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Guidelines on the management of atrial fibrillation (AF)

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These guidelines are based predominantly on:

ESC Guidelines for the management of atrial fibrillation developed in collaboration with EACTS. The Task Force for the management of atrial fibrillation of the European Society of Cardiology (ESC). European Heart Journal (2016) 37, 2893–2962

These guidelines are a summary of the current guidelines and consensus documents of the ESC and are intended for internal use within the SHSCT only.

1.1 Abbreviations:

AF	Atrial fibrillation
ALS	Advanced life support
CAD	Coronary artery disease
CKD	Chronic kidney disease
CVA	Cerebrovascular accident
DCC	Direct current cardioversion
DM	Diabetes mellitus
ECG	Electrocardiogram
FBP	Full blood picture
HF	Heart failure
LFT	Liver function tests
NOAC	Non-vitamin k antagonist oral anticoagulant
OAC	Oral anticoagulant
PAF	Paroxysmal atrial fibrillation
PVI	Pulmonary vein isolation
TFT	Thyroid function tests
TIA	Tranient ischaemic attack
TTE	Transthoracic echocardiogram
TTR	time in therapeutic range
VKA	Vitamin K antagonist
VHD	Valvular heart disease
SCD	Sudden cardiac death
SHSCT	Southern Health and Social Care Trust
TOE	Transoesphageal echocardiography
U+E	Urea and Electrolytes

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1.2 Key messages:

- ✓ Screening for AF in at-risk patients is important to facilitate prompt treatment and reduce the risk of ischaemic stroke
- ✓ Suboptimal use of oral anticoagulants for stroke prevention has been widely reported from observational studies
- ✓ OAC is recommended if CHA2DS2VASC of 2 or above, should be considered if 1 or above (exception = young females)
- ✓ NOACs are associated with a lower risk of stroke and reduced risk of major bleeding versus warfarin
- ✓ NOACs are not recommended in mechanical heart valves or rheumatic mitral stenosis
- ✓ Appropriate dosing is key; if a patient is not appropriately anticoagulated this can lead to an increased risk of stroke
- ✓ HAS-BLED score should not be used as a reason not to anticoagulate
- ✓ Consider switching from VKA to NOAC in certain circumstances
- ✓ Rhythm control therapy is indicated for symptom improvement in patients with AF.
- ✓ Do not use rhythm control therapy in asymptomatic AF patients, nor in patients with permanent AF
- ✓ With the exception of amiodarone, oral antiarrhythmic therapy should not be used in patients with concomitant heart failure or ischaemic heart disease
- ✓ In general, patients presenting with presumed first presentation of documented AF should only be cardioverted if they are deemed haemodynamically unstable
- ✓ Immediate initiation of anticoagulation is important in all patients scheduled for cardioversion. Patients should start OAC at least 3 weeks before cardioversion and continue it for 4 weeks afterwards (in patients without a need for long-term anticoagulation). OAC should be continued indefinitely in patients at risk of stroke.
- ✓ For cardioversion of AF/atrial flutter, effective anticoagulation is recommended for a minimum of 3 weeks before cardioversion.

- ✓ Do not perform cardioversion or catheter ablation without anticoagulation, unless an atrial thrombus has been ruled out transoesophageal echocardiogram.

- ✓ NOACs (apixaban, dabigatran, edoxaban, and rivaroxaban) are not recommended in patients with mechanical heart valves or moderate to severe rheumatic mitral stenosis.

- ✓ Renal function should be evaluated before initiating a NOAC and re-checked at least yearly, and more regularly in patients who may have compromised renal function such as elderly or frail patients. Dabigatran is contraindicated when CrCl <30mls/min and factor Xa inhibitors are contraindicated when CrCl is <15mls/min.

2. Introduction

Despite good progress in the management of patients with atrial fibrillation (AF), this arrhythmia remains one of the major causes of stroke, heart failure (HF), sudden cardiac death (SCD), and cardiovascular morbidity in the world. Furthermore, the number of patients with AF is predicted to rise steeply in the coming years. To meet the growing demand for effective care of patients with AF, new information is continually generated and published, and the last few years have seen substantial progress.

Estimates suggest an AF prevalence of approximately 3% in adults aged 20 years or older with greater prevalence in older persons and in patients with conditions such as hypertension, HF, coronary artery disease (CAD), valvular heart disease (VHD), obesity, diabetes mellitus (DM), or chronic kidney disease (CKD).

The increase in AF prevalence can be attributed both to better detection of silent AF, alongside increasing age and conditions predisposing to AF. AF is independently associated with a two-fold increased risk of all-cause mortality in women and a 1.5-fold increase in men. Death due to stroke can largely be mitigated by anticoagulation, while other cardiovascular deaths, for example due to HF and SCD, remain common even in AF patients treated according to the current evidence base.

AF is also associated with increased morbidity, such as HF and stroke. Despite these advances, substantial morbidity remains. Oral anticoagulation (OAC) with vitamin K antagonists (VKAs) or non- VKA oral anticoagulants (NOACs) markedly reduces stroke and mortality in AF patients.

Other interventions such as rate control and rhythm control improve AF-related symptoms and may preserve cardiac function, but have not demonstrated a reduction in long-term morbidity or mortality.

3. Purpose of this policy

This policy aims to assist the attending health care professionals in treating patients with AF in both the acute and chronic setting.

4. Scope

This document provides guidance for any professional involved in the clinical management of patients presenting to either primary or secondary care with AF. This will include:

- Consultants
- SAS doctors
- SpRs
- Junior Doctors
- Specialist Nurses
- Nursing Staff
- General Practitioners

5. Diagnosis of atrial fibrillation

AF and its associated symptoms of dyspnoea, palpitations, dizziness and chest discomfort are very common presenting complaints to the emergency department and/or acute medical unit. Assessment of these patients with AF should include clinical evaluation, 12 lead electrocardiogram (ECG) and echocardiographic evaluation. Prompt pharmacological therapy is the mainstay of treatment in this group of patients to minimise associated mortality and morbidity. Further long term evidence based drug therapy reduces future cardiovascular morbidity.

External stressors such as structural heart disease, hypertension, possibly DM, but also AF itself induce a slow but progressive process of structural remodelling in the atria. Structural remodelling results in electrical dissociation between muscle bundles and local conduction heterogeneities, favouring re-entry and perpetuation of the arrhythmia. In many patients, the structural remodelling process occurs before the onset of AF. As some of the structural remodelling will be irreversible, early initiation of treatment seems desirable.

5.1. Clinical Classification of AF

The diagnosis of AF requires rhythm documentation using an ECG showing the typical pattern of AF:

- Absolutely irregular RR intervals and no discernible, distinct P waves.

By accepted convention, an episode lasting at least 30 seconds is diagnostic. Individuals with AF may be symptomatic or asymptomatic ('silent AF'). Many AF patients have both symptomatic and asymptomatic episodes of AF. Silent, undetected AF is common with severe consequences such as stroke and death. Prompt recording of an ECG is an effective and cost-effective method to document chronic forms of AF. The technology to detect paroxysmal, self-terminating AF episodes is rapidly evolving such as smartphone cases with ECG electrodes, smart watches, 'AliveCor devices' and blood pressure machines with AF detection algorithms, although these have not yet been formally evaluated against an established arrhythmia detection method.

In many patients, AF progresses from short, infrequent episodes to longer and more frequent attacks. Over time, many patients will develop sustained forms of AF. In a small proportion of patients, AF will remain paroxysmal over several decades (2–3% of AF patients). The distribution of paroxysmal AF recurrences is not random, but clustered. AF may also regress from persistent to paroxysmal AF. Furthermore, asymptomatic recurrences of AF are common in patients with symptomatic AF. Based on the presentation, duration, and spontaneous termination of AF episodes, five types of AF are traditionally distinguished: first diagnosed, paroxysmal, persistent, long-standing persistent, and permanent AF (see table 1):

Table 1: AF patterns

AF pattern	Definition
First diagnosed AF	AF that has not been diagnosed before, irrespective of the duration of the arrhythmia or the presence and severity of AF-related symptoms.
Paroxysmal AF	Self-terminating, in most cases within 48 hours. Some AF paroxysms may continue for up to 7 days.[a] AF episodes that are cardioverted within 7 days should be considered paroxysmal.[a]
Persistent AF	AF that lasts longer than 7 days, including episodes that are terminated by cardioversion, either with drugs or by direct current cardioversion, after 7 days or more.
Long-standing persistent AF	Continuous AF lasting for ≥1 year when it is decided to adopt a rhythm control strategy.
Permanent AF	AF that is accepted by the patient (and physician). Hence, rhythm control interventions are, by definition, not pursued in patients with permanent AF. Should a rhythm control strategy be adopted, the arrhythmia would be re-classified as 'long-standing persistent AF'.

ESC Guidelines for the management of atrial fibrillation developed in collaboration with EACTS. The Task Force for the management of atrial fibrillation of the European Society of Cardiology (ESC). European Heart Journal (2016) 37, 2893–2962

5.2 Symptoms

Patients with AF have significantly poorer quality of life than healthy controls, experiencing a variety of symptoms including lethargy, palpitations, dyspnoea, dizziness, chest tightness, sleeping difficulties, and psychosocial distress. Improved quality of life has been noted with both pharmacological and interventional therapies, but there are limited data to compare the benefit of different treatments.

