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# Cardiac arrhythmias in coronary heart disease

A national clinical guideline



February 2007

## KEY TO EVIDENCE STATEMENTS AND GRADES OF RECOMMENDATIONS

### LEVELS OF EVIDENCE

| | |
|---|---|
| $1^{++}$ | High quality meta-analyses, systematic reviews of randomised controlled trials (RCTs), or RCTs with a very low risk of bias |
| $1^{+}$ | Well conducted meta-analyses, systematic reviews of RCTs, or RCTs with a low risk of bias |
| $1^{-}$ | Meta-analyses, systematic reviews of RCTs, or RCTs with a high risk of bias |
| $2^{++}$ | High quality systematic reviews of case control or cohort studies<br>High quality case control or cohort studies with a very low risk of confounding or bias and a high probability that the relationship is causal |
| $2^{+}$ | Well conducted case control or cohort studies with a low risk of confounding or bias and a moderate probability that the relationship is causal |
| $2^{-}$ | Case control or cohort studies with a high risk of confounding or bias and a significant risk that the relationship is not causal |
| 3 | Non-analytic studies, eg case reports, case series |
| 4 | Expert opinion |

GRADES OF RECOMMENDATION

*Note: The grade of recommendation relates to the strength of the evidence on which the recommendation is based. It does not reflect the clinical importance of the recommendation.*

| | |
|---|---|
| **A** | At least one meta-analysis, systematic review of RCTs, or RCT rated as $1^{++}$ and directly applicable to the target population*; or*<br>A body of evidence consisting principally of studies rated as $1^{+}$, directly applicable to the target population, and demonstrating overall consistency of results |
| **B** | A body of evidence including studies rated as $2^{++}$, directly applicable to the target population, and demonstrating overall consistency of results*; or*<br>Extrapolated evidence from studies rated as $1^{++}$ or $1^{+}$ |
| **C** | A body of evidence including studies rated as $2^{+}$, directly applicable to the target population and demonstrating overall consistency of results*; or*<br>Extrapolated evidence from studies rated as $2^{++}$ |
| **D** | Evidence level 3 or 4*; or*<br>Extrapolated evidence from studies rated as $2^{+}$ |

GOOD PRACTICE POINTS

| | |
|---|---|
| ☑ | Recommended best practice based on the clinical experience of the guideline development group. |

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This guideline is dedicated to the memory of
Malcolm John McDonald


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**Scottish Intercollegiate Guidelines Network**
**28 Thistle Street, Edinburgh EH2 1EN**

**www.sign.ac.uk**

# 1 Introduction

## 1.1 REMIT OF THE GUIDELINE

This guideline provides evidence based recommendations for the management of cardiac arrest and the arrhythmias associated with acute coronary syndromes, chronic coronary heart disease and cardiac surgery. It excludes arrhythmias not associated with coronary heart disease such as supraventricular tachycardias associated with accessory pathways or dual atrioventricular (AV) nodal physiology, arrhythmias caused by inherited ion channel disorders (eg long QT syndrome, Brugada syndrome) and arrhythmias associated with non-ischaemic cardiomyopathies. The evidence base in some areas (eg management of cardiac arrest and atrial fibrillation) does not accurately distinguish between patients whose arrhythmia has an ischaemic or non-ischaemic aetiology but wherever possible, the recommendations made are specific to coronary heart disease.

The rhythm management of atrial fibrillation (AF), a common manifestation of acute and chronic coronary heart disease, is outlined in this guideline.

Recommendations on antithrombotic prophylaxis for atrial fibrillation are contained in SIGN guideline 36 antithrombotic therapy.[1]

### 1.1.1 PATIENT VERSION

A patient version of this guideline is available from the SIGN website, **www.sign.ac.uk**

### 1.1.2 ADDITIONAL ADVICE TO NHSSCOTLAND FROM NHS QUALITY IMPROVEMENT SCOTLAND AND THE SCOTTISH MEDICINES CONSORTIUM

NHS QIS processes multiple technology appraisals (MTAs) for NHSScotland that have been produced by the National Institute for Health and Clinical Excellence (NICE) in England and Wales. The Scottish Medicines Consortium (SMC) provides advice to NHS Boards and their Area Drug and Therapeutics Committees about the status of all newly licensed medicines and any major new indications for established products. SMC advice and NHS QIS validated NICE MTAs relevant to this guideline are summarised in the section on implementation.

## 1.2 STATEMENT OF INTENT

This guideline is not intended to be construed or to serve as a standard of care. Standards of care are determined on the basis of all clinical data available for an individual case and are subject to change as scientific knowledge and technology advance and patterns of care evolve. Adherence to guideline recommendations will not ensure a successful outcome in every case, nor should they be construed as including all proper methods of care or excluding other acceptable methods of care aimed at the same results. The ultimate judgement must be made by the appropriate healthcare professional(s) responsible for clinical decisions regarding a particular clinical procedure or treatment plan. This judgement should only be arrived at following discussion of the options with the patient, covering the diagnostic and treatment choices available. It is advised, however, that significant departures from the national guideline or any local guidelines derived from it should be fully documented in the patient's case notes at the time the relevant decision is taken.

# 2 Arrhythmias associated with cardiac arrest

Management of cardiac arrest in the UK follows the Resuscitation Council (UK) guidelines.[2] These are based on the International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations (CoSTR).[3] Evidence presented in this section is based largely on the evidence evaluation worksheets from the CoSTR project which are referenced where appropriate.

## 2.1 PRIMARY PREVENTION OF SUDDEN CARDIAC DEATH

Coronary heart disease is the cause of sudden cardiac death (SCD) in approximately 70% of cases.[4] SCD occurs as a primary event in patients without previously recognised coronary heart disease (CHD), as well as in those with known CHD, and shares the same risk factors. These include diabetes mellitus, hypertension and left ventricular hypertrophy, hyperlipidaemia, dietary factors, excessive alcohol consumption, physical inactivity and smoking.[4] 4

Asymptomatic individuals with multiple risk factors are at highest risk for primary SCD.[4] There is no randomised controlled trial (RCT) evidence that interventions in asymptomatic individuals prevent primary SCD. Clinical trials of lipid lowering and antihypertensive drugs in primary prevention of CHD have had insufficient statistical power to show reduction in SCD as a separate end point. A case control study found that SCD was decreased in those whose leisure activity was gardening and/or walking for more than 60 minutes per week.[5] 4 2+

**D** **Efforts to prevent sudden cardiac death should include:**

- **risk factor intervention in those individuals who are at high risk for coronary heart disease**
- **health promotion measures and encouragement of moderate intensity physical activity in the general population.**

See also SIGN guideline 97 on risk estimation and the prevention of cardiovascular disease.[6]

## 2.2 BYSTANDER CARDIOPULMONARY RESUSCITATION

Bystander cardiopulmonary resuscitation (CPR) is associated with increased survival in out-of-hospital cardiac arrest. The likelihood of survival to hospital discharge approximately doubles when bystanders initiate CPR prior to the arrival of the emergency services.[7] A systematic review incorporating a range of quality of life (QoL) measurement tools, time points and comparison groups, found that the provision of CPR, defibrillation, and emergency cardiovascular care to victims of cardiac arrest in the general population, both for out-of-hospital and in-hospital patients, is worthwhile in terms of quality of life for survivors.[8] CPR training positively influences laypersons' willingness to perform CPR.[9] 2+

**C** **The number of lay people trained to initiate CPR in out-of-hospital cardiac arrest should be increased.**

Targeted training of lay people selected by occupation, low training costs, or having high-risk household companions is substantially more cost effective than training unselected lay people.[10]

**D** **Lay people identified as having a high probability of witnessing a cardiac arrest should be offered CPR training.**

☑ CPR should be performed in accordance with the Resuscitation Council (UK) guidelines.

☑ All healthcare workers who have direct patient contact should have annual refresher training in cardiopulmonary resuscitation.

The delivery of CPR training programmes in schools is an effective way to promote widespread knowledge and retention of resuscitation skills.[11] | 4

D **CPR should be taught as part of the school curriculum.**

## 2.3 DEFIBRILLATION

Defibrillation is the definitive intervention in ventricular fibrillation (VF) or pulseless ventricular tachycardia (VT) and is the most important determinant of survival in cardiac arrest. Survival to hospital discharge is inversely related to the time interval between the onset of VF and the delivery of the first shock. A one minute delay in defibrillation is associated with a reduction in odds of survival of up to 21%.[12] However, in adult out-of-hospital cardiac arrest with ventricular fibrillation and response time greater than five minutes, a period of two minutes of CPR before attempting defibrillation may improve return of spontaneous circulation (ROSC) and survival to hospital discharge.[13] | 2++

☑ Defibrillation should be administered in accordance with the Resuscitation Council (UK) guidelines.

B **Defibrillation in patients with VF or pulseless VT should be administered without delay for witnessed cardiac arrests and immediately following two minutes of CPR for unwitnessed out-of-hospital cardiac arrests.**

C **Prompt defibrillation should be available throughout all healthcare facilities.**

C **All healthcare workers trained in CPR should also be trained, equipped, authorised and encouraged to perform defibrillation.**

Defibrillators delivering biphasic waveforms require lower energy shocks to terminate VF than those delivering monophasic waveforms and result in less myocardial damage. Studies in patients do not show consistent differences between the type of waveform used during defibrillation and ROSC or survival to hospital discharge after cardiac arrest.[14] | 1+

### 2.3.1 AUTOMATED EXTERNAL DEFIBRILLATORS

Automated external defibrillators (AEDs) are devices which guide first responders when assessing whether defibrillation is indicated to resuscitate a collapsed patient. AEDs provide spoken instructions to the responder on when and how to deliver the defibrillation shock.

Use of AEDs by trained first responders dispatched by the emergency medical services has been shown to be clinically effective.[12,15-18] | 1+ 2+

It is cost effective in urban areas and where vehicle response times are minimised within the emergency medical services system.[19,20]

A **Automated external defibrillators should be used by trained first responders, with their use integrated within the emergency medical services system.**

Use of AEDs in public areas (public access defibrillators) has been shown to be clinically effective in locations associated with a high probability of a cardiac arrest event.[17,21,22] | 2+ 1+

B **Automated external defibrillators should be sited in locations which have a high probability of a cardiac arrest event.**

The cost effectiveness of public access defibrillators has not been demonstrated.[23] The major factors influencing cost effectiveness are frequency of cardiac arrest, consistent rapid response times by the responders and the cost of the equipment and associated training.[24,25]

No evidence was identified on the clinical or cost effectiveness of AEDs in rural areas.

There are no studies which provide evidence that provision of AEDs to the families of patients with previous myocardial infarction (MI) or cardiac arrest reduces the patients' mortality or improves their quality of life.[17] An ongoing major international trial, the home AED trial (HAT), is addressing this evidence gap.

## 2.4 ADJUNCTIVE THERAPIES IN THE PERI-ARREST PERIOD

### 2.4.1 REFRACTORY VT/VF

In VT/VF, pressor agents are administered to increase coronary perfusion pressure and increase myocardial oxygen delivery. Despite the widespread use of adrenaline/epinephrine during resuscitation and several studies involving vasopressin, there is no placebo-controlled study that shows that the routine use of any vasopressor at any stage during human cardiac arrest increases survival to hospital discharge.[3] | 4

In a meta-analysis, high-dose adrenaline/epinephrine (5–15 mg) produced higher ROSC but a trend towards lower survival to hospital discharge without neurological damage compared to standard dose (1 mg).[26] | 1+

Vasopressin acts on specific non-adrenergic receptors and theoretically may not produce the adverse increase in myocardial oxygen consumption seen with adrenaline/epinephrine. In a meta-analysis of the use of vasopressin with cardiac arrest there was no advantage over adrenaline/epinephrine in ROSC, survival to hospital admission or discharge from hospital.[27] | 1+

Amiodarone is effective in increasing ROSC and survival to hospital admission when given in refractory VT/VF, but there is no evidence that it increases survival to discharge from hospital.[28] | 1+

**D** **Intravenous adrenaline/epinephrine should be used for the management of patients with refractory VT/VF.**

**A** **Intravenous amiodarone should be considered for the management of refractory VT/VF.**

☑ Adjuvant therapies should be administered in accordance with the Resuscitation Council (UK) guidelines.

