Arrhythmias - Full Clinical Guideline

Reference no.: CG-CARDIO/2018/013

1. Introduction

This clinical guideline applies to all adult patients with cardiac arrhythmias at Derby Teaching Hospitals NHS Foundation Trust

2. Aim and Purpose

To provide a clear and simple flowchart approach to decision making around treatment of cardiac arrhythmias

3. Keywords

Cardiology,
CCU,
arrhythmia,
ACS,
NSTEMI,
STEMI,
bradycardia,
tachycardia,
SVT,
VT,
broad complex tachycardia,
AF,
flutter,
heart block,
WPW

4. Clinical Guideline

(a) Sinus Bradycardia post-ACS

Moderate sinus bradycardia is common and benign. If the heart rate is persistently less than 40, and there are associated symptoms, treatment with ***Atropine*** 0.5-1mg (maximum of 3mg) IV is advised to increase the heart rate to 50-60 per minute.

(b) Sinus Tachycardia post-ACS

Persistent heart rates of greater than 120 after acute myocardial infarction worsen myocardial ischaemia, and therefore prognosis. Unless there is any contra-indication, such as pulmonary oedema or asthma, beta blockade (***Atenolol*** 50-100mg bd, ***Metoprolol*** 50-100mg bd, ***Bisporolol*** 2.5-10mg od or ***Timolol*** 5-10mg bd) should be considered. Note sinus tachycardia should prompt assessment for LVF, pain, sepsis etc.

(c) **Supraventricular (AVRT and AVNRT) Tachycardia**

Vagal stimulation should be tried using carotid sinus stimulation, and is sometimes helpful. ***Adenosine*** is very effective in SVT involving the AV node. It is given as a rapid IV bolus (3-12mg in incremental boluses) and has a short half-life, but may produce transient hypotension and flushing. ***Adenosine*** should be avoided in asthmatics. ***Verapamil*** (5-10mg slowly IV) is an alternative, but may be dangerous in patients who are already on Beta-blockers. If ***Adenosine*** and ***Verapamil*** are ineffective, particularly if the patient is symptomatic and hypotensive, DC cardioversion under GA should be performed (see Appendix 9). Intravenous ***Esmolol*** and ***Amiodarone*** (see Appendix 2 for dosages) may also convert SVT, but ***Amiodarone*** is more toxic.

See Appendix 2 for anti-arrhythmic drug therapy and Appendix 3 for arrhythmia management in the Wolff- Parkinson-White Syndrome.

(d) **Atrial Flutter**

Atrial flutter may be terminated by either ***Esmolol*** (40mg IV over 1 minute, followed by infusion of 4mg/min) or ***Amiodarone*** (300mg IV over 20 minutes). Flecainide should be avoided due to risk of 1:1 AV conduction resulting in very high rates (especially if not on AV node blocking medication such as a betablocker). Usually treatment dose ***LMWH*** should be given.

If the duration of atrial flutter is more than 48 hours the patient should be given, ***Anticoagulation*** (as per AF) should be commenced if the flutter persists, unless there are contra-indications. Standard rate control medication (including betablockers or rate limiting calcium channel blockers, often combined with digoxin) should be used – rate control can be difficult to achieve.

Some patients with recurrent atrial flutter maybe suitable for ablation and referral for electrophysiology should be considered.

(e) Atrial Fibrillation

(for out patient AF management refer to the GP shared guidelines section – available on Flo via link in Cardiology Guidelines)

(See Appendix 9 for cardioversion guidelines)

<u>Post- ACS</u>

β **Blockers or rate limiting calcium channel blockers**, combined with ***Digoxin*** if necessary, are the drugs of choice to slow the ventricular rate. Unless there is a contra-indication, the patient should be anti-coagulated with ***LMWH***. AF following myocardial infarction frequently converts spontaneously to sinus rhythm after 24-48 hours, but if it persists elective DC cardioversion should be considered.

<u>Acute AF (Clear onset <48 hours)</u>

Outside the context of acute myocardial infarction, cardioversion may be achieved "chemically" with ***Flecainide*** or electrically. ***Flecainide*** (2mg/kg up to a maximum of 150mg IV over 10 minutes) should be used, ***Flecainide*** should not be used if LV function is known to be impaired or there is clinical suspicion of heart failure (check CXR), or significant myocardial ischaemia or previous myocardial infarction (check ECG). It is most likely to be effective in AF of recent onset if there is no severe left atrial enlargement with little or no

underlying structural heart disease and no metabolic abnormalities. Cardioversion should be considered in patients presenting within 48 hours of onset of AF unless there is an underlying cause that requires treatment first such as sepsis or overt thyrotoxicosis.