6. Treatment of AF

6.1 General management of AF patients

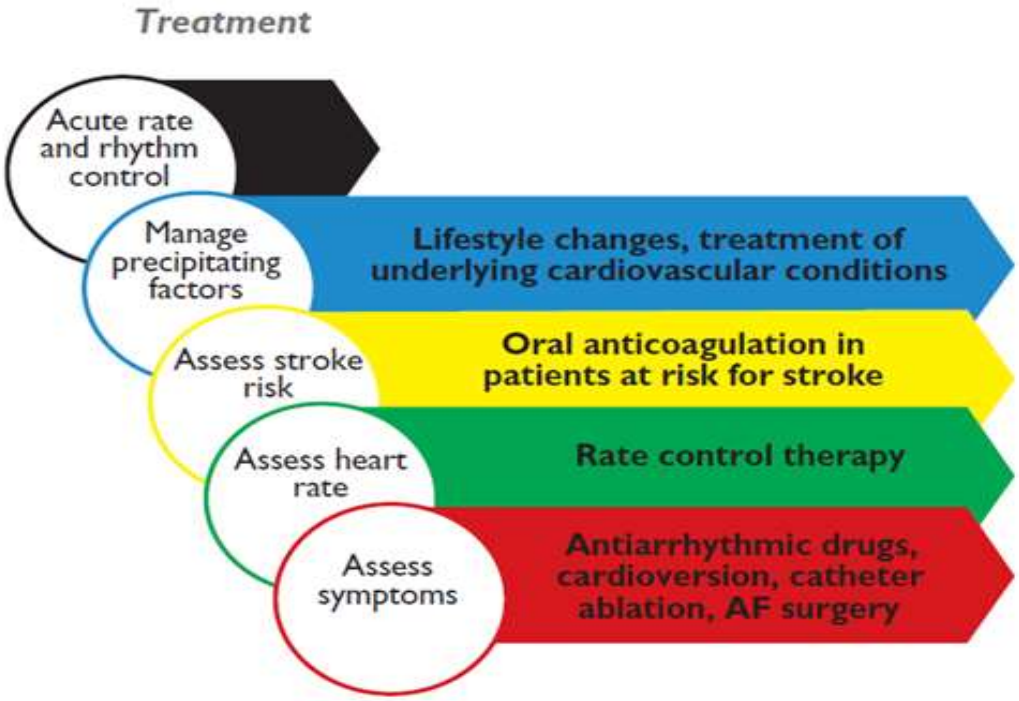
Tailored patient education is recommended in all phases of AF management to support patients' perception of AF and to improve management. A full cardiovascular evaluation, including an accurate history, careful clinical examination, and assessment of concomitant conditions, is recommended in all AF patients. Transthoracic echocardiography (TTE) is recommended in all AF patients to guide management. The assessment of kidney function by serum creatinine or creatinine clearance is recommended in all AF patients to detect kidney disease and to support correct dosing of AF therapy.

All patients with unstable AF should have cardiac monitoring. ECG analysis should be performed in all patients also. Baseline investigations include:

- Bloods: full blood picture (FBP), urea and electrolytes (U+E), liver function tests (LFT), coagulation studies and thyroid function tests (TFT).
- ECG: loss of regular p wave activity with no discernible p waves and an irregular R-R interval
- Echocardiogram: assessing LV systolic function, valvular function, left atrial size, cardiomyopathy assessment, assess for structural heart disease, assess for myocarditis especially if in a young patient presentation.

Figure 1 gives an overview of AF treatment including rate control strategy, stroke risk assessment and rhythm assessment.

Figure 1: Summary of AF treatment



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6.2 Rate control strategy

Beta-blockers (starting dose Bisoprolol 2.5 mg with dose adjustment to control rate) and / or digoxin (especially if respiratory disease were beta-blockers are contraindicated) are recommended to control heart rate in AF patients. Bisoprolol 1.25mg can be considered if patients are elderly, frail or have underlying bradycardia. A target heart rate of <110bpm is advised. Diltiazem or verapamil can also be used to control heart rate in AF patients with LVEF ≥40%. Amiodarone is an option for patients who are unstable (see figure 2 and 3).

In patients with permanent AF (i.e. where no attempt to restore sinus rhythm is planned), antiarrhythmic drugs should not routinely be used for rate control.

Figure 2: Acute heart rate control of AF

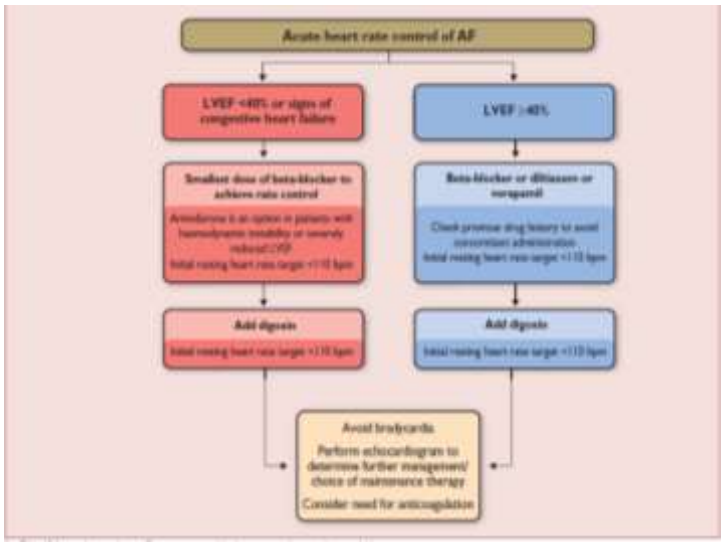

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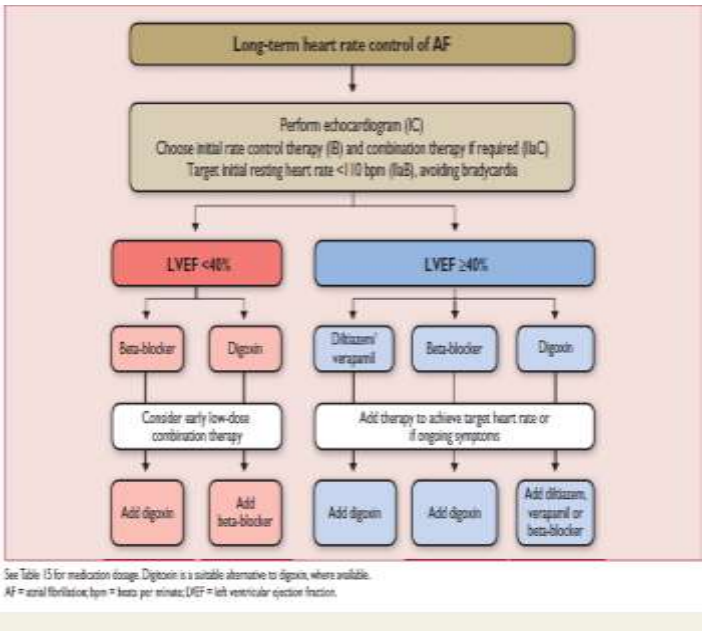
Figure 3: Long-term heart rate control of AF



See Table 15 for medication dosage. Digitoxin is a suitable alternative to digoxin, where available.
AF = atrial fibrillation; bpm = beats per minute; LVEF = left ventricular ejection fraction.

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Catheter ablation

Catheter ablation / pulmonary vein isolation (PVI) of symptomatic paroxysmal AF is recommended to improve AF symptoms in patients who have recurrent symptomatic AF despite rhythm control therapy without structural heart disease.

Atrioventricular node ablation and pacing

Ablation of the atrioventricular node/His bundle and implantation of a VVI pacemaker can control ventricular rate when medications fail to control rate and symptoms. AV nodal ablation renders patients pacemaker-dependent for the rest of their lives, limiting AV nodal ablation and pacing to patients whose symptoms cannot be managed by rate-controlling medication or by reasonable rhythm control interventions. The choice of pacing therapy (right ventricular or biventricular pacing with or without an implantable defibrillator) will depend on individual patient characteristics, including LV function.

Management of typical atrial flutter with ablation of the cavotricuspid isthmus is recommended for patients failing antiarrhythmic drug therapy.

6.3 Rhythm control strategy

Cardioversion of AF (either electrical or pharmacological) is recommended in symptomatic patients with persistent or long-standing persistent AF as part of rhythm control therapy.

Rhythm control therapy is indicated for ***symptom improvement*** in patients with AF.

For paroxysmal AF choose an antiarrhythmic drug in consultation with a cardiologist. For persistent AF refer for direct current cardioversion (DCC). In permanent AF, no further rhythm strategy is planned.

Initiation of long-term therapy to improve symptoms is summarised in figure 4 and figure 5. Drug types and dosage is summarised in table 2.

Pharmacological cardioversion

Antiarrhythmic drugs can restore sinus rhythm in patients with AF. Pharmacological cardioversion restores sinus rhythm in approximately 50% of patients with recent-onset AF.

In the short-term, electrical cardioversion restores sinus rhythm quicker and more effectively than pharmacological cardioversion and is associated with shorter hospitalization.

The aim of antiarrhythmic drug therapy is improvement in AF-related symptoms. Hence, the decision to initiate long-term antiarrhythmic drug therapy needs to balance symptom burden, possible adverse drug reactions and patient preferences.

Flecainide and propafenone

Flecainide and propafenone are effective for pharmacological cardioversion, but their use is restricted to patients without structural heart disease. They should only be used in patients without significant ischaemic heart disease or heart failure to avoid the risk of life-threatening ventricular arrhythmias. High ventricular rates resulting from the conversion of AF into atrial flutter with 1:1 conduction by flecainide or propafenone can be prevented by pre-administering a beta-blocker, verapamil, or diltiazem.

Sotalol

Sotalol is also effective for pharmacological cardioversion. It can also not be used in patients with significant ischaemic heart disease or heart failure to avoid the risk of life-threatening ventricular arrhythmias. Sotalol has beta-blocker effects therefore co-administration with other beta-blockers is not recommended.

Amiodarone

Amiodarone is an effective multichannel blocker, reduces ventricular rate, and may be used in patients with heart failure and ischaemic heart disease but can be associated with long term toxicity.
Monitoring and follow up should be undertaken according to the Northern Ireland Formulary niformulary.hscni.net/formulary regional shared care guidelines www.ipnsm.hscni.net/download/shared_care_guidelines/AmiodaroneSCGJun2019.pdf

Dronedarone

Dronedarone can be used to maintain sinus rhythm, reduces ventricular rate, and prevent cardiovascular hospitalizations and cardiovascular death in patients with paroxysmal or persistent AF or flutter who have at least one relevant cardiovascular comorbidity. Dronedarone has been associated with increase mortality in patients with recently decompensated heart failure and in patients with permanent AF in whom sinus rhythm is not restored. Its use should therefore be limited to an experienced cardiologist.
Monitoring and follow up should be undertaken according to the Northern Ireland Formulary niformulary.hscni.net/formulary regional shared care guidelines www.ipnsm.hscni.net/download/shared_care_guidelines/DronedaroneSCG2017.pdf

Do not use rhythm control therapy in asymptomatic AF patients, nor in patients with permanent AF.