### 2.4.2 SUSTAINED VT (NO CARDIAC ARREST)

For patients with sustained VT who are haemodynamically unstable, electrical cardioversion is the immediate treatment of choice.[3] | 4

No evidence has been found comparing the efficacy of electrical cardioversion versus anti-arrhythmic drugs (AADs) in patients with sustained VT.

Intravenous amiodarone, procainamide or sotalol are effective in terminating haemodynamically stable VT.[29,30] Intravenous AADs can produce hypotension.[29] | 3

No studies have been sufficiently powered to provide mortality data or compare benefits and harms of AADs in a systematic manner.

**D** **Intravenous amiodarone, procainamide or sotalol should be used in the management of patients with haemodynamically stable VT.**

☑ Intravenous drug therapy for ventricular tachycardia should ideally be given under expert guidance. If the first IV drug fails to restore sinus rhythm, electrical cardioversion or anti-tachycardia pacing should be considered.

Recurrent polymorphic VT (torsades de pointes) is usually associated with QT interval prolongation, and can be secondary to hypokalaemia, hypomagnesaemia and certain QT prolonging drugs. Treatment is by withdrawal of QT interval prolonging drugs and administration of intravenous magnesium. Hypokalaemia should be corrected where present, and bradycardia treated by temporary pacing or isoprenaline infusion.[3,31] | 4

**D** **Patients with polymorphic VT should be treated with intravenous magnesium. QT interval prolonging drugs, if prescribed, should be withdrawn. If present, hypokalaemia should be corrected by potassium infusion and bradycardia by temporary pacing or isoprenaline infusion.**

### 2.4.3 ASYSTOLE AND PULSELESS ELECTRICAL ACTIVITY

Asystole accounts for 20-40%, and pulseless electrical activity (PEA) for up to 10%, of all cardiac arrests. Patients with cardiac arrests due to asystole or PEA occurring outside hospital have a poor outlook with less than 4% survival to hospital discharge. Within hospital 10% of patients are resuscitated.[32] | 2+

Adrenaline/epinephrine is the pressor agent routinely used to increase coronary perfusion pressure in cardiac arrest but there is no evidence that it improves survival. Its efficacy is impaired by the prevailing acidosis and hypoxia in cardiac arrest but there is no conclusive evidence that vasopressin is superior in the treatment of asystole or PEA.[3,27] | 4 1+

**D** **Patients with cardiac arrest secondary to asystole or pulseless electrical activity should receive intravenous adrenaline/epinephrine.**

### 2.4.4 BRADYCARDIA/SINOATRIAL DYSFUNCTION/HEART BLOCK

Intravenous atropine improves heart rate and symptoms and signs in patients with symptomatic bradycardia in both the hospital and pre-hospital setting.[33] | 2+

Where transcutaneous pacing is started within five minutes of onset of bradycardia, resuscitation is successful in 75% of patients.[33] | 3

Secondline drugs for symptomatic bradycardia include adrenaline/epinephrine, dopamine, isoprenaline and aminophylline.[3] | 4

Glucagon is only likely to be useful in drug-induced bradycardias eg beta blocker and calcium channel blocker overdose.[34] | 3

**C** **Atropine should be used in the treatment of patients with symptomatic bradycardia.**

**D** **Temporary transcutaneous pacing should be initiated quickly in patients not responding to atropine.**

**D** **When atropine or transcutaneous pacing is ineffective consider adrenaline/epinephrine, dopamine, isoprenaline or aminophylline infusions before transvenous pacing is instituted.**

☑ Transcutaneous pacing should be followed by transvenous pacing if bradycardia persists.

# 3 Arrhythmias associated with acute coronary syndromes

## 3.1 ATRIAL FIBRILLATION

In patients with acute MI treated with thrombolytic therapy, new atrial fibrillation occurs in 7-10% of cases. The majority (70-100%) of these patients will be in sinus rhythm by the time of hospital discharge regardless of treatment strategy.[35]

In acute MI, atrial fibrillation occurs more commonly in those who are older, have greater haemodynamic disturbance (eg higher Killip class) and have left ventricular (LV) dysfunction. Atrial fibrillation is an independent risk factor for mortality. Stroke rates are increased in patients with AF.[36]

Recommendations on antithrombotic prophylaxis for atrial fibrillation are contained in SIGN guideline 36 on antithrombotic therapy.[1]

### 3.1.1 PROPHYLAXIS

Recurrence of AF is observed in around 20% of cases.[37] There is no evidence to support the use of prophylactic anti-arrhythmic therapy for patients with acute coronary syndrome or acute MI who have had AF but who have returned to sinus rhythm. Drugs which are used for their proven benefits on mortality, including beta blockers and angiotensin converting enzyme (ACE) inhibitors, may also reduce the incidence of AF in patients with acute MI.[38,39] 1+

### 3.1.2 ANTI-ARRHYTHMIC DRUG THERAPY/CARDIOVERSION

There is limited evidence on the use of amiodarone in patients with AF following acute MI, with one small randomised study which showed no difference in restoration of sinus rhythm compared to digoxin,[40] and observational studies which have shown no mortality benefit with amiodarone compared to no anti-arrhythmic treatment.[35,41] 1+ 2+

The safety and efficacy of class 1C drugs *(see Annex 1)* for the treatment of AF in acute MI have not been studied in large scale trials. Flecainide was associated with increased mortality in patients with frequent ventricular premature beats and LV dysfunction following MI.[42] 1++

Propafenone has not been studied in the context of acute MI.

**B** **Class 1C anti-arrhythmic drugs should not be used in patients with AF in the setting of acute MI.**

No good quality studies have assessed effectiveness of synchronised direct current (DC) cardioversion in this patient population.

Recommendations for treatment are based on previous consensus guidelines and expert opinion.[43,44] These guidelines suggest that underlying causes and aggravating features should be corrected (eg heart failure, hypokalaemia and hypoxia). Further treatment measures then depend on the clinical condition of the patient with regard to haemodynamic instability (hypotension, heart failure) and ongoing ischaemia. 4

**D** **Patients with AF and haemodynamic compromise should have urgent synchronised DC cardioversion or be considered for anti-arrhythmic and rate-limiting therapy using:**

- **intravenous amiodarone**

  **or**
- **digoxin, particularly in presence of severe LV systolic dysfunction with heart failure.**

D **Patients with AF with a rapid ventricular response, without haemodynamic compromise but with continuing ischaemia should be treated with one of:**

- **intravenous beta blockade, in the absence of contraindications**
- **intravenous verapamil where there are contraindications to beta blockade and there is no LV systolic dysfunction**
- **synchronised DC cardioversion.**

D **Patients with AF without haemodynamic compromise or ischaemia should be treated with rate-limiting therapy, preferably a beta blocker, and be considered for chemical cardioversion with amiodarone or DC cardioversion.**

☑ Where indicated, cardioversion should be performed under short-acting general anaesthesia or conscious sedation.

## 3.2 CONDUCTION DISTURBANCES AND BRADYCARDIA

There are no published randomised trials comparing different strategies of managing conduction disturbances after acute MI or acute coronary syndrome. All recommendations are based on previous consensus guidelines and expert opinion based on case series, largely from the pre-reperfusion era.[36]

Sinus bradycardia occurs in 40 to 70% of patients post-MI, either spontaneously or in response to beta blocker therapy.[45] Where sinus bradycardia is asymptomatic and haemodynamically well tolerated no action is required. Symptomatic bradycardia usually responds to atropine and the withdrawal of rate slowing agents. Heart block occurs in 5-10% of patients with ST elevation MI (STEMI), and intraventricular conduction disturbances in up to 20%.[36] 4

Temporary pacing is used to treat symptomatic bradycardia. This can be achieved either transcutaneously or transvenously. Temporary transvenous pacing is used for symptomatic or prognostically significant bradycardias. Standby transcutaneous pacing is suitable for less threatening rhythms. The requirement for temporary pacing is not in itself an indication for permanent pacing. Permanent pacemaker implantation can be deferred if the peri-infarction AV block is expected to resolve.[36] 4

D **In patients with symptomatic bradycardia/conduction disturbance, concurrent therapies which predispose to bradycardia** *(eg beta blockers, digoxin, verapamil)* **should be discontinued.**

D **Isolated first degree heart block/Mobitz type I second degree heart block require no treatment.**

D **Transvenous temporary pacing should be considered for patients with:**

- **sinus bradycardia** *(heart rate < 40 beats per minute)* **associated with symptoms and unresponsive to atropine**
- **alternating left and right bundle branch block**
- **Mobitz type II AV block with new bundle branch block**
- **third degree AV block in inferior MI, if unresponsive to atropine and haemodynamically compromised, and in all cases of anterior MI**
- **ventricular standstill.**

**Transcutaneous pacing should be available to all patients with other atrioventricular and intraventricular conduction disturbances.**

D **Permanent pacing is indicated for patients with persistent Mobitz type II second degree block, or persistent third degree AV block.**

D **Permanent pacing should be considered for patients who have had transient second degree or third degree AV block with associated bundle branch block.**

☑ All patients requiring a permanent pacemaker should be evaluated for an implantable cardioverter defibrillator and/or biventricular pacing.

## 3.3 VENTRICULAR ARRHYTHMIAS

### 3.3.1 VENTRICULAR ARRHYTHMIAS AND ACUTE MI

Sustained ventricular arrhythmias, VT and/or VF, occur in up to 20% of patients with acute coronary syndromes. In hospitalised patients with STEMI, the most common sustained ventricular arrhythmia is primary VF, which occurs in 3-5% of patients within the first few hours following onset of infarction (75% of these within the first hour).[46,47]

Early (< 48 hours) post infarction primary VF is associated with increased in-hospital mortality, but those who survive to hospital discharge have a similar outcome to patients without primary VF. Late VF (> 48 hours) is less common (1-2%) and is associated with increased short and long term mortality.[48,49] Monomorphic VT (early or late) is associated with increased short term mortality, to a lesser degree than VF, but is also associated with an increase in long term mortality. Patients who have had both VT and VF have the worst short and long term mortality (eg in one study of patients with STEMI, 30 day mortality was 31% for patients with VF, 24% for patients with VT, 44% for patients with both and 6% for patients with neither).[49] The extent to which recurrent arrhythmia rather than heart failure or reinfarction contributes to the increased mortality in patients with VT and/or VF is not known. Secondary VF, in patients with heart failure or shock, is associated with high in-hospital mortality (up to 50%).[50] 3

There are no randomised studies of therapies to improve outcome after early VT/VF.

The incidence of primary VF in acute MI has not decreased in the last two decades, but there is evidence that modern medical management can reduce the subsequent mortality.[51] 3

The defibrillator in acute myocardial infarction trial (DINAMIT) of early (6-40 days) implantable cardioverter defibrillator (ICD) implantation after acute MI showed no benefit, but patients with VT/VF > 48 hours after onset of MI were excluded.[52] Expert opinion concludes that such patients should be assessed for revascularisation and/or ICD implantation.[36] 1+ 4

The indications for ICD implantation are discussed in section 4.2.2

The incidence of VT/VF is less in non-STEMI patients (about 2%; median time 78 hours) but is also associated with increased short and long term mortality.[53] 3

C **Patients who have primary VF should be recognised as being at increased risk during their hospital stay, and medical therapy should be optimised.**

D **Patients who have monomorphic VT following acute MI, or VF greater than 48 hours after infarction, should be recognised as being at increased short and long term risk and should be considered for revascularisation and ICD.**

The emergency treatment of sustained ventricular arrhythmias associated with acute coronary syndromes is discussed in sections 2.3 and 2.4.

