SUMMARY Re-thinking the management of atrial fibrillation



A common misconception is that the initial management of AF is always an emergency situation

- Many clinicians immediately focus on the need for restoring normal sinus rhythm within a 48-hour window using electrical cardioversion and do not initiate anticoagulation first
- Hesitancy regarding anticoagulation may relate to ingrained perceptions associated with warfarin, which requires intensive monitoring and can have unfavourable adverse effects



Best practice: treatment for AF should be progressive and tailored to your patient

- ✓ All patients that meet the clinical threshold should receive oral anticoagulation early to reduce stroke risk
- / DOACs, e.g. dabigatran and rivaroxaban, should be favoured over warfarin if there are no contraindications
- For many patients with AF, rate control and anticoagulation are sufficient to control symptoms and reduce the risk of stroke; there is often no need to use electrical cardioversion unless the patient is haemodynamically unstable

An individualised and progressive approach works best once AF is detected



Antiplatelet treatment such as aspirin is no longer recommended for long-term stroke prevention in patients with AF

score should be used.

- * Including accurate history, clinical examination and assessment of concomitant conditions.
- † Recommended to guide treatment decisions but should not delay intervention where a need is identified.

Why prescribe DOACs over warfarin?

Superior to treatment with warfarin for preventing stroke and major/intracranial bleeding



Rapid onset of action^{*}

Does not require routine anticoagulant monitoring, i.e. INR



Fewer medicine- and food interactions compared with warfarin

* Time to onset of action for DOACs can be within 30 mins versus 48–72 h for warfarin

✓ AF diagnosis confirmed — with ECG

Should this patient receive DOACs?

- ✓ CHA_2DS_2 -VASc score of ≥ 1 _____ for men and ≥ 2 for women
- 🗙 CrCl is < 30 mL/min 🗕
- 🗙 High HAS-BLED score ——
- Prosthetic heart valve or moderate-to-severe mitral stenosis
- Pregnancy may consider warfarin but not in 1st trimester or 2–4 weeks before delivery

If switching from warfarin to a DOAC:

** Clinical need for anticoagulation should be assessed using the

‡ Favour DOACs over warfarin if not contraindicated.

 \rightarrow DOAC

Consider

warfarin

CHA, DS, -VASc score. To calculate the risk of bleeding, the HAS-BLED

Stop warfarin and initiate

the DOAC at the normal recommended dose after waiting for the patient's INR to be \leq 2.0 for dabigatran or \leq 3.0 for rivaroxaban

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Rate control is preferred for managing AF symptoms



Get an echocardiogram early to inform treatment

Target an initial resting heart rate of < 110 bpm*



Beta-blockers are considered first-line for both acute and long-term rate control



Combination therapy may be required if heart rate target is not achieved with monotherapy



Consider the patient's LVEF and symptoms; this will inform drug choice and dosing

* Lower targets of <80 or 90 bpm may be suitable for some patients, such as those with known left ventricular dysfunction.

† Ensure beta-blocker is at the lowest possible dose for acute heart rate control.

Patient condition	Monotherapy	Combination therapy
LVEF ≥ 40%	Beta-blocker (first-line)	_
	Diltiazem	Add digoxin
	Verapamil	
	Digoxin**	Add beta-blocker, diltiazem or verapamil
Signs of congestive heart failure and LVEF < 40%	Beta blockers [†]	Add digoxin
	Digoxin**	Add beta-blocker [†]
Haemodynamic instability or severely reduced LVEF	Amiodarone	Add digoxin

** Used infrequently for monotherapy in primary care due to its potential for medicine interactions, lack of effect on heart rate during physical activity and narrow therapeutic index.



If patients have ongoing symptoms despite optimal use of rate control medicines, consider rhythm control strategies (see below); decisions here are generally guided by input from a cardiologist

What are the main treatment options?

Treatment	Medicine/optic	on	Dose range	Notes
Lifestyle modifications	Weight loss, exercise, alcohol reduction, smoking cessation		tion, smoking cessation	Reduces symptoms and AF burden
Anticoagulation	DOACs (preferred)	Dabigatran	110-150 mg BD	DOACs preferred unless contraindications
		Rivaroxaban	20 mg OD	Reversal now possible for dabigatran
	VKAs	Warfarin	1–10 mg OD (adjust dose based on INR)	
Rate control	Beta-blockers (preferred)	Bisoprolol*	1.25-20 mg OD	Rate control is preferred over rhythm control where possible; beta-blockers are the first-line rate control medicines; do not use sotalol for rate control
		Metoprolol succinate	23.75-190 mg OD	
		Carvedilol*	3.125-50 mg BD	Target HR should be < 110 bpm
	Rate limiting CCB	Diltiazem ^{*†}	60 mg TDS up to 360 mg max total daily dose (120–360 mg OD modified release)	Only use CCBs in patients with LVEF ≥ 40% Combination treatment may be needed if target HR is not met
		Verapamil**	40–120 mg TID (120–480 mg OD modified release*)	
	Cardiac glycosides	Digoxin (infrequently used for monotherapy)	0.75–1.5 mg over 24 h in divided doses (loading dose) 0.0625–0.25 mg OD (maintenance dose)	
Rhythm control	Pharmacological cardioversion e.g. flecainide, amiodarone		flecainide, amiodarone	 Indicated for symptomatic patients with AF Better for paroxysmal versus persistent Not used for permanent AF
	Electrical cardioversion			
	Advanced procedures e.g. catheter or surgical ablation			Anticoagulation is recommended before and after cardioversion

* Unapproved indication † Caution should be taken if considering use alongside beta-blockers

** Do not use alongside beta-blockers unless under specialist supervision

For further information on managing AF see: "An update on managing patients with atrial fibrillation" at https://bpac.org.nz/2017/af.aspx. AF, atrial fibrillation; BD, twice daily; bpm, beats per minute; CCB, calcium channel blocker; CrCl, creatinine clearance; DOACs, direct oral anticoagulant; ECG, electrocardiogram; INR, international normalised ratio; LVEF, left ventricular ejection fraction; OD, once daily; TDS, three times daily; VKA, vitamin K antagonist. 1. Tomlin AM, Lloyd HS, Tilyard, MW. Eur J Prev Cardiol. 2017;24:311–9. doi: 10.1177/2047487316674830; 2. Kirchhof P, Benussi S, Kotecha D, et al. Eur Heart J. 2016;37:2893–962. doi: 10.1093/eurheartj/ehw210; 3. NZ Formulary. NZF v83. 2019. Available from: www.nzf.org.nz (Accessed May, 2019).