Figure 4: Initiation of long-term rhythm control therapy to improve AF symptoms

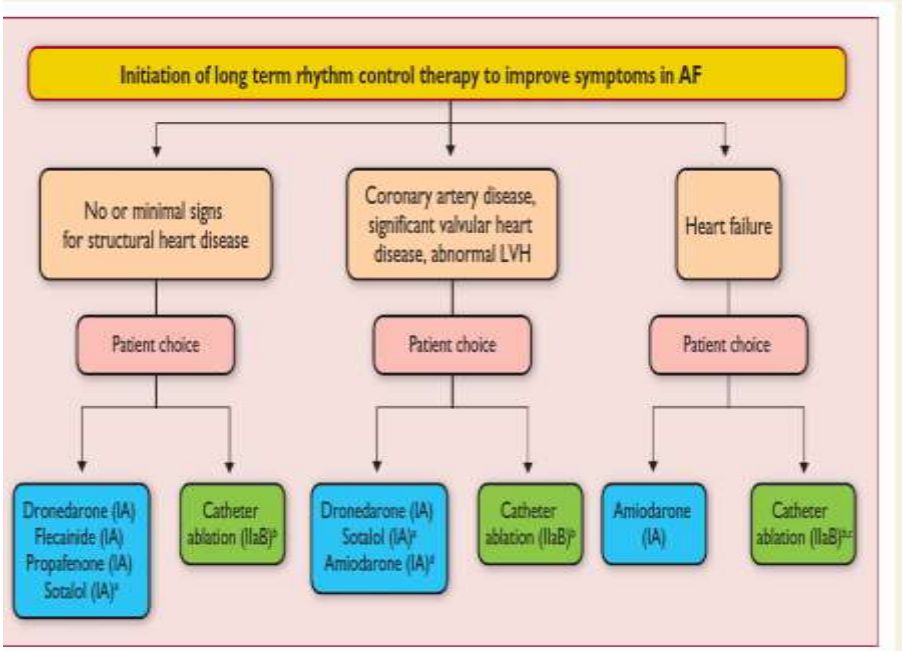

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Table 2: Drug types and dosage

Drug	Dose	Main contraindication
Amiodarone	600mg in divided doses for 4 weeks, 400mg for 4 weeks, then 200mg once daily	Caution when using concomitant therapy with QT-prolonging drugs and in patients with SA or AV node conduction disease. The dose of VKA and of digoxin should be reduced. Increased risk of myopathy with statins Caution in pre-existing liver disease
Dronedarone	400mg twice daily	Contra-indicated in NYHA III or IV or unstable heart failure, during concomitant therapy with QT-prolonging drugs, or powerful CYP3A4 inhibitors (e.g. verapamil, diltiazem, azole antifungal agents) and when CrCl <30mls/min. The dose of beta blockers should be reduced. **Digoxin should be avoided.** Caution in pre-existing liver disease. Elevations in serum creatinine of 0.1-0.2mg/dL are common and do not reflect a decline in renal function.
Flecanide	100-150mg twice daily	Contra-indicated if CrCl<50mg/ml, liver disease, IHD or reduced LV ejection fraction. Caution in the presence of SA or AV conduction system disease. CYP2D6 inhibitors (e.g. fluoxetine or tricyclic antidepressants) increase plasma concentration.
Propafenone	150-300mg three times daily	Contra-indicated in IHD or reduced LV ejection fraction. Caution in the presence of SA or AV node conduction disease. Caution in renal or liver disease and asthma.
Sotalol	80-160mg twice daily	Contra-indicated in the presence of significant LV hypertrophy, systolic heart failure, asthma, pre-existing QT prolongation, hypokalaemia, CrCl <50mls/min. Moderate renal dysfunction requires careful adaptation of dose.

Adapted from: ESC Guidelines for the management of atrial fibrillation developed in collaboration with EACTS. The Task Force for the management of atrial fibrillation of the European Society of Cardiology (ESC). European Heart Journal (2016) 37, 2893–2962.

Anti-arrhythmic therapy should only be commenced in consultation with a cardiologist

Pill in pocket

In selected patients with infrequent symptomatic episodes of paroxysmal AF, a single bolus of oral flecanide (200–300 mg) or propafenone (450–600 mg) can be self-administered by the patient at home ('pill in the pocket' therapy) to restore sinus rhythm, after safety has been established in the hospital setting.

Electrical cardioversion

Synchronized direct current cardioversion (DCC) quickly and effectively converts AF to sinus rhythm, and is the method of choice in severely haemodynamically compromised patients with new-onset AF (see figure 5).

Electrical cardioversion can be performed safely in sedated patients treated with intravenous midazolam and / or fentanyl (see safe sedation protocol).

Pre-treatment with amiodarone (requiring a few weeks of therapy) can improve the efficacy of electrical cardioversion, and similar effects are likely for flecanide, propafenone or dronedarone.

When antiarrhythmic drug therapy is planned to maintain sinus rhythm after cardioversion, it seems prudent to start therapy 1–3 days before cardioversion (Amiodarone: a few weeks) to promote pharmacological conversion and to achieve effective drug levels

Cardioversion carries an inherent risk of stroke in non-anticoagulated patients which is reduced substantially by the administration of anticoagulation. Immediate initiation of anticoagulation is important in all patients scheduled for cardioversion.

For cardioversion of AF/atrial flutter, effective anticoagulation is recommended for a minimum of 3 weeks before cardioversion.

Patients should start OAC **at least 3 weeks before cardioversion** and continue it for **4 weeks afterwards** (in patients without a need for long-term anticoagulation). OAC should be continued indefinitely in patients at risk of stroke.

Do not perform cardioversion or catheter ablation without anticoagulation, unless an atrial thrombus has been ruled out transoesophageal echocardiogram (TOE).

In general, patients presenting with presumed **first presentation of documented AF** should only be cardioverted if they are deemed haemodynamically unstable (ongoing chest pain / dyspnoea, hypotensive <90/60mmHg). If this is the case then please follow advanced life support (ALS) guidelines.

When early cardioversion is desired, TOE can exclude the majority of left atrial thrombi as an alternative to preprocedural anticoagulation when early cardioversion is planned.

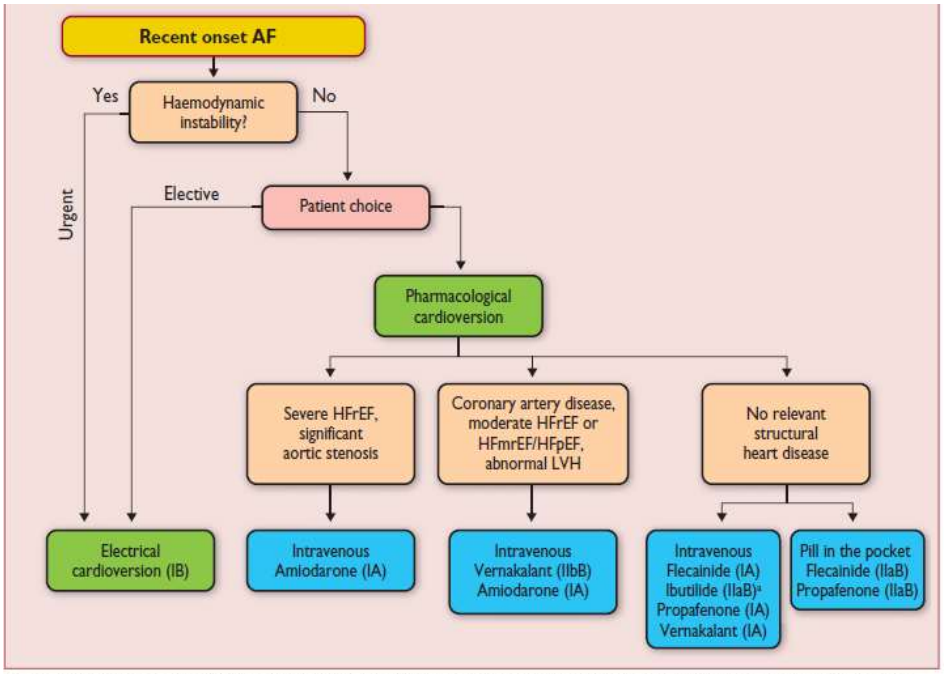
In all other patients, the decision to cardiovert a patient (e.g. in the emergency department) **should be discussed with a cardiologist** to determine the risk of a stroke / complications. It is usually safer to anticoagulate a patient as above and bring them back for a scheduled DCC after at least 3 weeks of full OAC compliance.

If a patient with known AF is taking anticoagulation and has been fully compliant with anticoagulation for at least 3 weeks then it is acceptable to proceed with DCC after discussing with a cardiologist.

ALL DCC decisions should be in consultation with a cardiologist. It is no longer safe to presume a patient is presenting within 48 hours onset as much AF is subclinical and asymptomatic therefore there is a risk of LAA thrombus which could embolise and result in an ischaemic CVA.

The SHSCT DCC referral form is available in appendix 2.

Figure 5: Treatment of recent onset AF.



AF = atrial fibrillation; HFmrEF = heart failure with mid-range ejection fraction; HFpEF = heart failure with preserved ejection fraction; HFrEF = heart failure with reduced ejection fraction; LVH = left ventricular hypertrophy.
[a]Ibutilide should not be used in patients with long QT interval.

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6.4 Anticoagulation strategy

OAC therapy can prevent the majority of ischaemic strokes in AF patients and can prolong life. It is superior to no treatment or aspirin in patients with different profiles for stroke risk. The net clinical benefit is almost universal, with the exception of patients at very low stroke risk, and OAC should therefore be used in most patients with AF. Despite this evidence, underuse or premature termination of OAC therapy is still common. Bleeding events, both severe and nuisance bleeds, a perceived 'high risk of bleeding' on anticoagulation, and the efforts required to monitor and dose-adjust VKA therapy are among the most common reasons for withholding or stopping OAC. However, the considerable stroke risk without OAC often exceeds the bleeding risk on OAC, even in the elderly, in patients with cognitive dysfunction, or in patients with frequent falls or frailty.

Vitamin K antagonist (VKA)

Warfarin and other VKAs were the first anticoagulants used in AF patients. VKA therapy reduces the risk of stroke by two-thirds and mortality by one-quarter compared with control (aspirin or no therapy). The use of VKAs is limited by the narrow therapeutic interval necessitating frequent monitoring/dose adjustment, and multiple interactions with drugs and foods but VKAs, when delivered with adequate time in therapeutic range (TTR), are effective for stroke prevention in AF patients. VKAs are currently the only treatment with established safety in AF patients with moderate to severe rheumatic mitral valve disease and/or a mechanical heart valve prosthesis. Limitations of warfarin is summarised in table 3.