### 3.3.2 PREVENTION OF VENTRICULAR ARRHYTHMIAS AND SUDDEN DEATH

Although there is a large volume of evidence on the benefits of drugs on survival post-MI, the primary outcome is usually total mortality, with sudden cardiac death as a secondary outcome or sub-analysis.

The main evidence of benefit is for treatments which reduce total mortality, in part by reducing sudden death, in particular thrombolysis, beta blockade, ACE inhibitors and statins.[36,38,43,54-56] Thrombolytic therapy may be associated with an early increased incidence of VF, but this is offset by the subsequent benefits of reperfusion, including reduction in sudden death.[4] Studies of intravenous beta blockers showed reduction in the incidence of early VF, but evidence from the thrombolytic era has indicated no additional total mortality benefit of intravenous compared to oral administration.[36,38,43,55,57] 1++

***Anti-arrhythmic drugs***

Anti-arrhythmic drugs (lidocaine, amiodarone) reduce arrhythmic death but with no, or minor, overall benefit on total mortality. Class 1C drugs have been shown to be associated with adverse outcomes following MI.[4,36,43,58,59] 1++

**A** **Routine use of anti-arrhythmic drugs is not recommended following MI.**

***Omega-3 fatty acid supplements***

There is conflicting evidence that omega-3 fatty acid supplements reduce the relative risk of sudden death, as there is for total or cardiovascular mortality (*see SIGN guideline 97 on risk estimation and the prevention of cardiovascular disease*).[6] Omega-3 fatty acid supplements reduced the relative risk of sudden death by 26% and total mortality by 14% compared to placebo in a single open-label study of patients with recent MI. The absolute risk reduction in sudden death was 0.7% (2.2% vs 2.9%).[60] A Cochrane meta-analysis identified four additional studies reporting on the risk of sudden death following the consumption of fish oil fatty acids or capsules.[61] The overall relative risk was 0.85 (95% confidence interval, 0.49 to 1.48). There was a marked heterogeneity and when all studies at medium or high risk of bias were excluded, one study of 551 patients undergoing angioplasty remained. One sudden death was observed in the placebo and none in the omega-3 supplemented group.[62] The three recent studies of omega-3 capsules in patients with implanted cardioverter-defibrillators also failed to show a consistent effect on time to first discharge (from a 33% decrease to 28% increase).[63-65] The value of this therapy to prevent sudden death in patients with coronary heart disease is uncertain. 1+

***Aldosterone receptor antagonists***

Eplerenone, an aldosterone receptor antagonist, showed a 21% relative risk reduction in sudden death and a 15% relative risk reduction in total mortality compared to placebo in a single study of patients with acute MI, LV dysfunction (left ventricular ejection fraction, LVEF $\leq$0.40) and heart failure or diabetes mellitus. There was a small excess of serious hyperkalaemia (serum potassium $>$6.0 mmol/l) with eplerenone (5.5% vs 3.9% with placebo).[66] 1+

**B** **Patients who have suffered a recent myocardial infarction and with LVEF $\leq$0.40 and either diabetes or clinical signs of heart failure should receive eplerenone unless contraindicated by the presence of renal impairment or high potassium levels.**

### 3.3.3 ASSESSMENT OF RISK OF SUDDEN DEATH

Following acute MI, sudden death is a continuing cause of mortality (7-10% at two years; up to 50% of total mortality), with the greatest risk being in the first 30 days (1.4% per month), declining during follow up (0.14% per month after two years).[4,36,43,67] The risk is associated with LV dysfunction, but sudden death can also occur in patients with preserved LV function post-MI. LV dysfunction is also a risk factor for non-sudden cardiac death.[67] 2-

There have been many studies of variables that may identify patients at risk of sudden death post-MI. This risk may be related to the arrhythmia substrate (LV dysfunction; late potentials on signal-averaged electrocardiogram (ECG); inducibility at electrophysiology study; microvolt T-wave alternans), the occurrence of triggers (ventricular premature beats, non-sustained VT) and autonomic dysfunction (heart rate variability; baroreceptor sensitivity; heart rate turbulence). Such investigations have relatively low sensitivity (<50%), low positive predictive accuracy (<30%) but high negative predictive accuracy (>90%).[4,36,68] The predictive value can be improved by combining investigations but this is at the expense of sensitivity. Their predictive value has been reduced by the decrease in incidence of sudden death with modern management post-MI.[67,69,70] Many of these investigations are specialised and not widely available. 2+ 3

There have only been a few studies in which these investigations have been used to guide therapy following acute MI. The DINAMIT study of patients with reduced LV function and abnormal cardiac autonomic function post-MI (6-40 days) did not show benefit of ICD implantation.[52] 1+

In the absence of proven benefit, the cost of these specialist investigations cannot yet be justified.

C **LV function should be assessed in all patients with acute MI during the index admission.**

C **Non-invasive assessment of the risk of ventricular arrhythmias may be considered but is not routinely recommended.**

C **Invasive electrophysiological studies are not routinely recommended for all patients post-MI.**

# 4 Arrhythmias associated with chronic coronary heart disease/left ventricular dysfunction

## 4.1 ATRIAL FIBRILLATION

### 4.1.1 INTRODUCTION

Atrial fibrillation is a significant burden on health and is associated with a substantially increased risk of stroke and sudden death.[71] A large North American study estimated that the prevalence of AF in the population aged over 20 years was 0.95%.[71] Assuming a similar rate in Scotland, around 40,000 patients had AF in 2005. The prevalence of AF rises markedly with advancing age, and is around 6% in those aged over 80 years.[44] The total burden of AF is set to rise as the population ages.

Although AF is a common complication of ischaemic heart disease, most of the studies of the management of AF have also encompassed patients with AF from other causes, including valvular heart disease and hypertension. The evidence base for treatment of patients with AF caused by ischaemic heart disease is diluted by the inclusion of patients with these other disease processes.

Factors associated with predisposition to atrial fibrillation include hypertension, left ventricular hypertrophy or dysfunction and heart failure. Several randomised controlled trials of beta-adrenoceptor antagonists or inhibitors of the renin-angiotensin-aldosterone system in patients with these conditions have reported reductions in the incidence of atrial fibrillation as a secondary end point, or in retrospective analysis.[38,72] Meta-analyses of the effects of angiotensin converting enzyme inhibitors or angiotensin receptor blockers have reported relative risk reductions of around 40% for the incidence of atrial fibrillation in patients with chronic heart failure, compared with a non-significant reduction of 27% in post-infarction studies.[72,73] Since treatment with beta-adrenoceptor antagonists and inhibitors of the renin-angiotensin-aldosterone system is already indicated for reduction in mortality in patients post-infarction or with chronic left ventricular dysfunction or heart failure, there is no need for a specific recommendation with respect to prevention of atrial fibrillation.

Recommendations on antithrombotic prophylaxis for atrial fibrillation are contained in SIGN guideline 36 on antithrombotic therapy.[1]

### 4.1.2 ANTI-ARRHYTHMIC DRUGS

Both amiodarone and sotalol are effective in preventing AF recurrence.[74-78] Evidence is limited by lack of specific reporting of coronary heart disease subgroups within trials. In a large (n = 665) randomised, double blind trial, amiodarone and sotalol were superior to placebo in restoring sinus rhythm, and in preventing recurrence of AF. Amiodarone and sotalol were equally effective in restoring sinus rhythm, but amiodarone was superior to sotalol in preventing AF recurrence. A pre-specified subgroup analysis of patients with ischaemic heart disease revealed no significant difference in efficacy between amiodarone and sotalol.[74] Amiodarone use may be associated with serious non-cardiac side effects including pneumonitis, thyroid disorders, liver dysfunction, photosensitivity and warfarin interaction.[79-81] These side effects are related to the dose and duration of exposure to the drug. Class 1C drugs (flecainide, propafenone) should not be used in patients with ischaemic heart disease.[42,44,82]

1+
4

A **Amiodarone or sotalol treatment should be considered where prevention of atrial fibrillation recurrence is required on symptomatic grounds.**

☑
- Patients with arrhythmias successfully controlled on amiodarone should have the dose titrated down to the lowest effective level.
- Patients taking amiodarone should have thyroid and liver function measured at baseline and at six monthly intervals. A baseline set of lung function tests should be performed (including transfer factor of carbon monoxide; DLCO).
- Patients with new or increasing cough or breathlessness during amiodarone therapy should be promptly referred for respiratory evaluation.
- Patients receiving amiodarone therapy should be provided with information on potential adverse effects.

### 4.1.3 RATE VERSUS RHYTHM CONTROL

In RCTs of patients with well tolerated AF, rate control is superior to rhythm control in terms of morbidity and avoidance of hospitalisation. There is no difference between the two strategies in the incidence of thromboembolism, incident heart failure, or mortality.[83-87] 1+ 1++

**A** **Rate control is the recommended strategy for management of patients with well tolerated atrial fibrillation.**

☑ Patients who are haemodynamically compromised, have myocardial ischaemia or are severely symptomatic as a result of AF with a rapid ventricular response should be treated promptly by electrical cardioversion.

☑ Patients with AF who remain severely symptomatic despite adequate rate control should be considered for rhythm control.

### 4.1.4 PHARMACOLOGICAL THERAPIES FOR RATE CONTROL

Although there have been few long term studies, a systematic review of multiple small-scale trials over periods of up to four weeks demonstrated that beta blockers, calcium channel blockers (verapamil or diltiazem), digoxin and amiodarone are all capable of controlling ventricular rate in AF.[88] 1+

There is no strong evidence to show superiority of any individual rate control drug or class of drugs over another. The choice of agent usually depends on clinical factors including cardiac indications for specific drug groups, or contraindications.

Digoxin does not control rate effectively during exercise and should be used as first line therapy only in people who are sedentary, or in overt heart failure.[88] 1+

In some people a combination of drugs may be required to control heart rate in atrial fibrillation. Options include the addition of digoxin to either a beta blocker or a rate-limiting calcium channel blocker.[89,90] The combination of beta blocker plus verapamil can cause severe bradycardia and should normally only be prescribed by cardiologists.[91] 1+ 3

**A** **Ventricular rate in AF should be controlled with beta blockers, rate-limiting calcium channel blockers** *(verapamil or diltiazem)***, or digoxin.**

**C** **Digoxin does not control rate effectively during exercise and should be used as first line therapy only in people who are sedentary, or in overt heart failure.**

**C** **In some people a combination of drugs may be required to control heart rate in atrial fibrillation. Options include the addition of digoxin to either a beta blocker or a rate-limiting calcium channel blocker.**

### 4.1.5 NON-PHARMACOLOGICAL THERAPIES

Atrioventricular node ablation and permanent pacing improve heart rate control, ejection fraction, symptomatic and functional status, and quality of life in patients with AF whose ventricular rate is uncontrolled on medical therapy.[82,92] Long term right ventricular pacing may however be deleterious to left ventricular function.[93]

2+
4
1+

**B** **Ablation and pacing should be considered for patients with AF who remain severely symptomatic or have LV dysfunction in association with poor rate control or intolerance to rate control medication.**

☑ Patients with atrial fibrillation who are severely symptomatic despite optimum tolerated medical therapy should be referred to a cardiac rhythm specialist for consideration of non-pharmacological therapy, eg radiofrequency ablation.

## 4.2 VENTRICULAR ARRHYTHMIAS

### 4.2.1 REVASCULARISATION FOR SECONDARY PREVENTION OF VT/VF

Four small retrospective studies examined the effectiveness of revascularisation in improving survival and quality of life for patients with CHD who have had sustained VT or who have been resuscitated from VF.[94-97] Revascularisation (coronary artery bypass graft or percutaneous coronary intervention) reduced the subsequent development of ischaemic VT induced at electrophysiological study, sudden death and out-of-hospital collapse.

2+
3

**C** **Revascularisation should be considered in patients who have had sustained VT or VF.**

☑ Patients with previous sustained VT/VF should undergo assessment for inducible ischaemia by stress testing or myocardial perfusion imaging followed, if appropriate, by coronary arteriography and revascularisation. These patients should all be considered for implantable cardioverter defibrillator therapy.

