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Guidelines for the clinical management of atrial fibrillation: a practical perspective

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Summary

Purpose: Since the management of atrial fibrillation may be difficult in the individual patient, our purpose was to develop simple clinical recommendations to help the general internist manage this common clinical problem.

Data sources: Systematic review of the literature with evaluation of data-related evidence and framing of graded recommendations.

Data synthesis: Atrial fibrillation affects some 1% of the population in Western countries and is linked to a significant increase in morbidity and mortality. The management of atrial fibrillation requires individualised evaluation of the risks and benefits of therapeutic modalities, relying whenever possible on simple and validated tools. The two main points requiring a decision in clinical management are 1) whether or not to implement thromboembolic prevention therapy, and 2) whether preference should be given to a "rate control" or "rhythm control" strategy. Thromboembolic prophylaxis should be prescribed after individualised risk assessment: for patients at risk, oral anticoagulation with warfarin decreases the rate of embolic complications by 60% and aspirin by 20%, at the expense of an increased incidence of haemorrhagic complications. "Rate control" and "rhythm control" strategies are probably equivalent, and the choice should also be made on an individualised basis. To assist the physician in making his choices for the care of an atrial fibrillation patient we propose specific tables and algorithms, with graded recommendations.

Conclusions: On the evidence of data from the literature we propose simple algorithms and tables for the clinical management of atrial fibrillation in the individual patient.

Key words: atrial fibrillation; guidelines; anticoagulation; anti-arrhythmic agents; cardioversion

Introduction

Atrial fibrillation (AF) is the most common cardiac arrhythmia in daily medical practice, and is very often evaluated and treated by non-specialists. In view of varying clinical presentations and associated problems, therapeutic options may vary widely from one patient to another. For the internist (and even for the experienced arrhythmia specialist), straightforward answers are not always found in the abundant medical literature. Systematic reviews allow methodological evaluation of the evidence and the development of guidelines designed to simplify and harmonise clinical management [1, 2]. Since 1996 we have conducted an evaluation of this kind and drawn up simple practice guidelines to be used in a general internal medicine ward. We propose algorithms and tables with graded recommendations easily usable by internists and general physicians for the individual care of atrial fibrillation patients. These guidelines are not intended to compete with the recently published and more extensive international guidelines, which represent the gold standard for AF management, but should rather be considered a simplified local approach [3].

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Methods

We selected important points requiring decision in the management of AF, such as pathogenesis, epidemiology, diagnosis, investigations, cardioversion techniques, rate and rhythm control, and thromboembolic prophylaxis. For each of these aspects we conducted a specific literature search using the Medline® database with the MeSH word "atrial fibrillation", limited to the publication types "review", "clinical study", and "meta-analysis". We restricted the search to articles issued after 1980. Articles specifically addressing the topic of interest were selected by reading the title and the abstract, with an assessment of the methodological quality of the data whenever possible. Case reports or small series were rejected. The extended versions of all selected papers were then more fully analysed. The evidence level of data was evaluated by consensus among the authors, based on the usual criteria. For each topic of interest guidelines were developed and graded on three levels: level A was based on at least 2 randomised controlled trials with a sufficient sample population, or on a meta-analysis with appropriate methodology, or lastly on basic science evidence. Level B recommendations were based on non-randomised trials or on trials covering an inadequate sample population. Experts' opinions, retrospective cohort analyses, and case-control studies resulted in level C recommendations. After internal and external review the guidelines were implemented in the Department of Internal Medicine of the Centre Hospitalier Universitaire Vaudois in Lausanne.

This paper presents the main results of this work of collaboration. In the form of answers to frequently asked questions, we propose a summary of the guidelines in conjunction with tables and algorithms. The recommendations are not intended to replace the physician's clinical judgment but rather to assist him in taking management decisions.

Results

Overview of the clinical problems of AF

What is the definition of AF?

AF is a rapid and irregular atrial arrhythmia with a frequency of over 300 beats per minute, characterised by irregular or absent auricular mechanical activity [4]. Diagnosis is based on the ECG, where normal auricular P waves are replaced by rapid and irregular oscillations corresponding to the atrial f (for atrial fibrillation) waves.