Table 3: Limitation of warfarin

Limitation	Possible consequence
▶ Narrow therapeutic window (INR range 2–3)[1]	▶ Increased risk of stroke (INR <2.0) ▶ Increased risk of bleeding (INR >3.0)
▶ Considerable variability in dose–response (genetic variations)[1] ▶ Interactions with drugs, alcohol and diet[1,2]	▶ Frequent monitoring of INR required ▶ Frequent dose adjustments required
▶ Long half-life[1] ▶ Slow onset and offset of action[1,3]	▶ Issue in perioperative anticoagulation (bridging)[3]

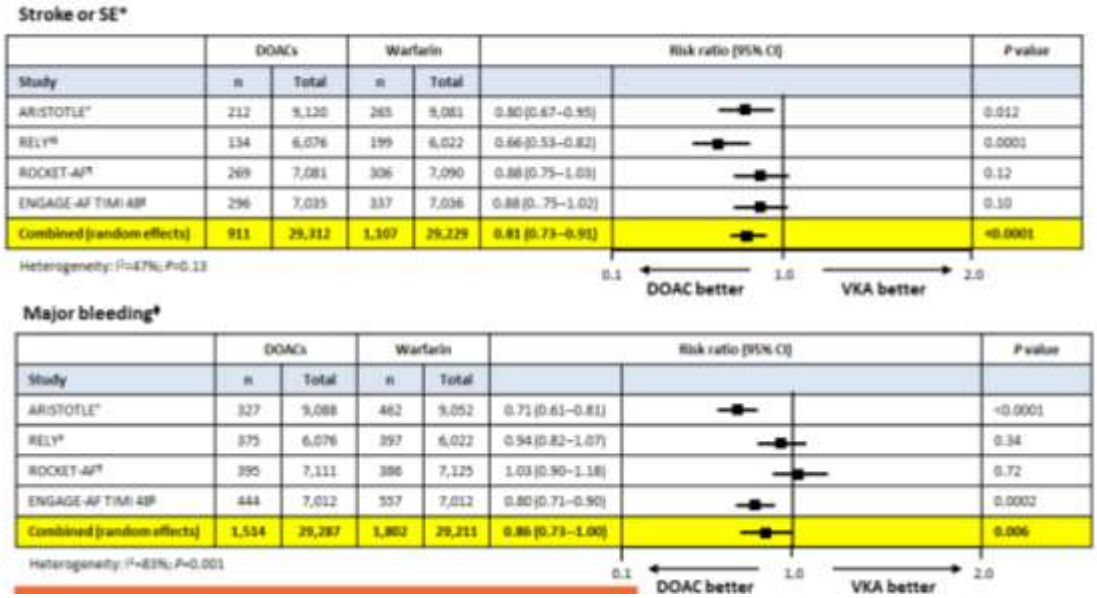
NOACs

NOACs, including the direct thrombin inhibitor dabigatran (RE-LY trial) and the factor Xa inhibitors apixaban (ARISTOTLE trial), edoxaban (ENGAGE-AF trial), and rivaroxaban (ROCKET AF trial), are suitable alternatives to VKAs for stroke prevention in AF. Their use in clinical practice is increasing rapidly. All NOACs have a predictable effect (onset and offset) without need for regular anticoagulation monitoring. The phase III trials

have been conducted with carefully selected doses of the NOACs, including clear rules for dose reduction that should be followed in clinical practice.

A comparison of the main NOACs and warfarin with regards to efficacy and safety is shown in figure 6.

Figure 6: Comparison of efficacy and safety of NOACs versus warfarin.



Ruff CT, et al. Lancet (2014);383(9921):955–962.

It is recommended to estimate stroke risk in AF patients based on the CHA2DS2-VASc score (table 4). In general, patients without clinical stroke risk factors do not need antithrombotic therapy, while patients with stroke risk factors are likely to benefit from OAC. There is now a growing evidence base regarding stroke risk in patients with one clinical risk factor.

Therefore OAC should be considered CHA2DS2-VASc ≥1 (excluding young females who only score for gender alone), balancing the expected stroke reduction, bleeding risk, and patient preference (figure 7).

Table 4: CHA2DS2-VASc score

CHA_2DS_2-VASc score

CHA_2DS_2-VASc risk factor	Points
Congestive heart failure Signs/symptoms of heart failure or objective evidence of reduced left ventricular ejection fraction	+1
Hypertension Resting blood pressure >140/90 mmHg on at least two occasions or current antihypertensive treatment	+1
Age 75 years or older	+2
Diabetes mellitus Fasting glucose >125 mg/dL (7 mmol/L) or treatment with oral hypoglycaemic agent and/or insulin	+1
Previous stroke, transient ischaemic attack, or thromboembolism	+2
Vascular disease Previous myocardial infarction, peripheral artery disease, or aortic plaque	+1
Age 65–74 years	+1
Sex category (female)	+1

CHA_2DS_2-VASc = Congestive Heart failure, hypertension, Age ≥75 (doubled), Diabetes, Stroke (doubled), Vascular disease, Age 65–74, and Sex (female).

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NOACs are the anticoagulant of choice in most cases of AF. NOACs should not be used in certain circumstances including mechanical valves, moderate to severe rheumatic mitral stenosis, creatinine clearance <15mls/min (<30mls/min for dabigatran), pregnancy, intolerance, and allergy. Caution is advised although the evidence is evolving in patients weighing >120kg and in oncology patients undergoing chemotherapy/radiotherapy. Examples of NOACs and their properties are shown in table 5.

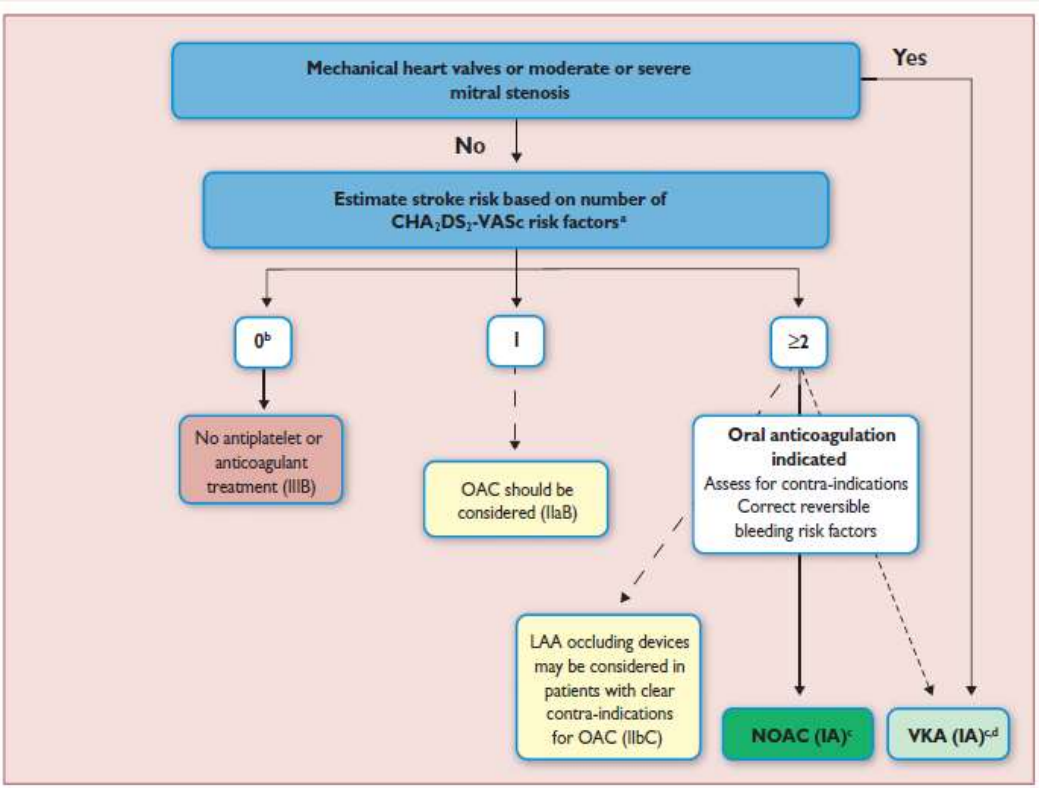
When patients are treated with a vitamin K antagonist, time in therapeutic range (TTR) should be kept as high as possible (minimum target TTR>65%) and closely monitored.

For patients with atrial flutter, antithrombotic therapy is recommended according to the same risk profile used for AF.

Table 5: Choice of anticoagulant

	Warfarin[1]	Apixaban[2,3]	Dabigatran[2,4]	Rivaroxaban[2,5]	Edoxaban[6]
Mechanism of action	Inhibitor of vitamin K-dependent factors	Direct factor Xa inhibitor	Direct thrombin inhibitor	Direct factor Xa inhibitor	Direct factor Xa inhibitor
Oral bioavailability	>95%	~50%	~6.5%	80–100%	~62%
Pro-drug	No	No	Yes	No	No
Food effect	Yes (foods high in vitamin K may affect anticoagulation effect)	No	No	Yes (20 mg and 15 mg doses need to be taken with food)	No
Renal clearance	8%	~27%	85%	~33%*	50%*
Mean half-life ($t_{1/2}$)	40 hours	12 hours	12–18 hours[4]	5–9 hours (young) 11–13 hours (elderly)	10–14 hours
T_{max}	Within 4 hours, but peak anticoagulation effect may require 72–96 hours[5]	3–4 hours	0.5–2 hours	2–4 hours	1–2 hours

Figure 7: Anticoagulation based on CHA2DS2-VASc score



ESC Guidelines for the management of atrial fibrillation developed in collaboration with EACTS. The Task Force for the management of atrial fibrillation of the European Society of Cardiology (ESC). European Heart Journal (2016) 37, 2893–2962

Bleeding risk

Several bleeding risk scores have been developed, mainly in patients on VKAs. These include HAS-BLED [(hypertension, abnormal renal/liver function (1 point each), stroke, bleeding history or predisposition, labile INR, elderly (65 years), drugs/alcohol concomitantly (1 point each)]. A high bleeding risk score should generally not result in withholding OAC. Rather, bleeding risk factors should be identified and treatable factors corrected, see table 6 and figure 8.