### 4.2.2 IMPLANTABLE CARDIOVERTER DEFIBRILLATOR THERAPY

***ICDs in patients at risk of life threatening arrhythmias - 'primary prevention'***

In patients with impaired LV function after previous MI in New York Heart Association (NYHA) classes I-III *(see Annex 2),* ICD therapy is superior to anti-arrhythmic drug therapy or usual medical management in reducing all-cause mortality.[98-101] There is no evidence that ICD implantation in the first month after myocardial infarction, or in conjunction with routine coronary artery bypass surgery reduces mortality in patients with impaired left ventricular ejection fraction.[52,102] Absolute benefit from ICD implantation is greatest in patients with spontaneous non-sustained VT plus inducible sustained VT,[100,101] more severely depressed ejection fraction ($<0.25$) or more prolonged QRS complex duration ($>120$ms).[98,99]

1+
1++

A further primary prevention trial compared cardiac resynchronisation therapy (CRT) alone or with a defibrillator (CRT-D) to optimal pharmacological therapy in patients with advanced heart failure (NYHA class III-IV), and QRS duration $>120$ms. CRT and CRT-D reduced the relative risk of the primary end point of death or hospitalisation for any cause by 19% and 20% respectively. The hazard ratio for all-cause mortality compared with pharmacological therapy was 0.76 (95% CI, 0.58 to 1.01, $p=0.059$) for the CRT group, and 0.64 (95% CI, 0.48 to 0.86, $p=0.003$) for CRT-D, although the difference between CRT and CRT-D was not statistically significant.[103] For further discussion of CRT see SIGN guideline 95 on the management of chronic heart failure.[104]

1+

A registry study from the MUSTT trial observed that patients with non-sustained VT who did not have sustained VT inducible at electrophysiological testing had a five year rate of cardiac arrest or death due to arrhythmia of 24%, as compared with 32% in patients with inducible tachyarrhythmias who were assigned to no anti-arrhythmic therapy (adjusted $p<0.001$). Patients with inducible tachyarrhythmias had a 48% five year mortality compared to 44% in non-inducible patients in the registry (adjusted $p=0.005$).[105]

2+

Table 1 summarises the entry criteria and results of the key trials.[52,98-103]

**A** **Patients with moderate to severe LV dysfunction** *(eg ejection fraction <0.35)*, **in NYHA class I-III at least one month after myocardial infarction should be considered for ICD therapy.**

**B** **Patients with spontaneous non-sustained ventricular tachycardia** *(especially if sustained ventricular tachycardia is inducible)*, **severely impaired ejection fraction** *(<0.25)* **or prolonged QRS complex duration** *(>120ms)* **should be prioritised for ICD implantation.**

**A** **Patients meeting criteria for ICD implantation who have prolonged QRS duration** *(>120ms)* **and NYHA class III-IV symptoms should be considered for CRT-D therapy.**

*Table 1 Entry criteria and results of trials examining ICD effectiveness in primary prevention of life threatening arrhythmias*

| Trial entry criteria | MADIT I[100] | CABG-PATCH[102] | MUSTT[101] | MADIT II[98] | COMPANION[103] *** | DINAMIT[52] | SCD-HEFT[99] |
|---|---|---|---|---|---|---|---|
| Non-sustained VT/inducible VT at EPS | Yes* | No | Yes | No | No | No | No |
| LV ejection fraction | ≤0.35 | ≤0.35 | ≤0.40 | ≤0.30 | ≤0.35 | ≤0.35 | ≤0.35 |
| NYHA class | I-III | 73% II&III | I-III | I-III | III-IV | I-III | II/III |
| Interval post-MI | ≥21 days | N/A | ≥4 days** | ≥1mth | N/A | 6-40 days | N/A |
| **Results** | | | | | | | |
| Mean follow up (months) | 27 | 32 | 39 | 20 | 12 | 30 | 46 |
| Control group mortality% | 39 | 21 | 55†¶ | 20 | 19 | 17 | 36† |
| ICD group mortality% | 16 | 23 | 24†¶ | 14 | 12 | 19 | 29† |
| RRR % (95% CI) | 54 (18 to74) | -7 (-42 to 19)‡ | 60¶ (41 to 73) | 31 (7 to 49) | 36 (14 to 52) | -8 (-55 to 24)‡ | 23 (4 to 38) |
| ARR% | 23 | -2 | 31†¶ | 6 | 7 | -2 | 7 |

* VT still inducible after intravenous procainamide
**83% of patients >1 month post-MI
***Cardiac resynchronisation plus ICD group vs optimal medical therapy
† 5-year Kaplan-Meier estimate
‡ Not statistically significant
¶ Non-randomised comparison
EPS Electrophysiology study
RRR Relative risk reduction
ARR Absolute risk reduction
N/A Not available

### *ICDs in patients surviving life threatening arrhythmias –'secondary prevention'*

Patients surviving VF or sustained symptomatic VT due to previous MI have improved survival following ICD implantation compared with amiodarone therapy.[106] In a meta-analysis of the key trials the overwhelming majority of patients were in NYHA classes I-III.[106] Patients with left ventricular ejection fraction ≤0.35 derived significantly more benefit from ICD therapy than those with better preserved left ventricular function. Patients with severe heart failure (eg NYHA class IV, with symptoms at rest) have been excluded from studies of ICDs, as such patients generally have poor prognosis due to progressive heart failure. 1+

Table 2 summarises the entry criteria and results of the key trials.[107-109]

A **Patients surviving the following ventricular arrhythmias in the absence of acute ischaemia or treatable cause should be considered for ICD implantation:**

- **cardiac arrest** *(VT or VF)*
- **VT with syncope or haemodynamic compromise**
- **VT without syncope if LVEF < 0.35** *(not NYHA IV).*

*Table 2: Entry criteria and results of trials examining ICD effectiveness in secondary prevention of life threatening arrhythmias.*

| Trial entry criteria | AVID[107] | CIDS[109] | CASH[108]* |
|---|---|---|---|
| Resuscitated cardiac arrest (VT/VF) | Yes | Yes | Yes |
| VT with syncope | Yes | Yes | - |
| VT with presyncope or angina + LVEF ≤0.35 | - | Yes | - |
| VT with presyncope/angina or congestive heart failure (CHF) + LVEF ≤0.40 | Yes | - | - |
| Unmonitored syncope w/inducible VT | - | Yes | - |
| **Results** | | | |
| Mean follow up (months) | 18 | 36 | 57 |
| Control group mortality% | 24 | 30 | 44 |
| ICD group mortality% | 16 | 25 | 36 |
| RRR% (95% CI) | 38(19 to 53) | 20(-8 to 40)‡ | 17(-33 to 48)‡ |
| ARR% | 8 | 5 | 8 |

* Comparison with amiodarone only
‡ Not statistically significant
RRR Relative risk reduction
ARR Absolute risk reduction

***Cost effectiveness of ICDs***

The evidence on the cost effectiveness of ICDs is weak.[110,111] Both improved targeting of patients at greatest risk of sudden cardiac death and fewer hospital admissions for maintenance and replacement of devices are likely to be necessary before the cost effectiveness ratios for ICDs relative to medical therapy approach conventional cost effectiveness thresholds.

### 4.2.3 ANTI-ARRHYTHMIC DRUG THERAPY

***Class 1 anti-arrhythmic drugs***

Class 1 anti-arrhythmic drugs used for treatment of premature ventricular beats or non-sustained VT in patients with previous MI show a strong trend towards increased risk of death.[4,112] | $1^{++}$

A **Class 1 anti-arrhythmic drugs should not be used for treatment of premature ventricular beats or non-sustained VT in patients with previous MI.**

***Beta blockers***

Routine use of beta blockers in post-MI patients reduces the risk of sudden death and all-cause mortality.[4] | $1^{++}$

A **Long term beta blockers are recommended for routine use in post-MI patients without contraindications.**

***Amiodarone and sotalol***

Amiodarone therapy in post-MI patients with impaired left ventricular function or frequent ventricular premature beats reduces the risk of sudden death, but does not significantly reduce all-cause mortality.[113,114] | 1++

Amiodarone therapy in patients with LV dysfunction or congestive heart failure (CHF) without sustained ventricular arrhythmias reduces the risk of sudden death but does not significantly reduce all-cause mortality.[99,115] | 1++

Sotalol therapy in unselected post-MI patients did not significantly reduce mortality.[116] | 1+

**A** **Amiodarone therapy is not recommended for post-MI patients or patients with congestive heart failure who do not have sustained ventricular arrhythmias or atrial fibrillation.**

**B** **Sotalol therapy is not recommended for post-MI patients who do not have sustained ventricular arrhythmias or atrial fibrillation.**

In patients who have recovered from an episode of sustained VT (with or without cardiac arrest), amiodarone or sotalol therapy is more effective than electrophysiologically-guided class 1 anti-arrhythmic therapy in preventing recurrent arrhythmic events and cardiac death.[4] | 1++

**B** **In patients who have recovered from an episode of sustained ventricular tachycardia** *(with or without cardiac arrest)* **who are not candidates for an ICD, amiodarone or sotalol should be considered.**

***Calcium channel blockers***

Calcium channel blocker therapy in post-MI patients does not reduce all-cause mortality.[4] | 1++

**A** **Calcium channel blocker therapy is not recommended for reduction in sudden death or all-cause mortality in post-MI patients.**

# 5 Arrhythmias associated with coronary artery bypass graft surgery

## 5.1 INTRODUCTION

AF is a common complication of coronary artery bypass graft (CABG) surgery, occurring in 17-53% patients.[117-125] The condition is self limiting in over 90% of patients within four to six weeks of surgery.[126,127]

Although ventricular ectopics and runs of VT are frequent following CABG surgery, occurring in up to 36% of patients,[125] the incidence of VF or sustained VT is lower, ranging from 0.95%[128] to 3.1%,[129] although rates as high as 8.5% have been reported.[130]

Cardiac arrest as a result of VF/pulseless VT in the postoperative period has high in-hospital mortality.[129,131] VT/VF often occurs in the early postoperative period when the patient is intensively monitored in a critical care area but may also occur more than a week postoperatively when the patient is not monitored.[125,129,131] Non-sustained VT following CABG surgery is a less specific marker of future ventricular arrhythmias than in the non-surgical setting.[128] The long term prognosis of survivors of VF/VT arrest is similar to those who did not experience VF/ VT.[129,131]

## 5.2 RISK FACTORS

Two systematic reviews with important methodological limitations, previous guidelines and two epidemiological studies identify age (20% increase in incidence for every 10 years over age 65) and previous AF as risk factors strongly associated with development of AF postoperatively.[44,132-135] | 1- 4

Other preoperative factors have been indirectly associated with postoperative development of AF in trials of prophylactic interventions. These include male sex,[132] obesity,[136] hypertension,[135] chronic obstructive pulmonary disease (COPD),[137] digoxin use, peripheral arterial disease, valvular heart disease, left atrial enlargement, previous cardiac surgery, pericarditis, elevated postoperative adrenergic tone,[44] concurrent valve surgery, atrial enlargement, poor LV function,[133] P wave dispersion[140] and withdrawal of ACE inhibitors and beta blockers.[134] | 3 1- 4

The major preoperative risk factor for postoperative VT and VF is low left ventricular ejection fraction.[125] | 2+

**D** **In patients undergoing coronary artery bypass graft surgery, age, previous AF and left ventricular ejection fraction should be considered when assessing risk of postoperative arrhythmia.**

## 5.3 PROPHYLACTIC INTERVENTIONS

Although prophylaxis is effective in reducing the incidence of AF, the evidence is conflicting as to whether it decreases the incidence of stroke or mortality or shortens hospital stay.[121,122,124,141]

Even when such effects have been demonstrated, variation in study methodologies, including lack of definition of AF, adverse events not being primary outcomes, variation in definitions of end points, prolonged time period of studies during which beta blockers were widely introduced, and details of randomisation, concealment and attrition not being included limits the interpretation of these findings.[138,139] Pharmacological treatment of AF is often quickly effective in restoring sinus rhythm or controlling heart rate.[140]

Prophylaxis also reduces the incidence of ventricular arrhythmias but it does not reduce associated mortality.[122,138]

### 5.3.1 PHARMACOLOGICAL THERAPIES

***Amiodarone/beta blockers***

Amiodarone or beta blockers, including sotalol, reduce the incidence of AF following CABG surgery by a similar magnitude.[138,139,141,142] The potential relative risk reduction in incidence of AF with amiodarone is 46% and with beta blockers is 35%.[141] | 1++

**A** **Amiodarone may be used when prophylaxis for atrial fibrillation and ventricular arrhythmias is indicated following CABG surgery.**

**A** **Beta blockers including sotalol may be used when prophylaxis for atrial fibrillation is indicated following CABG surgery.**

☑ Preoperative beta blocker therapy should be reintroduced as soon as safe to do so after surgery.