Persistent AF (>48hrs or uncertain)

Patients presenting >48 hours should have rate control (with a β **blocker** or ***Diltiazem***/***Verapamil*** +/- ***Digoxin***) and anticoagulation for >3 weeks before elective DC cardioversion in selected cases. (See Cardioversion guidelines for cardioversion details).

In chronic AF (both rheumatic and non-rheumatic) the overall risk of embolic stroke is about 5% per year. This is reduced to 1.4% with ***anticoagulation***. All patients maintained in chronic AF should therefore be considered for long term anticoagulation **(*Warfarin or NOAC)*** unless there is a contra-indication. If on warfarin, ensure that the INR is within the 2.0-3.0 range. The recent AFFIRM study showed that the majority of strokes in AF patients occurred when the INR was <2.0.

The CHA_2DS_2-VASc is a scoring system which has been shown to predict embolic risk in AF patients, and appears to be superior to the previously used $CHADS_2$ system.

CHA_2DS_2-VASc		Score
C	Congestive heart failure	1
H	Hypertension	1
A	Age > 75 / >65	2/1
D	Diabetes	1
S	Stroke, TIA, embolus	2
Va	Vascular disease	1
S	Sex (female)	1

Stroke rate - per 100 patient years	
Low risk patients (score=0)	0.78
Intermediate risk (score=1)	2.01

If the score is zero or 1 (in a woman) no anticoagulation is indicated (except for prior to cardioversion). If the score is 1 (male), anticoagulation should be considered. If the score is 2 or more, anticoagulation should be offered to all but check HASBLED or ORBIT scores for modifiable bleeding risks that can be addressed. Aspirin is no longer indicated. Left atrial appendage closure should be considered if anticoagulation is contraindicated and stroke risk very high.

Patients with recurrent or paroxysmal AF are at risk of emboli, and anticoagulation should be considered. Prophylactic treatment with ***Sotalol*** (40-80mg bd) or ***Flecainide*** (50-100mg bd, usually combined with a standard betablocker) may be effective at preventing attacks. ***Amiodarone*** (200mg daily) is more effective but more toxic. ***Dronedarone*** (400 mg bd), is an alternative multichannel blocker to ***Amiodarone*** for maintaining sinus rhythm in patients with transient AF. It has fewer side effects than ***Amiodarone***, but is less effective and must be recommended by a cardiologist and prescribed in a shared care agreement ('Amber' drug) (see antiarrhythmic drugs).

An alternative approach, in patients without significant structural heart disease who have shown to respond safely to oral drug therapy in hospital, is the "Pill in the Pocket" self administration by the patient of a single oral dose of ***Flecainide*** (200-300mg) or ***Propafenone*** (450-600mg) out of hospital at the onset of symptoms. This has been shown

to be effective in 80-90% of patients, usually within one hour. There is a small risk (1-2%) of worsening arrhythmia requiring hospital admission.

Patients with paroxysmal AF who remain symptomatic and resistant to treatment should be considered for ablation. The AF focus is usually within one of the pulmonary veins. Electrophysiology with septal puncture and detailed electrical mapping is required. This is often a lengthy procedure (2-3 hours) and the current success rate for ablation is 75-85%. Complications (tamponade, stroke, arrhythmia recurrence and late pulmonary vein stenosis) occur in 6%. Mortality is low (0.5%) but not zero.

WPW (pre-excited) AF

AF due to the WPW syndrome is uncommon, but always consider in a young patient with a very rapid irregular broad complex tachycardia. ***Digoxin*** and ***Verapamil*** are contra-indicated in this situation and treatment should be with electrical cardioversion. IV ***Flecainide*** or ***Amiodarone***, (See Appendix 2 for dosage guidelines) are alternatives. The patient should stay for an inpatient ablation because of the risk of ventricular arrhythmias precipitated by high rates.

(f) Ventricular Ectopic Beats (VEBs) post ACS

Occasional VEBs are common and are almost always benign. Ventricular ectopic beats may degenerate into VT or VF and may be treated in the following situations after acute myocardial infarction.

i R on T VEBs
ii VEBs occurring in salvos
iii Multifocal VEBs (different QRS morphology)
iv Frequent VEBs (>10 per minute)

Ventricular arrhythmias may be due to hypokalaemia and will certainly be exacerbated by a low K^+, particularly in older patients on chronic diuretic therapy. Always check the electrolytes and give K^+ replacement if K^+ <4.5. Rarely, ventricular arrhythmias are due to hyperkalaemia, particularly in patients with renal failure who have taken K^+ conserving diuretics and ACE inhibitors. (See Appendix 4 for guidelines on K^+ replacement and management of hyperkalaemia).