Table 6: HASBLED score with modifiable and non-modifiable bleeding risk factors

Modifiable bleeding risk factors
Hypertension (especially when systolic blood pressure is >160 mmHg)[a,b,c]
Labile INR or time in therapeutic range <60%[a] in patients on vitamin K antagonists
Medication predisposing to bleeding, such as antiplatelet drugs and non-steroidal anti-inflammatory drugs[a,d]
Excess alcohol (≥8 drinks/week)[a,b]
Potentially modifiable bleeding risk factors
Anaemia[b,c,d]
Impaired renal function[a,b,c,d]
Impaired liver function[a,b]
Reduced platelet count or function[b]
Non-modifiable bleeding risk factors
Age[c] (>65 years)[a] (≥75 years)[b,c,d]
History of major bleeding[a,b,c,d]
Previous stroke[a,b]
Dialysis-dependent kidney disease or renal transplant[a,c]
Cirrhotic liver disease[a]
Malignancy[b]
Genetic factors[b]
Biomarker-based bleeding risk factors
High-sensitivity troponin[e]
Growth differentiation factor-15[e]
Serum creatinine/estimated CrCl[e]

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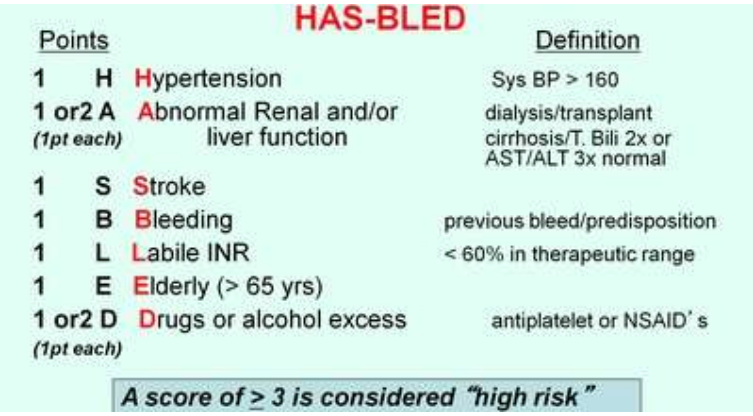
Figure 8: HAS-BLED score



High HAS-BLED scores are not absolute contra-indications to oral anticoagulation but highlights risk factors that may be modified

6.5 Left atrial appendage closure

The most common justification for LAA occlusion devices in clinical practice is a perceived high bleeding risk and, less often, contraindications for OAC. Interventional LAA occlusion and limited experience with percutaneous LAA ligation, has mainly been reported in observational studies and registries. Only one device (Watchman) has been compared with VKA therapy in randomized trials (PROTECT AF and PREVAIL).

6.6 Acute presentation of AF to the emergency department (ED)

Patients presenting to the acute emergency department with AF with unstable symptoms (haemodynamically unstable with ongoing chest pain / dyspnoea, hypotensive <90/60mmHg) should be considered for cardioversion as per ALS guidance. Otherwise, patients should be rate controlled, commenced on anticoagulation if indicated and considered for OP DCC (figure 9). Appendix 3 highlights a useful flowchart for new AF presentations.

Criteria for admission to acute cardiology:

Admit to Cardiology if:

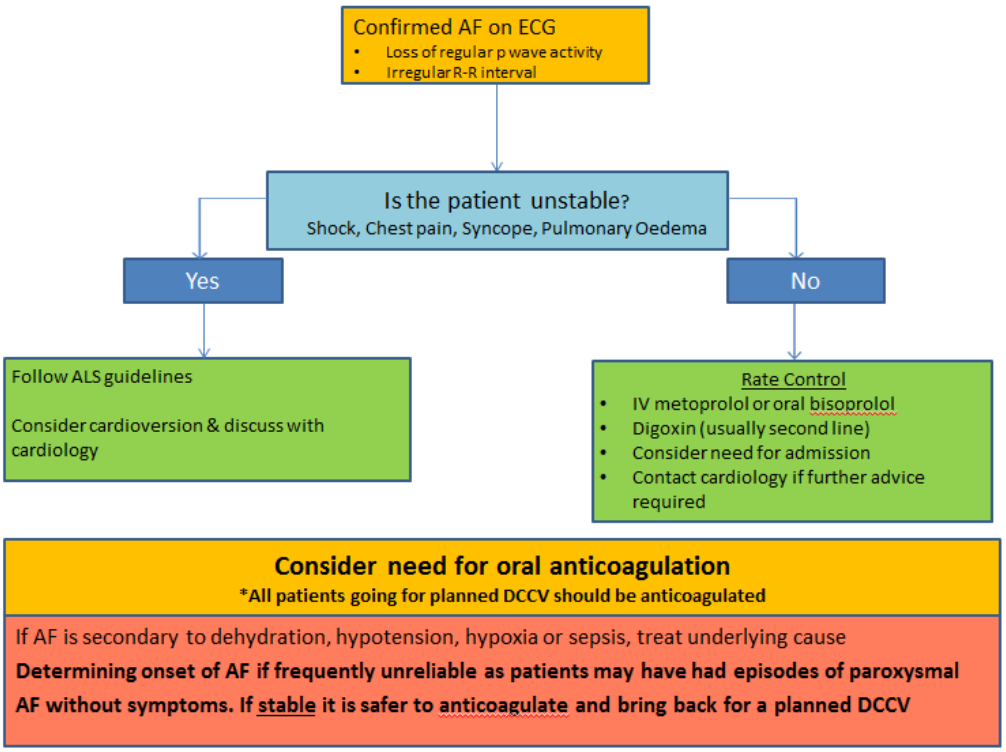
- Fast ventricular response >110 bpm at rest and symptomatic following trial of beta blocker
- Unstable or very symptomatic
- Signs of acute heart failure

Consider discharge home if:

- Rate controlled
- Stable and asymptomatic
- Consider need for anticoagulation before discharge
- Referral to OP cardiology/Arrhythmia clinic/Direct to OP DCC

If there is an underlying primary illness (i.e. LRTI, UTI, sepsis) causing fast AF refer to the appropriate specialty for treatment of that condition. Cardiology will provide input if required.

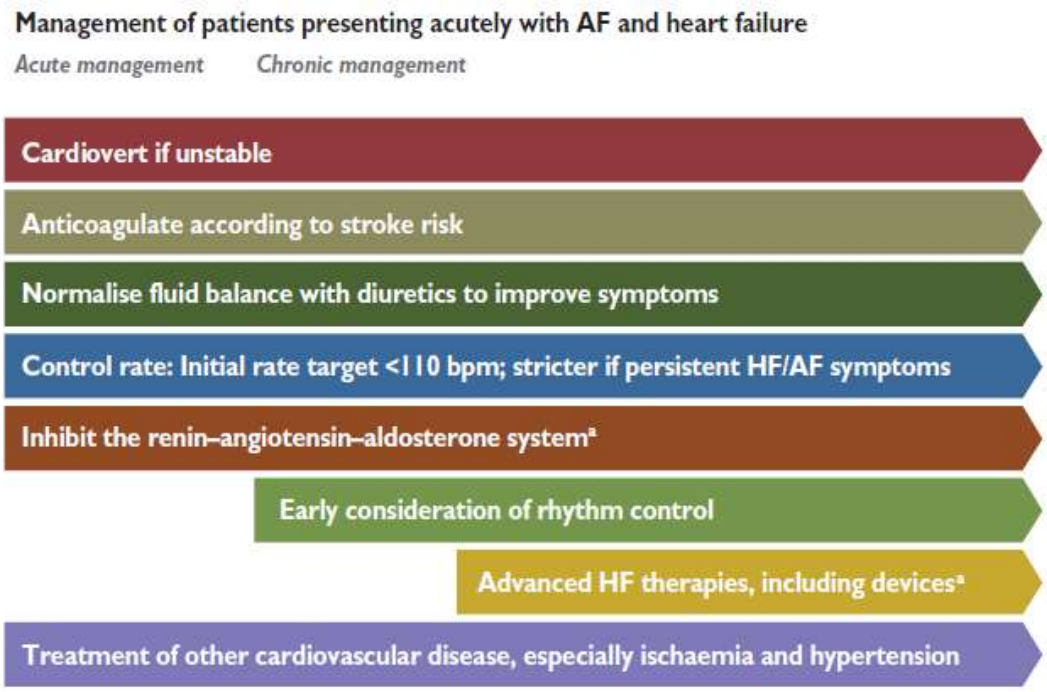
Figure 9: SHSCT ED AF flowchart



6.7 Acute HF presentation of AF to the emergency department

Patients presenting with acute HF and AF should also be considered for DCC. Further therpay can include intravenous diuretics, rate control, rhtyhm control, pharmacological HF therapy and / or device therapy work-up (figure 10).

Figure 10: AF presenting with acute heart failure



ESC Guidelines for the management of atrial fibrillation developed in collaboration with EACTS. The Task Force for the management of atrial fibrillation of the European Society of Cardiology (ESC). European Heart Journal (2016) 37, 2893–2962

7. Special circumstances and considerations in AF

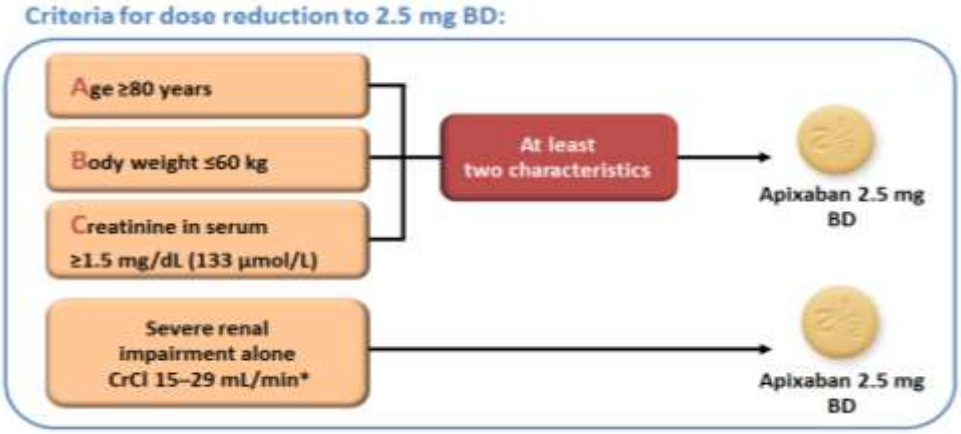
7.1 Commencing NOAC therapy in AF patients with CKD

Before commencing OAC, all patients should have a creatinine clearance calculated by Cockcroft Gault formula. Table 7 and figure 11 highlights renal prescribing guidance in CKD patients.