***Calcium channel blockers***

Rate-limiting calcium channel blockers, eg verapamil and diltiazem are effective in reducing the incidence of AF following surgery but dihydropyridines are ineffective.[143] | 1++

**B** **Verapamil and diltiazem may be used for prophylaxis of atrial fibrillation following CABG surgery.**

***Digoxin***

Digoxin does not reduce the incidence of AF following CABG surgery.[144] | 1+

**B** **Digoxin should not be used for prophylaxis of atrial fibrillation following CABG surgery.**

***Glucose-insulin-potassium***

One good quality RCT[145] and one retrospective analysis[146] suggest that glucose-insulin-potassium regimens do not reduce the incidence of AF following cardiac surgery. An RCT study with limitations around concealment of interventions showed some benefit.[147] | 1++ 2- 1-

**C** **Glucose-insulin-potassium regimens should not be used for prophylaxis of atrial fibrillation following CABG surgery.**

***n-3-polyunsaturated fatty acids***

In one RCT, n-3-polyunsaturated fatty acids (PUFAs) (2 g/day) administered for five days preoperatively and postoperatively until the day of hospital discharge reduced the incidence of postoperative AF following elective CABG and reduced hospital stay.[127] This single study (n = 160) provides insufficient evidence on which to base a recommendation. | 1+

### 5.3.2 MANIPULATION OF BLOOD ELECTROLYTES

Magnesium is effective in reducing the incidence of AF and ventricular arrhythmias after cardiac surgery.[118, 121,122] Magnesium confers no additional reduction in AF when co-administered with sotalol.[148] | 1++ 1+

**A** **Magnesium may be used when prophylaxis for atrial fibrillation and ventricular arrhythmias is indicated following CABG surgery.**

One study has associated hypokalaemia or hypocalcaemia with the occurrence of VT following CABG surgery.[130] | 2+

No studies were identified that investigated whether correction of hypokalaemia or hypocalcaemia reduced the incidence of VT. However, correction of these deficits is accepted clinical practice.

☑ Blood levels of potassium and calcium should be measured frequently following CABG surgery and corrected if necessary.

### 5.3.3 ANAESTHESIA AND ANALGESIA

Whilst spinal anaesthesia does not reduce the incidence of AF, epidural analgesia results in a 48% reduction in the incidence of AF and tachycardia after CABG surgery.[149] Amiodarone is more effective than epidural analgesia and their combination confers no additional benefit.[150] 1+

The choice of general anaesthetic agent (propofol, midazolam, sevoflurane, desflurane) does not influence the incidence of AF.[151] 1+

There were conflicting results in two studies of the effects of non-steroidal anti-inflammatory drugs (NSAIDs). Ketorolac reduced the incidence of AF by 65% (relative risk reduction) compared with a control group whilst naproxen did not influence the incidence of AF.[152,153] 1+ 1++

A **The choice of anaesthetic agent or technique and analgesia should be based on factors other than atrial fibrillation prophylaxis.**

### 5.3.4 SURGICAL TECHNIQUES

***Off-pump surgery***

Off-pump (without cardiopulmonary bypass) CABG surgery is associated with a reduction in the incidence of AF in elderly patients when compared with the use of cardiopulmonary bypass. The risks and benefits of off-pump CABG surgery remain controversial and the long term outcomes remain unknown.[117,120,154,155] 2++ 1++ 1+

A **The choice of whether or not to use cardiopulmonary bypass should be based on factors other than atrial fibrillation prophylaxis.**

***Atrial pacing***

Atrial pacing is associated with a 43% reduction in incidence of AF following CABG surgery. Although use of pacing avoids the potential side effects of pharmacological measures it carries an extremely small risk of tamponade and death. There are potential infection problems if wires cannot be completely removed.[141,156] 1++

A **Atrial pacing may be used for prophylaxis of AF in patients who have atrial pacing wires placed for other indications.**

***Fat pad preservation***

In one randomised study the preservation of the anterior epicardial fat pad between aorta and pulmonary artery when cross clamping during CABG reduced the incidence of postoperative AF when compared with fat pad dissection.[157] This small study (n = 55) does not provide sufficient evidence on which to base a recommendation. 1+

***Bonded cardiopulmonary bypass circuits***

A number of studies suggest that the use of heparin bonded circuits improves clinical outcomes in patients undergoing CABG.[140,158-161] Reduction in AF incidence is rarely a primary outcome and monitoring for AF is limited to 48 hours in some studies. It is unclear whether bonded cardiopulmonary bypass circuits are associated with a reduction in AF. 1+ 4

A **Bonded cardiopulmonary bypass circuits should not be used on the basis of AF prophylaxis alone.**

***Hypothermia***

In a randomised study patients underwent mild (34ºC) or moderate (28ºC) hypothermic cardiopulmonary bypass. AF rates in each group were determined retrospectively by review of hospital records. There was a significantly higher incidence of AF in the moderate compared with the mild hypothermic group.[162] 1-

### 5.3.5 DEFIBRILLATOR IMPLANTATION

In one study, prophylactic use of ICDs in patients at high risk for ventricular arrhythmias after CABG did not improve life expectancy in patients with a poor left ventricular ejection fraction.[102] 1+

**A** **Defibrillators should not be routinely implanted in patients with a poor left ventricular ejection fraction at the time of coronary artery bypass graft surgery.**

## 5.4 TREATMENTS FOR ATRIAL FIBRILLATION

### 5.4.1 PHARMACOLOGICAL THERAPIES

There is little evidence to guide treatment of AF following CABG surgery. Recommendations are based on expert opinion from previous guidelines (*see section 3.1 and 4.1*).[44] 4

**D**
- **Patients with AF and haemodynamic compromiseshould have synchronised cardioversion.**
- **In the immediate postoperative period, patients with persistent AF without haemodynamic compromise should be treated with rate-limiting therapy.**
- **Patients with persistent AF should be considered for elective synchronised cardioversion.**

☑ Whatever pharmacological therapy is used for treatment of AF, the need for continuing treatment should be reviewed within six weeks of hospital discharge.

***Anticoagulation***

A systematic review identified no studies demonstrating effects of immediate anticoagulation on stroke risk in patients with AF post cardiac surgery.[163] The review identified one cohort study that provided some evidence that there is a risk of pericardial effusions with early anticoagulation.[164] 1+ 2-

☑ Anticoagulation should be considered on a case-by-case basis for patients with AF following CABG where it is anticipated that the AF is likely to persist.

### 5.4.2 DC CARDIOVERSION

Although one small study (n = 48) found that DC cardioversion reduces the duration of AF there is no evidence that DC cardioversion reduces AF recurrence rate or the duration of hospital stay.[165] 1-

## 5.5 TREATMENTS FOR VENTRICULAR ARRHYTHMIAS

There is little evidence to guide treatment of ventricular arrhythmias associated with CABG surgery. Recommendations are based on published expert opinion.[3] 4

Sternal reopening for internal massage, defibrillation or control of bleeding is most effective if carried out in critical care, less than 24 hours from surgery and if performed less than 10 minutes from arrest. It is of limited value when performed more than 24 hours after surgery or in the general ward.[166,167] Institution of cardiopulmonary bypass may be indicated in the early postoperative period to correct surgical bleeding or coronary artery graft occlusion and rest an exhausted myocardium.[167] 3 4

D **Patients with VF or pulseless VT should be defibrillated immediately.**
- **Intravenous adrenaline/epinephrine should be used for the management of patients with refractory VT/VF.**
- **Sternal reopening, internal heart massage and internal defibrillation should be considered in patients with refractory VT/VF.**
- **Intravenous amiodarone should be considered for the management of patients with refractory VT/VF.**

☑
- Cardiac tamponade following CABG surgery is a cause of cardiac arrest and should be considered as a differential diagnosis.
- Should other methods fail, sternal reopening should be performed promptly for cardiac arrest if the patient is in critical care and within 24 hours of surgery. Sternal reopening after the first 24 hours in the general ward is unlikely to improve survival.
- The ability to institute cardiopulmonary bypass in the critical care area should be available in all units undertaking coronary artery bypass grafting surgery.

☑ Telemetric ECG monitoring of patients in the general ward allows early detection and treatment of patients in VT/VF.

☑ Patients suffering VT/VF >48 hours after CABG should be considered for ICD implantation.

### 5.5.1 BIPHASIC VERSUS MONOPHASIC DEFIBRILLATION

There is a high incidence of VF (38%) after declamping the aorta during CABG surgery.[168] In one high quality RCT biphasic defibrillation was more effective and used less energy than monophasic defibrillation when cardioverting VF that occurred on declamping the aorta.[168] 1++

A **Biphasic defibrillation should be used to terminate ventricular fibrillation that occurs on declamping the aorta.**

## 5.6 PREOPERATIVE INFORMATION

No evidence was identified which related directly to the effectiveness of provision of information on arrhythmias post CABG. A well conducted systematic review of a number of small, poor quality studies did not identify any benefit for preoperative education in respect of medical outcomes for CABG.[169] Additionally, an RCT examining the impact of standardised preoperative education on recovery following coronary artery bypass surgery found no difference in length of stay, anxiety, pain, depression or well-being between control and intervention groups.[170] However, as described in SIGN guideline 93 on acute coronary syndromes and SIGN guideline 96 on the management of stable angina there are a range of factors relating to information/education needs of patients which should be considered by all health professionals in conjunction with appropriate educational and psychological strategies.[171,172] 1+ 4

☑ Preoperative information/education, including that related to arrhythmias, should be tailored to individual patients' needs.