Occasionally recurrent unresponsive ventricular arrhythmias are associated with magnesium depletion (which may be associated with diuretic therapy) and these patients may respond to ***magnesium*** sulphate 8mmol bolus over 5 minutes and 65mmol infusion over 24 hours. Aim to maintain Mg >0.9.

Refractory cases can be treated with a ***Lidocaine*** bolus of 100mg over 30 seconds, which may be repeated after 5 minutes. If ***Lidocaine*** bolus appears effective, follow with an infusion (4mg/min reducing to 2mg/min after 30 minutes). If this fails, ***Amiodarone*** may be tried (AVOID in long QT). ***Disopyramide*** is also effective, but is more toxic. All class I anti-arrhythmic agents are negatively inotropic and should be used with caution in cardiac failure.

(g) Ventricular Tachycardia post ACS

Rapid, broad complex tachycardia shortly after myocardial infarction, and in older patients with CCF, particularly if associated with haemodynamic changes, is almost certainly ventricular tachycardia.

If a patient is not distressed and is haemodyncamically stable, drug treatment with IV ***Lidocaine*** 100-200mg may be tried. Patients not responding to ***Lidocaine*** should be treated with IV ***Amiodarone*** (AVOID in long QT). If the patient is unstable (BP <100 systolic, cardiac failure, ongoing ischaemia or if poor LV function – EF <35%), do not give ***Lidocaine*** but cardiovert without delay under GA. These patients are at high risk of deterioration and VF.

If the patient has a pacemaker or ICD in-situ try overdrive pacing. Pace at a faster rate than the tachycardia (increase in 20 bpm steps) to "capture" the rhythm, then gradually slow the pacing rate to below the sinus rate when sinus rhythm should re-establish (contact the pacing clinic in normal hours).

If two anti-arrhythmic drugs have been tried without success, or if the patient is deteriorating, immediate cardioversion under GA should be performed. Consider ***Sotalol*** or ***Esmolol*** for recurrent ventricular tachycardia. Overdrive pacing can also be useful for recurrent VT. (See Appendix 7).Maintain K >4.5 and Mg >0.9.

Supraventricular tachycardia with aberrant conduction (i.e. BBB) is relatively uncommon but may mimic VT. A very irregular broad complex tachycardia is usually AF with BBB and in a young patient always consider the WPW syndrome.

Features supporting a diagnosis of VT with a broad complex tachycardia are:-

i Wide QRS (>140 ms)
ii Left axis (> -40^{o})
iii RSR in V1 with R1 > R2
iv AV dissociation (evidence of independent atrial activity)
v Capture or fusion beats
vi Intrinsicoid deflection duration >40ms (i.e. Onset to peak of R wave > 1 small square broad indicative of intramyocardial conduction)

Ventricular ectopics in a previous ECG with very similar QRS morphology to the complexes in the tachycardia are also suggestive of VT.

If the ECG is suggestive of SVT, diagnostic use of ***Adenosine*** will convert re-entry tachycardia and will slow (and therefore unmask) atrial flutter and atrial tachycardia, but will have no effect with VT. Rarely an atrial electrode (either oesophageal or a right atrial wire electrode) is required to confirm or exclude independent atrial activity.

Patients presenting with primary ventricular fibrillation or syncopal ventricular tachycardia (which has not occurred within 48 hours of an acute myocardial infarction) should be considered for Internal Cardiac Defibrillator (ICD) implantation, prior to discharge, and should be referred to an electrophysiologist at Glenfield or Nottingham City Hospital. Any patient with LVEF <35% and life expectancy > 1yr with reasonable quality of life should now be considered for an ICD. If the patient has had an acute MI, the echo should be carried out 6 weeks post infarct as the LV may improve. Patients should have a diagnostic coronary angiogram and can usually be referred as an outpatient to the electrophysiology service.

In patients with an ICD in situ, who are having recurrent bouts of VT and ICD shocks, consider a combination of a **β Blocker** and ***Amiodarone***, and also consider adding oral ***Mexiletine***. The ICD implantation centre should be contacted with a view to review by an electrophysiologist and consideration for an EPS and ablation.