Table 7: NOAC prescribing in CKD including dose reduction fo Apixaban

CrCl	Apixaban[1,2] (5 mg BD)*	Dabigatran[1,3] (150 mg BD)†	Rivaroxaban[1,4] (20 mg OD)‡	Edoxaban[1,5] (60 mg OD)§
Normal renal function (>80 mL/min)	No dose adjustment necessary unless patient meets other criteria for dose reduction*	No dose adjustment necessary unless patient meets other criteria for dose reduction†	No dose adjustment necessary	No dose adjustment necessary unless patient meets other criteria for dose reduction§
Mild renal impairment (CrCl 50–80 mL/min)[3] (CrCl 51–80 mL/min)[5]				
Moderate renal impairment (CrCl 30–49 mL/min)[4] (CrCl 30–50 mL/min)[3,5]		Reduce to 110 mg BD if high risk of bleeding	Reduce to 15 mg OD‡	Reduce to 30 mg OD§
Severe renal impairment (CrCl 15–29 mL/min)	Reduce to 2.5 mg BD*	Contraindicated		
End-stage renal disease (CrCl <15 mL/min or undergoing dialysis)	Not recommended	Contraindicated	Not recommended	Not recommended

Figure 11: Apixaban dose reduction in CKD

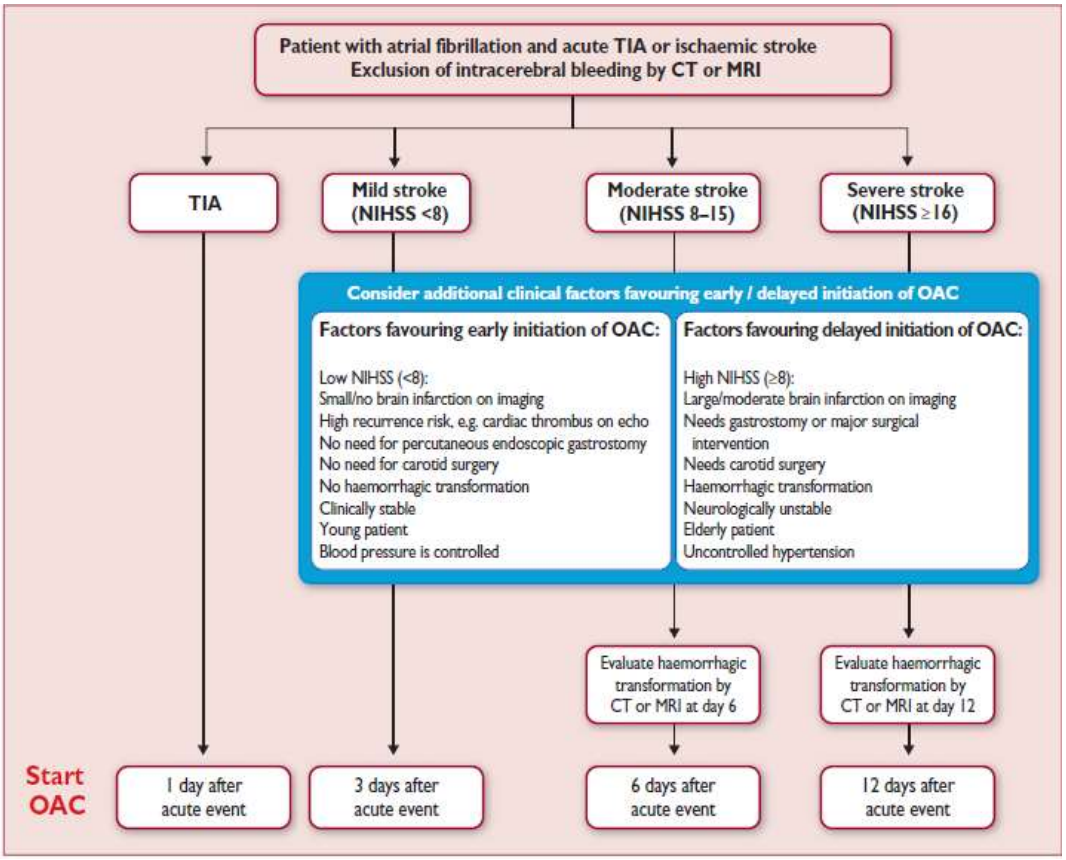


7.2 Commencing OAC in AF patients following acute TIA or ischaemic CVA.

Data on the optimal use of anticoagulants (heparin, low-molecular weight heparin, heparinoid, VKA, NOAC) in the first days after a cerebrovascular accident (CVA) are scarce. It seems likely that the bleeding risk on parenteral anticoagulation exceeds the stroke prevention benefit in the first days after a large stroke, whereas patients with a tranient ischaemic attack (TIA) or a small stroke may benefit from early (immediate)

initiation or continuation of anticoagulation. Therefore, it is proposed to initiate anticoagulation in AF patients between 1 and 12 days after an ischaemic stroke, depending on stroke severity (figure 12). Repeat brain imaging is suggested to determine the optimal initiation of anticoagulation in patients with a large stroke at risk for haemorrhagic transformation. Long-term OAC conveys benefits in AF patients who survived a stroke. NOACs seem to convey slightly better outcomes, mainly driven by fewer intracranial haemorrhages and haemorrhagic strokes. If a patient suffers a stroke or TIA whilst taking an anticoagulant, switching to another anticoagulant should be considered.

Figure 12: AF patients in the setting of CVA / TIA



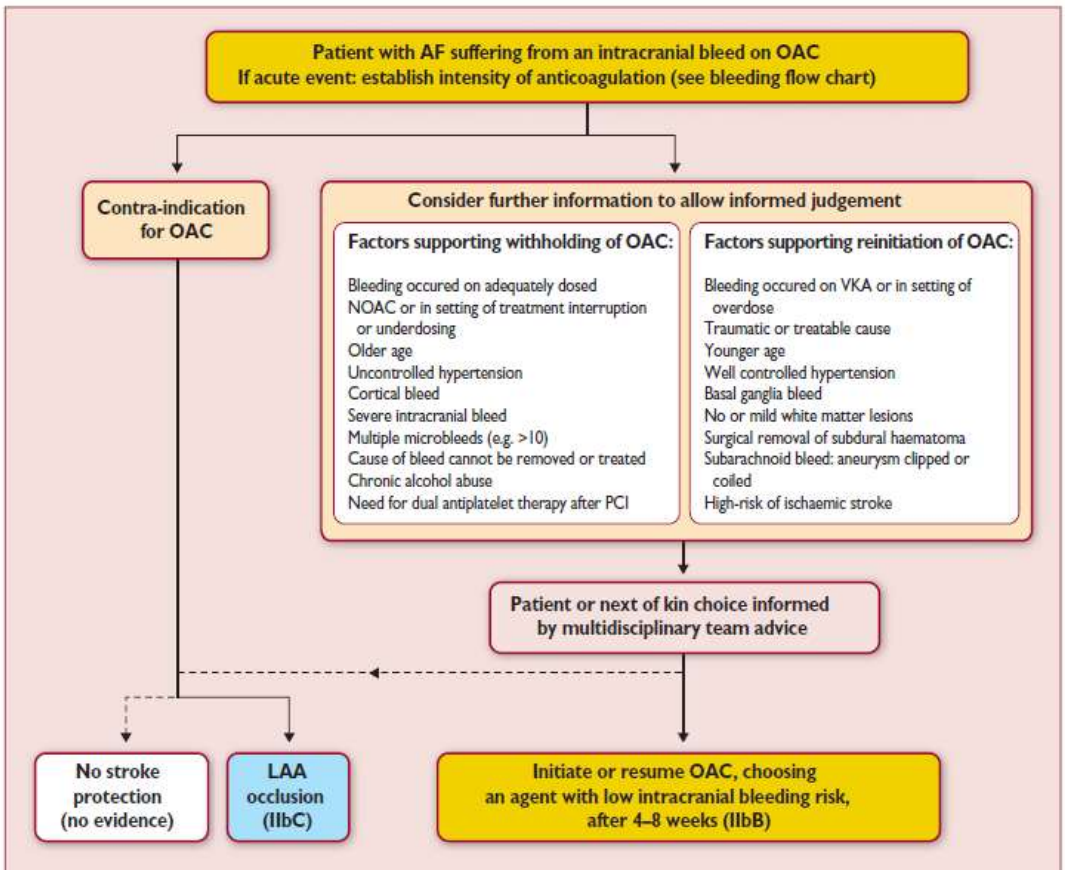
ESC Guidelines for the management of atrial fibrillation developed in collaboration with EACTS. The Task Force for the management of atrial fibrillation of the European Society of Cardiology (ESC). European Heart Journal (2016) 37, 2893–2962

7.3 Commencing OAC in AF patients following acute intracranial bleed

No prospective studies have investigated the benefit or risk of the initiation of OAC after intracranial haemorrhage and patients with a history of intracranial bleeding were excluded from the randomized trials comparing NOACs with VKAs. The available

evidence indicates that anticoagulation in patients with AF can be re-initiated after 4–8 weeks, especially when the cause of bleeding or the relevant risk factor (e.g. uncontrolled hypertension) has been treated, and that such treatment leads to fewer recurrent (ischaemic) strokes and lower mortality (figure 13). A multidisciplinary decision with input from stroke physicians/neurologists, cardiologists, neuroradiologists, and neurosurgeons is advised.

Figure 13: AF patients in the setting of intracranial bleeding



ESC Guidelines for the management of atrial fibrillation developed in collaboration with EACTS. The Task Force for the management of atrial fibrillation of the European Society of Cardiology (ESC). European Heart Journal (2016) 37, 2893–296

7.4 Previous bleeding event

History of bleeding events and the presence of anaemia are important parts of the assessment of all patients receiving OAC. The majority of bleeding events are gastrointestinal. In patients in whom the source of bleeding has been identified and corrected, OAC can be reinitiated.

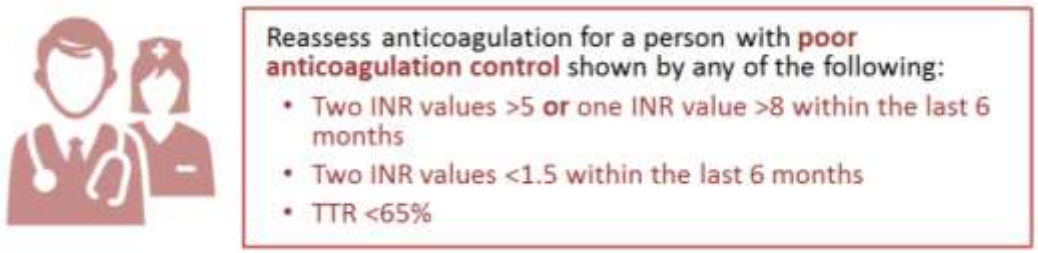
7.5 Labile INRs

TTR on VKA therapy is an important predictor of major haemorrhage. Therefore, we recommend targeting the INR between 2.0 and 3.0 in patients on VKAs, maintaining a high TTR (e.g. ≥70%), and to consider switching to a NOAC when a high TTR cannot be sustained.

Poor INR control is summarised in figure 14 and defined by NICE as:

- ✓ One INR >8 within the last 6 months
- ✓ Two INRs >5 within the last 6 months
- ✓ Two INRs <1.5 within the last 6 months
- ✓ TTR <65% over 6 months

Figure 14: Poor anticoagulation



7.6 Alcohol abuse

Alcohol excess is a risk factor for bleeding in anticoagulated patients, mediated by poor adherence, liver disease, variceal bleeding and risk of major trauma. Severe alcohol abuse and binge drinking habits should be corrected in patients eligible for OAC.