# 6 Psychosocial issues

## 6.1 INTRODUCTION

Psychosocial outcomes in patients with arrhythmias have mainly been examined as an adjunct to medical and mortality outcomes. Studies in this area often have methodological flaws and tend to focus on a limited range of quality of life issues: looking at physical or activity functioning rather than partner/family issues, memory difficulties and other psychosocial patient concerns.[173,174] 1+

Psychological or emotional factors can influence and confound the incidence of cardiac arrhythmia.[175,176] Although anxiety and depressive disorders occur frequently in patients with CHD they are rarely identified or managed in the cardiology setting. Some patients may have had pre-existing mental health problems including depression *(see SIGN guideline 97 on risk estimation and the prevention of cardiovascular disease for discussion of depression as a risk factor)*.[6] For other patients, cardiac ill health precipitates new anxieties, depression or cognitive dysfunction (including poor memory and concentration) affecting their ability to participate in treatment regimens and rehabilitation.[177-179] 1+ 2+

## 6.2 PSYCHOSOCIAL ASSESSMENT AND SCREENING

Physicians' and nurses' subjective judgements of patient anxiety are not as accurate as measurements of anxiety on validated scales *(see SIGN guideline 93 on acute coronary syndromes)*.[171] Standardised screening tools, such as the Hospital Anxiety and Depression Scale, are useful in psychological assessment with referral of more complex cases to psychological or other mental health services.[179] Selective screening of cardiac patients for cognitive problems will aid patient care post cardiac arrest and in older people where the relationship between depression and cognitive impairment is complex.[177,178,180] 2+ 4

**D** **Patients with chronic cardiac arrhythmias should be screened for anxiety or depressive disorders with referral to specialist mental health services where appropriate.**

**D** **Selective cognitive screening should be available especially for post arrest and older cardiac patients experiencing persistent memory or other cognitive difficulties.**

## 6.3 PSYCHOSOCIAL ISSUES FOR ICD RECIPIENTS

Fifteen to 60% of ICD recipients experience high levels of distress around the time of surgery, with anxiety and depression as the main emotional responses.[181] Although ICD implantation may relieve some of the fear of sudden death and is welcomed by most patients, it can impose new fears that can affect return to normal life roles or function. Specific ICD related fears include fear of shock, device malfunction or death. There may also be concerns around changes in body image.[174,182] 1+

Lifestyle changes including reduced physical and sexual activity and enforced driving restrictions can also have a negative impact on quality of life.[174,182] 1+

Psychosocial adjustment problems are more common in ICD recipients younger than age 50.[174,182] Functional status, coping style, family problems and inadequate social support are also associated with psychological adjustment.[182] Repeated shock experiences are associated with poor quality of life.[110] The phenomenon of phantom shocks may also contribute to maladjustment to ICD[183,184] although evidence on the association of repeated shock experience with psychological problems is inconclusive.[182] Families and partners of the patient can experience similar fears and anxiety: there are several case reports of family members becoming overprotective and spouses exhibiting hypervigilant behaviours and reporting low marital satisfaction. There are no large scale studies.[181] 1+ 3 4 2+

A meta-analysis found no difference in psychological outcome between patients with ventricular arrhythmia treated with ICDs or medical therapy, or between pre and post implant ICD patients.[185] This highlights that poor psychosocial outcome in ICD patients may also be associated with the underlying cardiac condition rather than a direct response to implantation of the ICD device and its therapy. 2+

C **Psychosocial implications for people experiencing cardiac arrhythmias should be considered by all healthcare staff throughout assessment, treatment and care.**

☑ Psychosocial support for patients experiencing cardiac arrhythmias should not be restricted to recipients of ICDs.

## 6.4 PSYCHOSOCIAL INTERVENTIONS

Psychological interventions can reduce anxiety and depression in patients with CHD as part of comprehensive cardiac rehabilitation programmes.[181] There is no evidence of effect on total or cardiac mortality.[186] 2+

One systematic review and two subsequent RCTs provide evidence of benefit for programmes incorporating cognitive behavioural therapy (CBT) in reducing anxiety and improving quality of life specifically in patients with ICDs.[182,187,188] SIGN guideline 57 on cardiac rehabilitation recommends that staff delivering psychosocial interventions in cardiac rehabilitation should be trained in CBT or similar techniques.[179] 1+ 4

As patients with ICDs have similar secondary prevention, lifestyle change and educational needs as other CHD patients it would be appropriate to include them as part of existing cardiac rehabilitation services.[189] 4

B **Psychosocial intervention offered as part of a comprehensive rehabilitation programme should encompass a cognitive behavioural component.**

## 6.5 ANTIDEPRESSANT MEDICATIONS IN PATIENTS WITH CORONARY HEART DISEASE

Clinical depression is a risk factor for cardiovascular mortality including cardiac arrest.[190] Following acute MI,15-20% of patients develop depression, which has been shown to be associated with increased mortality.[36,191] Tricyclic antidepressant drugs are associated with risk of cardiac arrhythmia and are considered to be contraindicated in acute myocardial infarction (see British National Formulary, BNF; www.bnf.org).[192,193] Selective serotonin reuptake inhibitors have been shown to be safe and effective in patients following acute MI in randomised controlled trials,[191] but have not been shown to improve prognosis.[36,194]

# 7 Sources of further information and support for patients and carers

**Action on Smoking and Health (ASH)**
8 Frederick Street, Edinburgh EH2 2HB
Tel: 0131 225 4725 • Fax: 0131 225 4759
www.ash.org.uk • Email: ashscotland@ashscotland.org.uk

ASH Scotland is a voluntary organisation providing expert information and advice on all aspects of tobacco. Provides a range of written information including advice on passive smoking, smoking and young people, smoking cessation and smoking policies in the workplace.

**Blood Pressure Association**
60 Cranmer Terrace, London, SW17 0QS
Tel: 020 8772 4994 (Best time to telephone: 9.30am - 5.30pm, Monday to Friday)
Fax: 020 8772 4999
www.bloodpressureuk.org.uk

The Blood Pressure Association (BPA) helps people with high blood pressure to become more involved in controlling their condition. Provides a range of information including management of hypertension, medications, lifestyle changes and other risk factors.

**British Cardiac Patients Association**
BCPA Head Office, 2 Station Road, Swavesey, Cambridge, CB4 5QJ
Tel: 0800 479 2800 • Fax: 01954 202 022
www.bcpa.co.uk • Email: enquiries@bcpa.co.uk

The British Cardiac Patients Association is a charitable organisation run by volunteers providing support, advice and information to cardiac patients and their carers.

**British Heart Foundation (Scotland)**
4 Shore Place, Edinburgh, EH6 6WW
Tel: 0131 555 5891 • Heart Information line: 08450 70 80 70
(Available: 9am-5pm, Monday to Friday)
www.bhf.org.uk • Email: scotland@bhf.org.uk

Provides a telephone information service for those seeking information on heart health issues. Also provides a range of written materials offering advice and information to CHD patients and carers. Topics include physical activity, smoking and diabetes.

**Chest Heart and Stroke Scotland**
65 North Castle Street, Edinburgh, EH2 3LT
Tel: 0131 225 6963 • Helpline: 0845 0776000
www.chss.org.uk • Email: admin@chss.org.uk

Provides a 24 hour advice line offering confidential, independent advice on all aspects of chest, heart and stroke illness. A series of information booklets, factsheets and videos are available free of charge to patients and carers. There are over 30 cardiac support groups in Scotland which are affiliated to CHSS, patients can contact CHSS for details of their nearest local support group.

**Depression Alliance Scotland**
3 Grosvenor Gardens, Edinburgh, EH12 5JU
Tel: 0131 467 3050
www.depressionalliance.org • Email: info@dascot.org

Provides information and support for people in Scotland who have depression.

**Diabetes UK**
10 Parkway, London, NW1 7AA
Tel: 020 7424 1000 • Careline: 0845 120 2960
(Best time to telephone: 9.30am - 5.30pm, Monday to Friday)
www.diabetes.org.uk • Email: careline@diabetes.org.uk

Diabetes UK is a national organisation providing information and advice on all aspects of diabetes such as diabetic care and diet. Provides a series of information leaflets including Diabetes UK's own magazine *Balance*.

**Heart Surgery in Great Britain**
http://heartsurgery.healthcarecommission.org.uk/

This website has been developed by the Healthcare Commission and the Society for Cardiothoracic Surgery in Great Britain and Ireland to help heart surgery patients make informed choices about their treatment. It provides patients and carers with information on the different operations available and the benefits of having heart surgery.

**Heart UK**
7 North Road, Maidenhead, Berkshire, SL6 1PE
Tel: 01628 628 638 (Best time to telephone: 9.30am - 4pm, Monday to Friday)
Fax: 01628 628 698
www.heartuk.org.uk • Email: ask@heartuk.org.uk

Heart UK is a national charity aiming to offer information and support to anyone at high risk of CHD, particularly families with inherited high cholesterol. Provides a range of information including management of CHD by lifestyle, drugs and diet.

**High Blood Pressure Foundation**
Department of Medical Sciences, Western General Hospital, Edinburgh, EH4 2XU
Tel: 0131 332 9211 (Best time to telephone: 9.30am - 5pm, Monday to Friday)
Fax: 0131 332 9211
www.hbpf.org.uk • Email: hbpf@hbpf.org.uk

The High Blood Pressure Foundation is a registered charity which aims to improve the assessment, treatment and public awareness of high blood pressure. Provides a range of information leaflets including understanding high blood pressure and cholesterol and cardiovascular risk.

**Implanted Defibrillator Association of Scotland**
10 Selkirk Avenue, Paisley, PA2 8JF
Tel: 01505 813 995
Email: hanheart@aol.com

This group provides information, advice and practical support to patients who have implantable defibrillators.

**Mental Health Foundation (Scotland)**
Merchant's House, 30 George Square, Glasgow, G2 1EG
Tel: 0141 572 0125
www.mentalhealth.org.uk • Email: Scotland@mhf.org.uk

The Mental Health Foundation helps people prevent, cope with and recover from mental health problems. Provides a range of factsheets on mental health issues including anxiety and depression.

**NHS Health Scotland**
Woodburn House, Canaan Lane, Edinburgh, EH10 4SG
Tel: 0131 536 5500 • Textphone: 0131 535 5503 • Fax: 0131 535 5501
www.hebs.com • Email: publications@health.scot.org.uk (information on obtaining Health Scotland publications); library.enquiries@health.scot.nhs.uk (help with general health information enquiries)

NHS Health Scotland is a special health board within NHSScotland. The organisation provides information on projects, publications, support groups and information leaflets relating to CHD.

**NHS 24**
Tel: 08454 24 24 24
www.nhs24.com

NHS 24 is a nurse-led service for members of the public. It is a helpline offering health information, advice and help over the telephone.

**Scotland's Health on the Web**
www.show.scot.nhs.uk

This website provides public access to publications relating to CHD such as the strategy for CHD and stroke in Scotland.

**Scottish Association for Mental Health (SAMH)**
Cumbrae House, 15 Carlton Court, Glasgow, G5 9JP
Tel: 0141 568 7000 (Best time to telephone: 2pm - 4.30pm, Monday to Friday)
www.samh.org.uk • Email: enquire@samh.org.uk

Provides patients and carers with information on all aspects of mental health.

# 8 Implementation, audit and research

## 8.1 LOCAL IMPLEMENTATION

Implementation of national clinical guidelines is the responsibility of each NHS Board and is an essential part of clinical governance. It is acknowledged that every Board cannot implement every guideline immediately on publication, but mechanisms should be in place to ensure that the care provided is reviewed against the guideline recommendations and the reasons for any differences assessed and, where appropriate, addressed. These discussions should involve both clinical staff and management. Local arrangements may then be made to implement the national guideline in individual hospitals, units and practices, and to monitor compliance. This may be done by a variety of means including patient-specific reminders, continuing education and training, and clinical audit.

## 8.2 KEY POINTS FOR AUDIT

ISD Scotland's CHD and Stroke National Clinical Datasets Development Programmes are working to develop national standard datasets for implementation in IT systems supporting patient care. The following clinical datasets have been developed and are available at www.datadictionary.scot.nhs.uk

- CHD core
- Acute coronary syndromes
- Cardiac rehabilitation
- Heart failure
- Electrophysiology.

## 8.3 RECOMMENDATIONS FOR RESEARCH

- Investigation of the impact of various types of arrhythmia treatment on the family/partner, potential cognitive dysfunction and effect of repeated shocks/unexplained shock experiences in ICD recipients
- Needs assessment of training in CPR
- Effectiveness of prophylaxis for arrhythmias in high risk patients undergoing cardiac surgery assessed on outcomes of stroke, intensive care unit admission, length of stay and cost effectiveness.

## 8.4 ADDITIONAL ADVICE TO NHSSCOTLAND FROM NHS QIS AND THE SCOTTISH MEDICINES CONSORTIUM

### 8.4.1 NHS QIS APPROVED NICE MTAS

NICE technology appraisal guidance no. 95. Implantable cardioverter defibrillators for arrhythmias.[195]

### 8.4.2 SMC ADVICE

The Scottish Medicines Consortium has issued advice on the use of eplerenone after myocardial infarction.

