Mehta, Dr Andrew Cox, SN Andrea Lavous
Lead Director / Chief of Service:	Dr Navin Chandra, Dr Sitara. Khan
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This guideline has been registered with the trust. However, clinical guidelines are guidelines only. The interpretation and application of clinical guidelines will remain the responsibility of the individual clinician. If in doubt contact a senior colleague or expert. Caution is advised when using guidelines after the review date. This guideline is for use in Frimley Health Trust hospitals only. Any use outside this location will not be supported by the Trust and will be at the risk of the individual using it.

Version Control Sheet

Version	Date	Guideline Lead(s)	Status	Comment
1	October 2011	Dr M Faircloth, Consultant Cardiologist	Final	Drugs and Therapeutics Committee, Date October 2011
2	June 2015	Dr M Faircloth, Consultant Cardiologist	Final	Reviewed, reissued unchanged, SN Andrea Lavous, June 2015
3	November 2026	Dr Iain Sim Consultant Cardiologist and Cardiac Electrophysiologist	Final	Cross site Clinical Governance Cardiology 24/06/2026 DTC: 11/11/2026

Related Documents

Document Type	Document Name