7.7 Falls and dementia

Falls and dementia are associated with increased mortality in AF patients, without evidence that these conditions markedly increase the risk of intracranial haemorrhage. Hence, anticoagulation should only be withheld from patients with severe uncontrolled falls (e.g. epilepsy or advanced multisystem atrophy with backwards falls), or in selected patients with dementia where compliance and adherence cannot be ensured by a caregiver.

7.8 Bridging anticoagulation

Some cardiovascular interventions can be performed safely on continued OAC. This should be discussed with the operator or follow local cath lab guidance. When interruption of OAC is required, bridging does not seem to be beneficial, except in patients with mechanical heart valves. Bridging should be minimized to prevent stroke.

7.9 Management of bleeding events

General assessment of an anticoagulated patient with AF experiencing a bleeding event should include the assessment of bleeding site, onset, and severity of the bleeding, the time-point of last intake of OAC and other antithrombotic drugs, and other factors influencing bleeding risk such as CKD, alcohol abuse, and concurrent medications. Laboratory tests should include haemoglobin, haematocrit, platelet count, renal function, and, for VKA patients, prothrombin time, activated partial thromboplastin time, and INR. Coagulation tests do not provide much information in patients on NOACs, except for activated partial thromboplastin time in the case of dabigatran.

ESC proposes a simple scheme to manage bleeding events in patients on OAC (figure 15). Minor bleeding events should be treated with supportive measures such as mechanical compression or minor surgery to achieve haemostasis. In patients receiving VKAs, the next dose of VKA can be postponed. NOACs have a short plasma half-life of approximately 12 h, and improved haemostasis is expected within 12– 24 hr after a delayed or omitted dose.

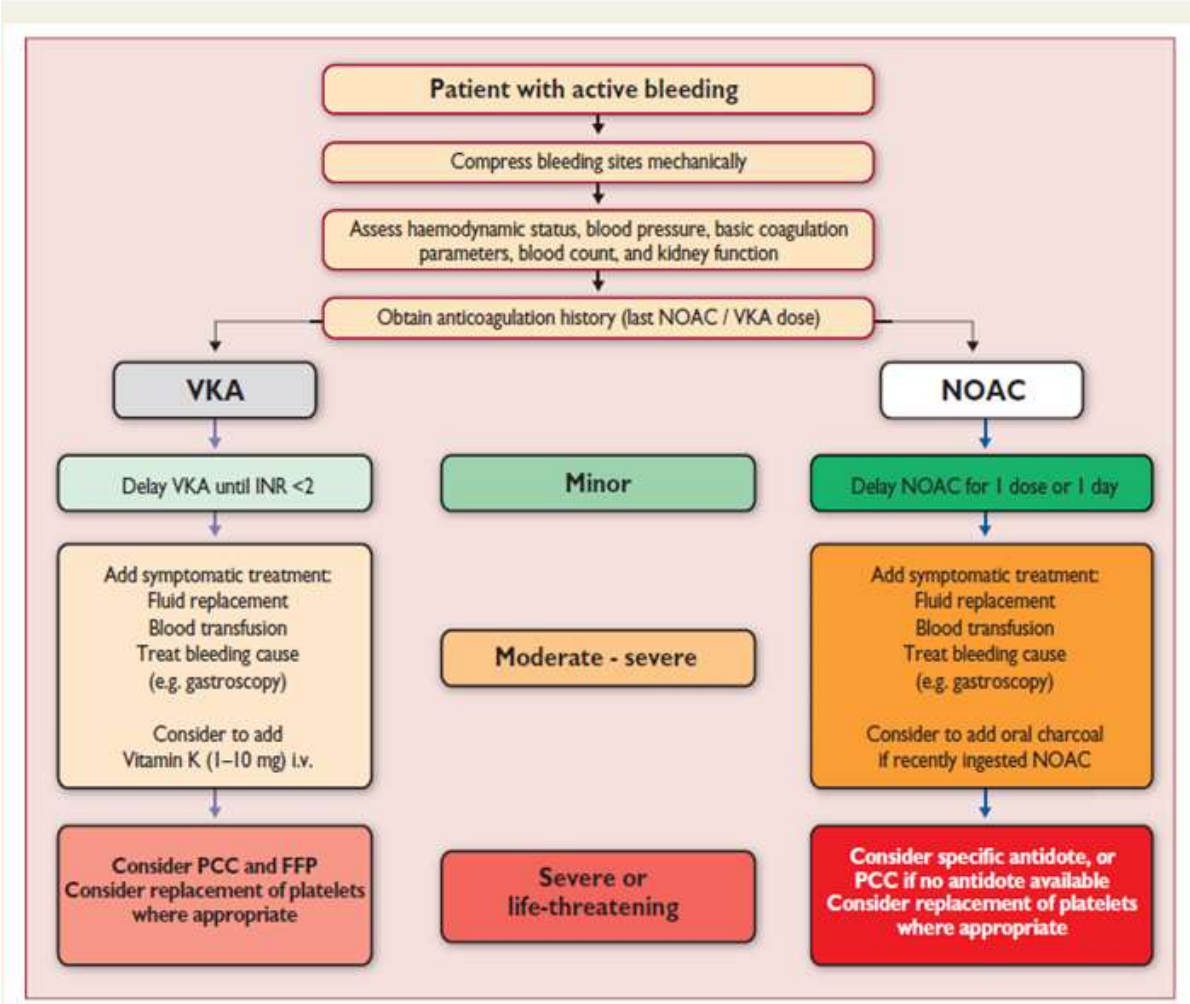
Treatment of moderate bleeding events may require blood transfusions and fluid replacement. Specific diagnostic and treatment interventions directed against the cause of the bleeding (e.g. gastroscopy) should be performed promptly. If the intake of NOAC was recent (2–4 hrs), charcoal administration and/or gastric lavage will reduce further exposure. Dialysis clears dabigatran but is less effective for the other NOACs.

Immediate reversal of the antithrombotic effect is indicated in severe or life-threatening bleeding events. For VKAs, administration of fresh frozen plasma restores coagulation more rapidly than vitamin K, and prothrombin complex concentrates achieve even faster blood coagulation.

Several antidotes to NOACs are under development. Idarucizumab (approved in 2015 by the US Food and Drug Administration and the European Medicines Agency) is a clinically available humanized antibody fragment that binds dabigatran and rapidly and dose-dependently reverses its effects without over-correction or thrombin generation.

Please refer to dedicated SHSCT guidelines / haematology guidelines for further information.

Figure 15: Patients with active bleeding



ESC Guidelines for the management of atrial fibrillation developed in collaboration with EACTS. The Task Force for the management of atrial fibrillation of the European Society of Cardiology (ESC). European Heart Journal (2016) 37, 2893–296.

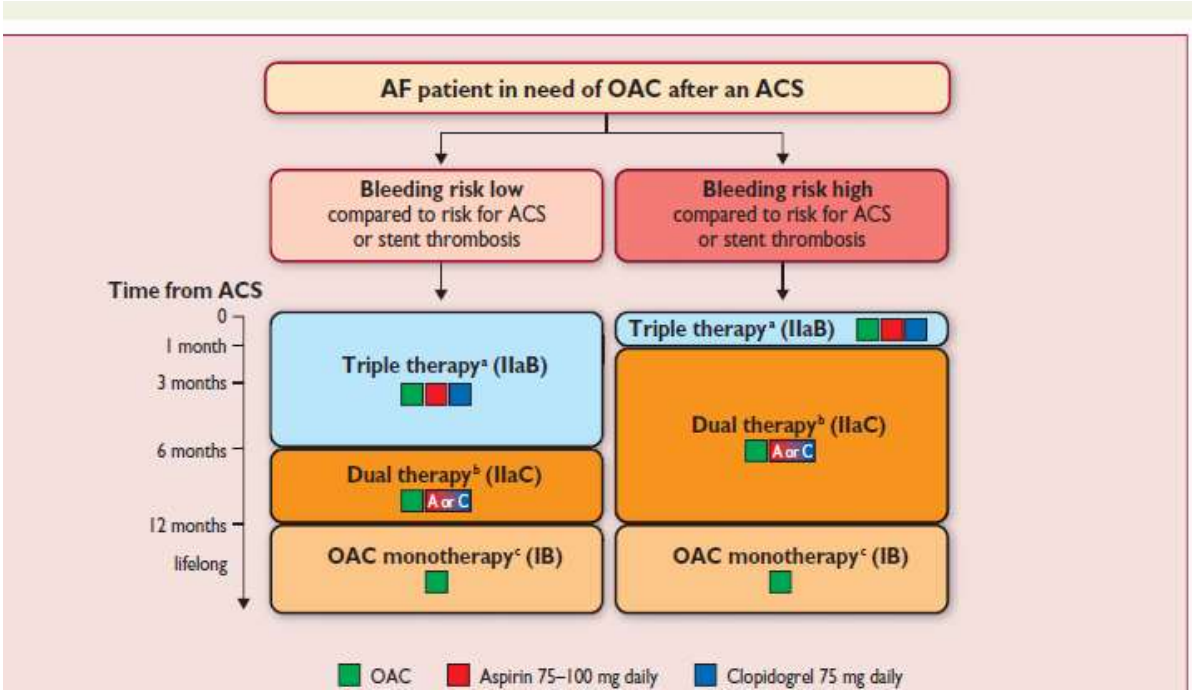
7.10 Concomitant OAC / antiplatelet therapy

Approximately 15% of AF patients in contemporary trials and registries have a history of myocardial infarction. Between 5–15% of AF patients will require stenting at some

point in their lives. This scenario requires careful consideration of antithrombotic therapy, balancing bleeding risk, stroke risk, and risk of ACS.