The annual rate of late (out of hospital) sudden cardiac death after myocardial infarction, due to presumed ventricular fibrillation, is 1-2% PA, over the subsequent five years. Cardiac failure has been shown to be a major risk predictor, rather than myocardial infarction.

Current NICE guidelines (January 2014) for ICD implementation for prevention of sudden cardiac death (SCD) are as follows:

(a) Secondary Prevention of SCD

Patients presenting without treatable cause with:

(i) Cardiac arrest due to VT or VF (outside context of acute MI)
(ii) Spontaneous sustained VT causing syncope or significant haemodynamic compromise.
(iii) Sustained VT without syncope associated with EF<35%.

(b) Primary Prevention of SCD after Myocardial infarction
(i) LV dysfunction (EF<35%) after 6 weeks

(c) Primary Prevention of SCD
(i) LV dysfunction (EF<35%)
(ii) Certain patients with HCM, ARVC, long QT, Fallots, Brugada etc

Patients with anterior STEMIs should have a predischarge echo (to exclude thrombus) and this should be repeated at 6 weeks unless only mildly impaired. Other ACS patients should have an echo at 6 weeks (unless already known to have severe LV impairment). Other selected STEMIS may have an IP echo at the discretion of the Cardiologist.

(h) The Long QT Syndrome and "Torsade de Pointes" Tachycardia

Prolonged ventricular repolarisation may reduce cellular refractory time leading to electrical instability and a specific ventricular tachycardia "Torsade de Pointes". It is manifested by prolonged QT interval and may be associated with dizziness, syncope and sudden death.

Torsade de Pointes ("Twisting of the Points") is a specific type of polymorphic ventricular tachycardia associated with a prolonged QT interval. The ECG shows a continually changing axis, with periodic twisting of the points of the QRS complex around the isoelectric line and heart rates of 200-250. Each cycle is usually preceded by a late cycle atrial or ventricular ectopic, followed by a compensatory pause and then a sequence of 5-20 complexes of uniform morphology during the tachycardia. The tachycardia is usually non-sustained and repetitive but may degenerate into ventricular fibrillation.

The QT interval is measured from the onset of the QRS to the end of the T wave and should be corrected for heart rate (QT_c). This is done automatically on a page writer ECG. On a 12 lead ECG the longest QT interval is considered to be the true QT.

$$\text{Corrected QT interval } (QT_c) = \frac{\text{QT interval}}{\sqrt{\text{RR interval}}}$$

The upper limit of normal for QT_c is 0.44s in men, 0.46s in women and 0.44s in children. Most patients with long QT syndrome and "Torsade de Pointes" have a QT_c >0.50s and a prominent U wave. Women and the elderly are more vulnerable to the long QT syndrome.

The Long QT syndrome maybe congenital or acquired, but the commonest cause in adult hospital practice is drug induced.

Congenital:

1. Romano-Ward syndrome (autosomal dominant)
2. Jervell-Lange-Nielson syndrome, associated with nerve deafness (autosomal recessive)
3. Non-familial sporadic long QT syndrome
4. (there are now 14 identified LQT1-14)

Acquired:

1. Drugs

Antibiotics	***Erythromycin, Clindamycin, Ciprofloxacin***
Anti-arrhythmics	***Disopyramide, Amiodarone, Sotalol, Propafenone***
Anti-depressants	***Amitriptyline, Imipramine, Dosulepin, Citalopram***
Anti-psychotics	***Haloperidol, Clozapine, Lithium***
Anti-histamines	***Terfenadine, Astemizole*** (both discontinued)
Anti-malarials	***Chloroquine***
Others	***Tacrolimus, Indapamide, Methadone***

(This list should not be regarded as comprehensive as less commonly used drugs have not been listed and new cases continue to be described)

2. Myocardial disease — Ischaemia and myocarditis
3. Electrolyte abnormalities — Hypokalaemia, hypomagnesaemia
4. Bradycardia and heart block
5. Thyroid disease (hypo and hyperthyroidism)
6. Anorexia nervosa and starvation

Management of Torsade de Pointes tachycardia:

(1) Stop anti-arrhythmics, anti-depressants, anti-histamines and ***Erythromycin***. Check remaining drugs.
(2) IV ***Magnesium Sulphate*** 8mmol IV over 5 minutes, followed by 72mmol in 1 litre 5% dextrose over 24 hours.
(3) Correct hypokalaemia (if present).
(4) Consider temporary pacing (rate 90-100) if above ineffective.

(Avoid *Amiodarone* and *Sotalol* which will increase the QT interval and worsen the arrhythmia)

Management of Chronic Long QT Syndrome:

Patients with congenital long QT >0.5secs are at increased risk of symptoms by the age of 40.