Further details are available from www.scottishmedicines.org.uk

# 9 Development of the guideline

## 9.1 INTRODUCTION

SIGN is a collaborative network of clinicians, other healthcare professionals and patient organisations and is part of NHS Quality Improvement Scotland. SIGN guidelines are developed by multidisciplinary groups of practising clinicians using a standard methodology based on a systematic review of the evidence. Further details about SIGN and the guideline development methodology are contained in "SIGN 50: A Guideline Developer's Handbook", available at **www.sign.ac.uk**

## 9.2 THE GUIDELINE DEVELOPMENT GROUP

| | |
|---|---|
| Professor Stuart Cobbe (Chair) | *Consultant Cardiologist, Glasgow Royal Infirmary* |
| Professor Andrew Rankin (Secretary) | *Consultant Cardiologist, Glasgow Royal Infirmary* |
| Dr R Peter Alston | *Consultant, Department of Anaesthesia, Critical Care and Pain Medicine, Royal Infirmary of Edinburgh* |
| Mr Geoff Berg | *Clinical Director of Cardiac Surgery, Western Infirmary, Glasgow* |
| Mr Ian Colquhoun | *Consultant in Cardiothoracic Surgery, Glasgow Royal Infirmary* |
| Ms Joyce Craig | *Senior Health Economist, NHS Quality Improvement Scotland, Glasgow* |
| Dr Mark Francis | *Consultant Cardiologist, Victoria Hospital, Fife* |
| Dr Stephen Glen | *Consultant Cardiologist, Stirling Royal Infirmary* |
| Mr Michael Hanley | *Lay Representative, Paisley* |
| Mr Malcolm McDonald | *Lay Representative, Angus (deceased)* |
| Mr Rod Moore | *Clinical Lead, Scottish Ambulance Service, Falkirk* |
| Dr Noelle Murphy | *Consultant in Accident and Emergency, Raigmore Hospital, Inverness* |
| Dr Morag Osborne | *Consultant Clinical Psychologist, Southern General Hospital, Glasgow* |
| Dr Terry Pringle | *Consultant Cardiologist, Ninewells Hospital, Dundee* |
| Mrs Mary Richardson | *Heartstart UK Manager, British Heart Foundation, Edinburgh* |
| Professor David Stott | *Professor of Geriatric Medicine, Glasgow Royal Infirmary* |
| Dr Lorna Thompson | *Programme Manager, SIGN* |
| Ms Joanna Welsh | *Information Officer, SIGN* |
| Dr Peter Wimpenny | *Associate Director, Joanna Briggs Collaborating Centre, The Robert Gordon University, Aberdeen* |
| Dr Simon Woldman | *Consultant Cardiologist, University College London Hospitals* |

The membership of the guideline development group was confirmed following consultation with the member organisations of SIGN. All members of the guideline development group made declarations of interest and further details of these are available on request from the SIGN Executive. Guideline development and literature review expertise, support and facilitation were provided by the SIGN Executive.

## 9.3 THE STEERING GROUP

A steering group comprising the chairs of the five SIGN CHD guidelines and other invited experts was established to oversee the progress of guideline development. This group met regularly throughout the development of the guidelines.

| | |
|---|---|
| Dr Kevin Jennings | *Co-chair and Consultant Cardiologist, Aberdeen Royal Infirmary* |
| Professor Lewis Ritchie | *Co-chair and Mackenzie Professor of General Practice, University of Aberdeen* |
| Dr Alan Begg | *Chair of SIGN stable angina guideline* |
| Dr Nick Boon | *Consultant Cardiologist, Royal Infirmary of Edinburgh* |
| Ms Marjory Burns | *Director for Scotland, British Heart Foundation* |
| Mr David Clark | *Chief Executive, Chest, Heart and Stroke Scotland* |
| Professor Stuart Cobbe | *Chair of SIGN arrhythmias guideline* |
| Ms Joyce Craig | *Senior Health Economist, NHS Quality Improvement Scotland* |
| Dr Iain Findlay | *Chair of SIGN acute coronary syndromes guideline* |
| Professor Keith Fox | *Professor of Cardiology, University of Edinburgh* |
| Dr James Grant | *Chair of SIGN prevention guideline* |
| Mr James Grant | *Lay representative, Balerno* |
| Dr Grace Lindsay | *Reader in Clinical Nursing Research, Glasgow Caledonian University* |
| Dr Moray Nairn | *Programme Manager, SIGN* |
| Professor Allan Struthers | *Chair of SIGN chronic heart failure guideline* |
| Dr Lorna Thompson | *Programme Manager, SIGN* |

## 9.4 SYSTEMATIC LITERATURE REVIEW

The evidence base for this guideline was synthesised in accordance with SIGN methodology. A systematic review of the literature was carried out using a search strategy devised by a SIGN Information Officer. Databases searched include Medline, Embase, Cinahl, PsychINFO, and the Cochrane Library. For most searches, the year range covered was 1999-2005. Internet searches were carried out on various websites including the New Zealand Guidelines Programme, NELH Guidelines Finder, and the US National Guidelines Clearinghouse. The Medline version of the main search strategies can be found on the SIGN website, in the section covering supplementary guideline material. The main searches were supplemented by material identified by individual members of the development group.

## 9.5 CONSULTATION AND PEER REVIEW

### 9.5.1 NATIONAL OPEN MEETING

A national open meeting is the main consultative phase of SIGN guideline development, at which the guideline development group presents its draft recommendations for the first time. The national open meeting for the five parallel SIGN guidelines on aspects of coronary heart disease was held on 16 September 2005 and was attended by over 600 representatives of all the key specialties relevant to the guideline. The draft guideline was also available on the SIGN website for a limited period at this stage to allow those unable to attend the meeting to contribute to the development of the guideline.

### 9.5.2 SPECIALIST REVIEW

This guideline was also reviewed in draft form by the following independent expert referees, who were asked to comment primarily on the comprehensiveness and accuracy of interpretation of the evidence base supporting the recommendations in the guideline. SIGN is very grateful to all of these experts for their contribution to the guideline.

| | |
|---|---|
| Dr Chris D Baker | *General Practitioner, Carlisle* |
| Professor Christine Bond | *Consultant in Pharmaceutical Public Health, Department of General Practice and Primary Care, University of Aberdeen* |
| Professor A John Camm | *Professor of Clincal Cardiology, St George's Hospital Medical School, University of London* |
| Dr Michael C Colquhoun | *Senior Lecturer, Department of Epidemiology, Statistics and Public Health, Cardiff University* |
| Dr Mohamed Elfellah | *Pharmacist, Aberdeen Royal Infirmary* |
| Professor Clifford Garratt | *Professor of Cardiology, Manchester Heart Centre* |
| Mrs Gillian A Jardine | *Principal Pharmacist – Clinical Services, Ayr Hospital* |
| Dr Colville Laird | *Chairman, BASICS Scotland, Perthshire* |
| Ms Susan Kinsey | *Lay representative* |
| Dr James Leask | *General Practitioner, Campbeltown* |
| Dr Alistair Macfie | *Consultant Anaesthetist, Western Infirmary, Glasgow* |
| Dr Janet McComb | *Consultant Cardiologist, Freeman Hospital, Newcastle upon Tyne* |
| Dr Jerry Nolan | *Consultant in Anaesthesia and Intensive Care Medicine, Royal United Hospital, Bath* |
| Dr David Smith | *Consultant/Senior Lecturer in Anaesthetics, Southampton General Hospital* |
| Dr Ellis J Simon | *Consultant Cardiothoracic Anaesthetist, Royal Infirmary of Edinburgh* |
| Ms Nicola Stuckey | *Head of Clinical Psychology, Astley Ainslie Hospital, Edinburgh* |

### 9.5.3 SIGN EDITORIAL GROUP

As a final quality control check, the guideline was reviewed by an editorial group comprising members of SIGN Council to ensure that the specialist reviewers' comments have been addressed adequately and that any risk of bias in the guideline development process as a whole has been minimised. The editorial group for this guideline was as follows.

| | |
|---|---|
| Dr Keith Brown | *Member of SIGN Council* |
| Dr Kevin Jennings | *Co-chair SIGN CHD Steering Group and Consultant Cardiologist, Aberdeen Royal Infirmary* |
| Professor Gordon Lowe | *Chair of SIGN; Co-Editor* |
| Ms Anne Matthew | *Member of SIGN Council* |
| Dr Safia Qureshi | *SIGN Programme Director; Co-Editor* |
| Professor Lewis Ritchie | *Co-chair SIGN CHD Steering Group and Mackenzie Professor of General Practice, University of Aberdeen* |
| Dr Sara Twaddle | *Director of SIGN; Co-Editor* |

## 9.6 ACKNOWLEDGEMENTS

SIGN is grateful to the following former members of the guideline development group and others who have contributed to the development of this guideline.

| | |
|---|---|
| Dr Kyle Lifson | *General Practitioner, Drymen Health Centre* |
| Mr Gordon McNeill | *Training Officer, Scottish Ambulance Service College, Edinburgh* |
| Mr Iain Lowis | *Head of Community Fundraising, British Heart Foundation, Edinburgh* |
| Mrs Hazel Moss | *Lay Representative, Implanted Defibrillator Association of Scotland* |
| Dr Rani Sinnak | *Consultant Clinical Psychologist, Ayrshire Central Hospital* |

# Abbreviations and definitions

| | |
|---|---|
| **AADs** | Anti-arrhythmic drugs |
| **ACE** | Angiotensin converting enzyme |
| **AED** | Automated external defibrillator |
| **AF** | Atrial fibrillation |
| **ARR** | Absolute risk reduction |
| **AV** | Atrioventricular |
| **AVID** | Antiarrhythmics Versus Implantable Defibrillators |
| **BNF** | British National Formulary |
| **CABG** | Coronary artery bypass graft |
| **CABG-PATCH** | Coronary Artery Bypass Graft Surgery with/without Simultaneous Epicardial Patch for Automatic Implantable Cardioverter Defibrillator |
| **CASH** | Cardiac Arrest Study Hamburg |
| **CBT** | Cognitive behavioural therapy |
| **CHD** | Coronary heart disease |
| **CHF** | Congestive heart failure |
| **CI** | Confidence interval |
| **CIDS** | Canadian Implantable Defibrillator Study |
| **COMPANION** | Comparison of medical therapy, pacing, and defibrillation in chronic heart failure |
| **COPD** | Chronic obstructive pulmonary disease |
| **CoSTR** | Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations |
| **CPB** | Cardiopulmonary bypass |
| **CPR** | Cardiopulmonary resuscitation |
| **CRT** | Cardiac resynchronisation therapy |
| **CRT-D** | Cardiac resynchronisation therapy + defibrillator |
| **DC** | Direct current |
| **DINAMIT** | Defibrillator in acute myocardial infarction trial |
| **DLCO** | Diffusing capacity of the lung for carbon monoxide (transfer factor) |
| **ECG** | Electrocardiogram |
| **EPS** | Electrophysiology study |
| **HAT** | Home AED trial |
| **ICD** | Implantable cardioverter defibrillator |
| **IV** | Intravenous |
| **LV** | Left ventricular |
| **LVEF** | Left ventricular ejection fraction |
| **MADIT** | Multicenter automatic defibrillator implantation trial |

| | |
|---|---|
| **MI** | Myocardial infarction |
| **MTA** | Multiple technology appraisal |
| **MUSTT** | Multicenter Unsustained Tachycardia Trial |
| **NHS** | National Health Service |
| **NICE** | National Institute for Health and Clinical Excellence |
| **NSAIDs** | Non-steroidal anti-inflamatory drugs |
| **NYHA** | New York Heart Association |
| **PEA** | Pulseless electrical activity |
| **PUFAs** | Polyunsaturated fatty acids |
| **QoL** | Quality of life |
| **QRS complex** | The principal deflection in the electrocardiogram, representing ventricular depolarisation |
| **QT interval** | The time elapsing from the beginning of the QRS complex to the end of the T wave in an electrocardiogram, representing the total duration of electrical activity of the ventricles |
| **RCT** | Randomised controlled trial |
| **ROSC** | Return of spontaneous circulation |
| **RRR** | Relative risk reduction |
| **SCD** | Sudden cardiac death |
| **SCD-HEFT** | Sudden cardiac death in heart failure |
| **SMC** | Scottish Medicines Consortium |
| **STEMI** | ST elevation myocardial infarction |
| **VF** | Ventricular fibrillation |
| **VT** | Ventricular tachycardia |