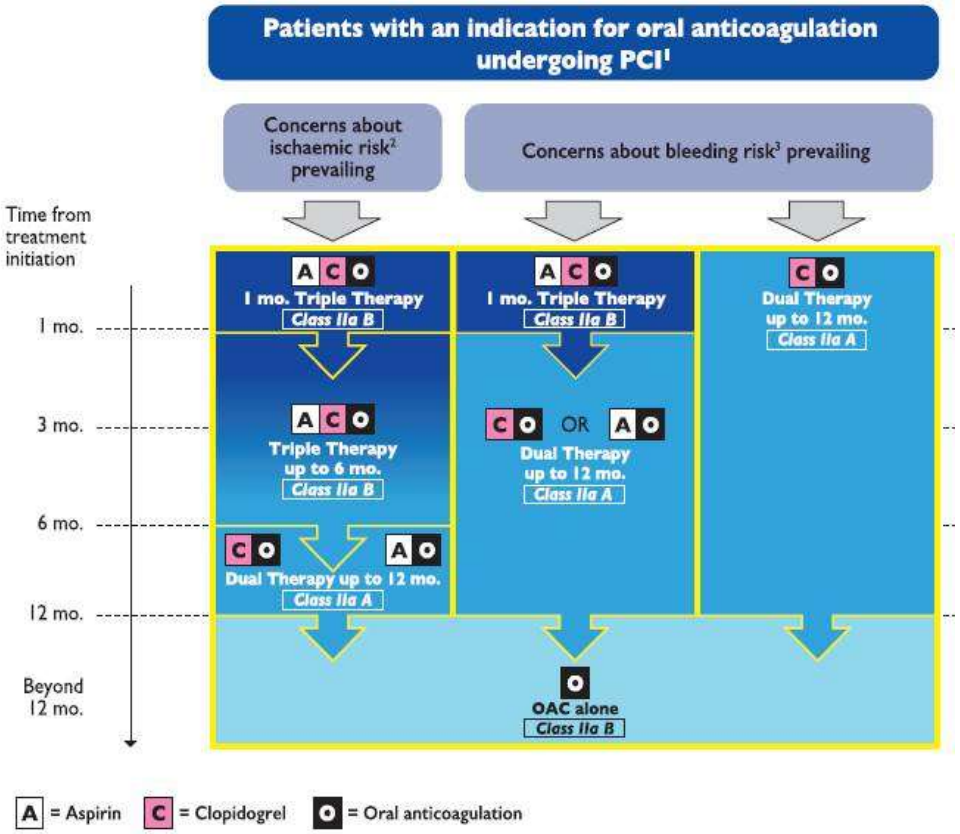
Departments and operators differ in their approach and therefore advice should be sought from the interventional cardiologist for specific cases. ESC guidelines are summarised in figure 16-17.

Figure 16: AF patient in need of OAC after ACS



ESC Guidelines for the management of atrial fibrillation developed in collaboration with EACTS. The Task Force for the management of atrial fibrillation of the European Society of Cardiology (ESC). European Heart Journal (2016) 37, 2893–2962

Figure 17: AF patient in need of OAC after elective PCI



ESC guidelines for the diagnosis and management of chronic coronary syndromes: The Task Force for the diagnosis and management of chronic coronary syndromes of the European Society of Cardiology (ESC). European Heart Journal; 41(£):407-477.

7.11 Prescriber guidance on NOAC dosing errors

Patients who miss doses, accidentally take a double dose, are poorly compliant or who overdose need specific guidance which is highlighted in table 8.

Table 8: Guidance on NOAC dosing errors

Missed dose	Twice daily: take missed dose up to 6 h after scheduled intake. If not possible skip dose and take next scheduled dose. Once daily: take missed dose up to 12 h after scheduled intake. If not possible skip dose and take next scheduled dose.
Double dose	Twice daily: skip next planned dose and restart twice daily after 24 h. Once daily: continue normal regimen.
Uncertainty about intake	Twice daily: continue normal regimen. Once daily: take another dose then continue normal regimen.
Overdose	Hospitalisation advised.

EHRA practical guide on the use of new oral anticoagulants in patient with non-valvular atrial fibrillation: executive summary. Eur Heart J (2013); 34(27):2094-2106.

7.12 Patients undergoing planned surgical intervention.

Some procedures can be done without interruption of anticoagulation. Other procedures should be performed after temporary cessation of the NOACS.

Advice should be sought from the team performing the procedure.

In general, low risk procedures should have NOAC therapy discontinued for ≥24 hours from last intake. High risk procedures should have NOAC therapy discontinued ≥48 hours from last ingestion. Table 9 shows examples of low and high risk procedures. Patients with CKD may need NOAC therapy held for 48 – 96 hours (discuss with operator).

Table 9: NOAC patient undergoing procedures

Not usually requiring cessation of NOAC	Dental extraction (of 1-3 teeth) Incision of abscess Implant positioning Endoscopy without surgery Superficial surgery Cataract or glaucoma intervention
Low Risk	Endoscopy with biopsy Prostate or bladder biopsy Electrophysiology study Right sided ablation Pacemaker or ICD implantation
High risk	Catheter ablation of left sided pathways (WPW) Spinal or epidural anaesthesia Lumbar puncture Thoracic surgery Abdominal surgery Major orthopaedic surgery Liver biopsy Transurethral resection of prostate Kidney biopsy

EHRA practical guide on the use of new oral anticoagulants in patient with non-valvular atrial fibrillation: executive summary. Eur Heart J (2013); 34(27):2094-2106.

7.13 Patients undergoing emergency surgical intervention.

If urgent surgery is required, NOAC therapy should be stopped immediately. Surgery should be deferred for 12 hours if possible, and ideally 24 hours after the last ingested dose.

For patients on Dabigatran, Idarucizumab 5mg IV reverses anticoagulation without pro-thrombotic side effects and may allow urgent intervention.

For patients on factor Xa inhibitors, there is no specific reversal agent as yet. Please follow best practice, refer to haematology team and consider guidance in figure 15 on active bleeding.

8. Obstructive sleep apnoea

AF has been associated with obstructive sleep apnoea. Multiple pathophysiological mechanisms can contribute to AF in obstructive sleep apnoea, including autonomic dysfunction, hypoxia, hypercapnia, and inflammation.

Obstructive sleep apnoea exaggerates intrathoracic pressure changes, which in itself and via vagal activation can provoke shortening of the atrial action potential and induce AF. Risk factor reduction and continuous positive airway pressure ventilation can reduce AF recurrence.

It seems reasonable to consider obstructive sleep apnoea screening in AF patients with risk factors. Obstructive sleep apnoea treatment should be optimized to improve AF treatment results in appropriate patients.

9. Lifestyle changes with AF

- **Changing diet**

- Advise people to eat a Mediterranean-style diet (more bread, fruit, vegetables and fish; less meat; and replace butter and cheese with products based on plant oils).

- **Alcohol consumption**

- Advise people who drink alcohol to keep weekly consumption within safe limits (no more than 14 units of alcohol per week for men and women and to avoid binge drinking (more than 3 alcoholic drinks in 1–2 hours).

- **Regular physical activity**

- Advise people to undertake regular physical activity sufficient to increase exercise capacity.

- Advise people to be physically active for 20–30 minutes a day to the point of slight breathlessness. Advise people who are not active to this level to increase their activity in a gradual, step-by-step way, aiming to increase their exercise capacity. They should start at a level that is comfortable, and increase the duration and intensity of activity as they gain fitness.

- **Smoking cessation**

- Advise all people who smoke to stop and offer assistance from a smoking cessation service. Advise referral to SHSCT smoking cessation nurse.

10. Follow-up

Most AF patients need regular follow-up to ensure continued optimal management. Follow-up may be undertaken in primary care, by specially trained nurses, by cardiologists, or by AF specialists. A specialist should co-ordinate care and follow-up. Follow-up should ensure implementation of the management plan, continued engagement of the patient, and therapy adaptation where needed. If AF is stabilised and optimised then it may be appropriate to discharge patients back to their GP assuming no further immediate treatment is required.

11. Update and review

- This document will be updated every 3 years.
- Revisions will be made ahead of the review date if new, relevant national guidelines are published. Where the revisions are significant and the overall policy is changed, the authors will ensure the revised document is taken through the standard consultation, approval and dissemination processes.

12. References

ESC Guidelines for the management of atrial fibrillation developed in collaboration with EACTS. The Task Force for the management of atrial fibrillation of the European Society of Cardiology (ESC). European Heart Journal (2016) 37, 2893–2962

Atrial fibrillation: the management of atrial fibrillation. National Institute for Health and Care Excellence CG180 (Published June 2014).

Ruff CT et al. Comparison of the safety and efficacy of new oral anticoagulants with warfarin in patients with atrial fibrillation: a meta-analysis of randomised trials. Lancet (2014);383(9921):955–962.

2018 European Heart Rhythm Association Practical Guide on the use of non-vitamin K antagonist oral anticoagulants in patients with atrial fibrillation. European Heart Journal 2018;39:1330–1393.

niformulary.hscni.net/formulary

Appendix 1 – Governance information

Document title	Guidelines for the management of atrial fibrillation
Date issued / approved:	**5th August 2020**
Date valid from:	**5th August 2020**
Date valid to:	**Initial date: 5th August 2023** **Extended date: 1st Jan 2027 (see below)**
Brief summary of contents	This document provides guidance for any professional involved in the clinical management of patients presenting to SHSCT with atrial fibrillation.
Policy objectives	To provide clear speciality agreed guidelines and pathways for the diagnosis and clinical management of patients with atrial fibrillation.
Keywords	Cardiology Atrial fibrillation Rate control Rhythm control Anticoagulation
Authorship	Dr David Mc Eneaney (cardiology clinical lead, consultant cardiologist) Kay Carroll (cardiology head of service) Dr Mick Connolly (consultant cardiologist) Dr Ian Menown (consultant cardiologist) Dr Neil Mc Aleavey (cardiology specialty doctor)

Addendum Jan 2024:

This policy was reviewed by the consultant cardiologists and discussed at the cardiology governance meeting in Winter 2023. It was sent out for a 6 week consultation period. No amendments were suggested by the wider cardiology team. Following universal unanimous agreement by the consultant body at this governance meeting it has been approved for 3 further years until 1st Jan 2027.

Signed: Dr Mick Connolly Consultant cardiologist

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Southern Health and Social Care Trust

Quality Care - for you, with you

Appendix 2 – DCC referral form

DCC referral form

Date: Click here to enter a date.

Name: Click here to enter text.

H&C: Click here to enter text.

Date of Birth: Click here to enter a date.

Address: Click here to enter text.

Consultant: Click here to enter text.

Has this patient had a cardioversion in the last 12 months? Choose an item.

If 'Yes' have rate/rhythm medications been adjusted? Choose an item.

Rate/ rhythm medications:

Propafenone ☐ Bisoprolol ☐

Sotalol ☐ Digoxin ☐

Flecainide☐ Verapamil ☐

Dronedarone☐ Other ☐

Amiodarone☐

Anticoagulant:_____

Does this patient have any of the following? (Please select if Yes)

- Sleep Apnoea ☐
- Severe respiratory disease ☐
- Dyspnoea lying flat ☐
- BMI >35 ☐
- Previous history of difficulty sedating or requiring more than 5mg of midazolam ☐

IF YES PATIENT WILL BE PLACED ON ANAESTHETIC LIST

Signature of requesting doctor _____

Forward requests to CathLabCare.AssessmentSisters@southerntrust.hscni.net

Appendix 3 – New AF flowchart