1. (a) Asymptomatic patients with a positive family history of sudden death or complex ventricular arrhythmias on ECG monitoring, treat with β blockers increasing to the maximum tolerated dose (e.g. ***Atenolol*** 25mg up-titrated to 100mg daily). Some of these patients may benefit from ICD implantation and counselled to avoid drugs that prolong long QT.

2. (b) Symptomatic patients with syncope, documented VT or previous cardiac arrest may have an ICD in addition to β blockade.

(i) **Ventricular Fibrillation and Cardiac Arrest**

VF may follow warning complex VEBs or VT (especially polymorphic), but may also occur spontaneously following acute myocardial infarction. Immediate cardioversion must be performed (see Appendix 14 for cardiopulmonary resuscitation protocol). Other corrective management as per VT.

(j) **"Electrical Storm"**

This uncommon, but often fatal, complication of myocardial infarction is defined as multiple episodes of haemodynamically destabilizing VT or VF within a period of 24 hours, usually in the setting of an extensive recent myocardial infarction with LV dysfunction. These patients are usually unresponsive to ***Lidocaine*** or ***Mexiletine*** but may respond to a combination of IV ***Amiodarone*** and a β blocker (IV ***Propranolol*** 0.15mg/kg over 10 minutes, followed by 3.5mg IV 6 hourly or ***Esmolol*** 40mg IV over 1 minute followed by an infusion of 4mg/min). Some of these patients have ischaemia related ventricular irritability with severe underlying coronary disease, and CABG or angioplasty may be associated with improved long-term survival, but investigation and intervention is only feasible when the "electrical storm" has been calmed. Some patients with ischaemic electrical storm also settle on an intra-aortic balloon pump

2. Conduction Defects

(a) First Degree Block

No specific treatment is required.
If ***Digoxin*** toxicity is suspected, ***Digoxin*** therapy should be discontinued.

(b) Second Degree Block (Wenckebach)

This occurs quite frequently after inferior infarction, and does not commonly progress to complete heart block. No specific treatment is warranted. Outside the context of AMI, it rarely requires treatment as is often associated with high vagal tone. If syncope, pacing may be required.

(c) Second Degree Block (Mobitz Type II)

This is less common, but indicates much more serious damage to the conduction system. It may progresses suddenly to complete heart block.
Temporary pacemaker insertion should be considered in symptomatic patients. Outside the context of MI symptomatic second degree heart block is a class I indication for permanent pacing. Pacing may be considered in asymptomatic patients.

(d) Complete (Third Degree) Heart Block

This may occur due to right coronary artery occlusion in association with inferior infarction, when there is usually a ventricular rate of greater than 40 per minute, and a narrow QRS complex. Complete heart block is usually transient and prognosis is generally good.

Complete heart block associated with anterior infarction is usually due to extensive septal infarction with damage to all three bundles. There is usually a bradycardia of about 30 beats per minute with a wide QRS complex, and the prognosis is poor.

Temporary pacemaker insertion is recommended for all cases of complete heart block secondary to anterior myocardial infarction. Pacing may not be required in inferior myocardial infarction, if the patient is haemodynamically stable.

Outside the context of acute MI, permanent pacing is usually required. Recent evidence suggests that a high proportion of patients who have CHB in the context of rate limiting medication require pacing within 12 months, even if their conduction appears to recover after discontinuation of the drugs.

(e) Bifascicular Block (RBBB + Left Anterior Hemiblock or LBBB)

Progress to complete heart block is uncommon and no specific management is warranted. The presence of LBBB usually makes ECG diagnosis of infarction or ischaemia difficult and clinical judgment or comparison with old ECGs (if available) is crucial.

(f) "Trifascicular Block"

This is a misnomer but is taken to indicate extensive conduction system disease. Most commonly this manifests as RBBB + LAD + 1st degree AV block (PR>200ms) and has a high rate of progression to CHB. Therefore presyncope or syncope is an indication for permanent pacing.

(g) Treatment of Heart block/Temporary pacing

Guidance for assessment and treatment of bradycardias is summarised in the attached summary clinical guideline. In heart block, atropine can be tried but is rarely successful and isoprenaline or salbutamol are more often successful in restoring conduction.

Defibrillators with external pacing facility are present throughout the Trust – your resuscitation trolley will have a sign saying where the nearest one is

If a temporary pacing wire is required out of hours, contact the on-call Consultant Cardiologist via switchboard

5. Documentation Controls

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