# Annex 1

## Vaughan Williams classification of anti-arrhythmic drugs[196,197]

In this system drugs are classified according to their effects on the electrical behaviour of myocardial cells during activity:

- Class Ia, b, c: membrane stabilising drugs (eg quinidine, lidocaine, flecainide respectively)
- Class II: beta blockers
- Class III: amiodarone and sotalol (also Class II)
- Class IV: calcium channel blockers (includes verapamil but not dihydropyridines)

# Annex 2

## NYHA classification of symptoms of heart failure[198]

New York Heart Association classification

| Class | Symptoms |
|---|---|
| I | No limitation: ordinary physical exercise does not cause undue fatigue, dyspnoea or palpitations |
| II | Slight limitation of physical activity: comfortable at rest but ordinary activity results in fatigue, palpitations or dyspnoea |
| III | Marked limitation of physical activity: comfortable at rest but less than ordinary activity results in symptoms |
| IV | Unable to carry out any physical activity without discomfort: symptoms of heart failure are present even at rest with increased discomfort with any physical activity. |

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### PHARMACOLOGICAL THERAPIES FOR RATE CONTROL

A **Ventricular rate in AF should be controlled with beta blockers, rate-limiting calcium channel blockers** *(verapamil or diltiazem)*, **or digoxin.**

C **Digoxin does not control rate effectively during exercise and should be used as first line therapy only in people who are sedentary, or in overt heart failure.**

C **In some people a combination of drugs may be required to control heart rate in atrial fibrillation. Options include the addition of digoxin to either a beta blocker or a rate-limiting calcium channel blocker.**

### NON-PHARMACOLOGICAL THERAPIES

B **Ablation and pacing should be considered for patients with AF who remain severely symptomatic or have LV dysfunction in association with poor rate control or intolerance to rate control medication.**

☑ Patients with atrial fibrillation who are severely symptomatic despite optimum tolerated medical therapy should be referred to a cardiac rhythm specialist for consideration of non-pharmacological therapy, eg radiofrequency ablation.

## VENTRICULAR ARRHYTHMIAS

### REVASCULARISATION FOR SECONDARY PREVENTION OF VT/VF

C **Revascularisation should be considered in patients who have had sustained VT or VF.**

### IMPLANTABLE CARDIOVERTER DEFIBRILLATOR THERAPY – PRIMARY PREVENTION

A **Patients with moderate to severe LV dysfunction** *(eg ejection fraction $<0.35$)*, **in NYHA Class I-III at least one month after myocardial infarction should be considered for ICD therapy.**

B **Patients with spontaneous non-sustained ventricular tachycardia** *(especially if sustained ventricular tachycardia is inducible)*, **severely impaired ejection fraction** *($<0.25$)* **or prolonged QRS complex duration** *($>120$ms)* **should be prioritised for ICD implantation.**

A **Patients meeting criteria for ICD implantation who have prolonged QRS duration** ($>120$ms) **and NYHA class III-IV symptoms should be considered for CRT-D therapy.**

### IMPLANTABLE CARDIOVERTER DEFIBRILLATOR THERAPY – SECONDARY PREVENTION

A **Patients surviving the following ventricular arrhythmias in the absence of acute ischaemia or treatable cause should be considered for ICD implantation:**

- **cardiac arrest** *(VT or VF)*
- **VT with syncope or haemodynamic compromise**
- **VT without syncope if LVEF $<0.35$** *(not NYHA IV)*.

### ANTI-ARRHYTHMIC DRUG THERAPY

A **Class 1 anti-arrhythmic drugs should not be used for treatment of premature ventricular beats or non-sustained VT in patients with previous MI.**

A **Long term beta blockers are recommended for routine use in post-MI patients without contraindications.**

A **Calcium channel blocker therapy is not recommended for reduction in sudden death or all-cause mortality in post-MI patients.**

## ARRHYTHMIAS ASSOCIATED WITH CORONARY ARTERY BYPASS GRAFT SURGERY

### RISK FACTORS

D **In patients undergoing coronary artery bypass graft surgery, age, previous AF and left ventricular ejection fraction should be considered when assessing risk of postoperative arrhythmia.**

### PROPHYLACTIC INTERVENTIONS

#### PHARMACOLOGICAL THERAPIES

A **Amiodarone may be used when prophylaxis for atrial fibrillation and ventricular arrhythmias is indicated following CABG surgery.**

A **Beta blockers including sotalol may be used when prophylaxis for atrial fibrillation is indicated following CABG surgery.**

B **Verapamil and diltiazem may be used for prophylaxis of atrial fibrillation following CABG surgery.**

#### MANIPULATION OF BLOOD ELECTROLYTES

A **Magnesium may be used when prophylaxis for atrial fibrillation and ventricular arrhythmias is indicated following CABG surgery.**

## TREATMENTS FOR ATRIAL FIBRILLATION

### PHARMACOLOGICAL THERAPIES

D
- **Patients with AF and haemodynamic compromise should have synchronised cardioversion.**
- **In the immediate postoperative period, patients with persistent AF without haemodynamic compromise should be treated with rate-limiting therapy.**
- **Patients with persistent AF should be considered for elective synchronised cardioversion.**

## PSYCHOSOCIAL ISSUES

C **Psychosocial implications for people experiencing cardiac arrhythmias should be considered by all healthcare staff throughout assessment, treatment and care.**

☑ Psychosocial support for patients experiencing cardiac arrhythmias should not be restricted to recipients of ICDs.

### PSYCHOSOCIAL ASSESSMENT AND SCREENING

D **Patients with chronic cardiac arrhythmias should be screened for anxiety or depressive disorders with referral to specialist mental health services where appropriate.**

D **Selective cognitive screening should be available especially for post arrest and older cardiac patients experiencing persistent memory or other cognitive difficulties.**

### PSYCHOSOCIAL SUPPORT AND INTERVENTION

D **Psychosocial implications for people experiencing cardiac arrhythmias should be considered by all healthcare staff throughout assessment, treatment and care.**

D **Psychosocial intervention offered as part of a comprehensive rehabilitation programme should encompass a cognitive behavioural component.**

# CARDIAC ARRHYTHMIAS IN CORONARY HEART DISEASE

## ARRHYTHMIAS ASSOCIATED WITH CARDIAC ARREST

### BYSTANDER CARDIOPULMONARY RESUSCITATION

C The number of lay people trained to initiate CPR in out-of-hospital cardiac arrest should be increased.

D Lay people identified as having a high probability of witnessing a cardiac arrest should be offered CPR training.

D CPR should be taught as part of the school curriculum.

### DEFIBRILLATION

B Defibrillation in patients with VF or pulseless VT should be administered without delay for witnessed cardiac arrests and immediately following two minutes of CPR for unwitnessed out-of-hospital cardiac arrests.

C Prompt defibrillation should be available throughout all healthcare facilities.

C All healthcare workers trained in CPR should also be trained, equipped, authorised and encouraged to perform defibrillation.

### AUTOMATED EXTERNAL DEFIBRILLATORS

A Automated external defibrillators should be used by trained first responders, with their use integrated within the emergency medical services system.

B Automated external defibrillators should be sited in locations which have a high probability of a cardiac arrest event.

### ADJUNCTIVE THERAPIES IN THE PERI-ARREST PERIOD

#### REFRACTORY VT/VF

D Intravenous adrenaline/epinephrine should be used for the management of patients with refractory VT/VF.

A Intravenous amiodarone should be considered for the management of refractory VT/VF.

#### SUSTAINED VT (NO CARDIAC ARREST)

D Intravenous amiodarone, procainamide or sotalol should be used in the management of patients with haemodynamically stable VT.

D Patients with polymorphic VT should be treated with intravenous magnesium. QT interval prolonging drugs, if prescribed, should be withdrawn. If present, hypokalaemia should be corrected by potassium infusion and bradycardia by temporary pacing or isoprenaline infusion.

#### ASYSTOLE AND PULSELESS ELECTRICAL ACTIVITY

D Patients with cardiac arrest secondary to asystole or pulseless electrical activity should receive intravenous adrenaline/epinephrine.

### BRADYCARDIA/SINOATRIAL DYSFUNCTION/HEART BLOCK

C Atropine should be used in the treatment of patients with symptomatic bradycardia.

D Temporary transcutaneous pacing should be initiated quickly in patients not responding to atropine.

D When atropine or transcutaneous pacing is ineffective consider adrenaline/epinephrine, dopamine, isoprenaline or aminophylline infusions before transvenous pacing is instituted.

## ARRHYTHMIAS ASSOCIATED WITH ACUTE CORONARY SYNDROMES

### ATRIAL FIBRILLATION

B Class 1C anti-arrhythmic drugs should not be used in patients with AF in the setting of acute MI.

D Patients with AF and haemodynamic compromise should have urgent synchronised DC cardioversion or be considered for anti-arrhythmic and rate-limiting therapy using:

- intravenous amiodarone

  or
- digoxin, particularly in presence of severe LV systolic dysfunction with heart failure.

D Patients with AF with a rapid ventricular response, without haemodynamic compromise but with continuing ischaemia should be treated with one of:

- intravenous beta blockade, in absence of contraindications
- intravenous verapamil where there are contraindications to beta blockade and there is no LV systolic dysfunction
- synchronised DC cardioversion.

D Patients with AF without haemodynamic compromise or ischaemia should be treated with rate-limiting therapy, preferably a beta blocker, and be considered for chemical cardioversion with amiodarone or DC cardioversion.

### CONDUCTION DISTURBANCES AND BRADYCARDIA

D Transvenous temporary pacing should be considered for patients with:

- sinus bradycardia *(heart rate < 40 beats per minute)* associated with symptoms and unresponsive to atropine
- alternating left and right bundle branch block
- Mobitz type II AV block with new bundle branch block
- third degree AV block in inferior MI, if unresponsive to atropine and haemodynamically compromised, and in all cases of anterior MI
- ventricular standstill.

Transcutaneous pacing should be available to all patients with other atrioventricular and intraventricular conduction disturbances.

D Permanent pacing is indicated for patients with persistent Mobitz type II second degree block, or persistent third degree AV block.

D Permanent pacing should be considered for patients who have had transient second degree or third degree AV block with associated bundle branch block.

### VENTRICULAR ARRHYTHMIAS

#### VENTRICULAR ARRHYTHMIAS AND ACUTE MI

C Patients who have primary VF should be recognised as being at increased risk during their hospital stay, and medical therapy should be optimised.

D Patients who have monomorphic VT following acute MI, or VF greater than 48 hours after infarction, should be recognised as being at increased short and long term risk and should be considered for revascularisation and ICD.

#### PREVENTION OF VENTRICULAR ARRHYTHMIAS AND SUDDEN DEATH

A Routine use of anti-arrhythmic drugs is not recommended following MI.

B Patients who have suffered a recent myocardial infarction and with LVEF $\leq 0.40$ and either diabetes or clinical signs of heart failure should receive eplerenone unless contraindicated by the presence of renal impairment or high potassium levels.

#### ASSESSMENT OF RISK OF SUDDEN DEATH

C LV function should be assessed in all patients with acute MI during the index admission.

## ARRHYTHMIAS ASSOCIATED WITH CHRONIC CORONARY HEART DISEASE/LEFT VENTRICULAR DYSFUNCTION

### ATRIAL FIBRILLATION

#### ANTI-ARRHYTHMIC DRUGS

A Amiodarone or sotalol treatment should be considered where prevention of atrial fibrillation recurrence is required on symptomatic grounds.

#### RATE VERSUS RHYTHM CONTROL

A Rate control is the recommended strategy for management of patients with well tolerated atrial fibrillation.

☑ Patients who are haemodynamically compromised, have myocardial ischaemia or are severely symptomatic as a result of AF with a rapid ventricular response should be treated promptly by electrical cardioversion.

☑ Patients with AF who remain severely symptomatic despite adequate rate control should be considered for rhythm control.