What is the mechanism of AF?

AF results from simultaneous reentrant wavelets, secondary to increased atrial automaticity and/or excitability, combined with slowing of conduction and/or shortening of the effective refractory period [4, 5]. Pulmonary veins are an important source of ectopic beats which may initiate AF, particularly when intra-atrial pressure is increased [6, 7]. The rapid onset of electrical atrial remodeling after AF initiation favours the perpetuation of the arrhythmia [4]. The autonomic nervous system plays a prominent role in the occurrence and persistence of the arrhythmia, by modulating the atrial refractory period [4, 8].

What is the epidemiology of AF?

In the Western world 5% of the population will develop AF during their lives [9]. The prevalence of AF in the general population is 0.5 to 1% but increases with age, rising to 10% in persons over 80 [10, 11]. The annual incidence varies from 0.1% under the age of 55 to more than 3% in the over-85s [10, 11]. AF is more frequent in men, but since women live longer they represent the majority of patients aged over 75 [10–12]. Ageing of the population increases the prevalence of AF and results in more frequent hospital admission [11, 13].

What are the clinical manifestations of AF?

Up to one-third of patients are asymptomatic [12, 14], but this proportion may be higher since asymptomatic patients often go undiagnosed. Most symptomatic patients report palpitations, dyspnoea, thoracic pain and asthenia, of increased intensity on physical activity. Clinical signs include an irregularly irregular pulse, often with a peripheral pulse deficit, an absent jugular venous *a* wave, and an irregular first heart sound [15].

Is there a simple clinical classification of AF?

Although somewhat arbitrary, clinical classifications of AF may simplify its clinical management [3, 16]. A collaborative working group recently proposed a consensus on nomenclature and classification of AF, in which initial episodes are distinguished from paroxysmal, persistent, or permanent ones [17] (table 1).

AF can also be classified into idiopathic or "lone" AF, which represents 40–60% of paroxystic episodes of AF, and secondary forms, most often associated with cardiac diseases [12]. Nowadays rheumatic heart disease is only rarely encountered, and hypertensive cardiopathy is the first cause of secondary AF, followed by coronary artery disease, myopericarditis, cardiomyopathies, non-rheumatic valvular disease and cardiac surgery [10, 12, 18]. Hyperthyroidism, alcohol consumption, lung disease and hypoxaemia, and electrolytic disturbances may also trigger AF [15, 19].

What are the clinical consequences of AF?

Epidemiological studies have shown that AF is associated with increased morbidity and mortality, with lowered quality of life, mainly due to stroke and heart failure [12, 18, 20–24].

AF is associated with a 5-fold increase in the risk of *stroke* (a 15-fold increase in rheumatic heart disease) and with an increase in the severity of

stroke, and is therefore the cause of the majority of cardioembolic strokes [9, 10, 25–29]. Cognitive defects may be detected in many patients with AF, together with asymptomatic embolic events on brain CT scan [30, 31]. Independent risk factors for development of stroke are age, hypertension, diabetes mellitus, previous stroke or transient ischaemic attack, heart failure and coronary artery disease, with a cumulative effect [3, 32, 33].

Decreased ventricular filling time and loss of atrial contractions may result in decreased cardiac output and overt *cardiac failure* in 15–50% of patients [15, 34]. However, AF and cardiac failure are so frequently associated that it is impossible to know which precedes the other. AF occurring in patients with heart failure and heart failure developing in AF patients significantly worsen the prognosis [35]. After several weeks of AF, mechanical remodeling or tachycardia-induced cardiomyopathy may affect the atrial myocardium, but its role in the development of heart failure may be of importance only in long-lasting episodes of AF [36, 37].

Management of AF: practical guidelines

What investigations should be performed at the time of diagnosis?

ECG must be performed to confirm a clinical diagnosis and to detect an underlying cardiac disease (Level A). History and clinical examination are important for the classification of AF (table 1), the evaluation of AF tolerance, to detect associated diseases and to guide investigations (Level C) [37, 38]. Hyperthyroidism screening is necessary only in the presence of suggestive clinical signs and for recurrent AF episodes (Level C) [39, 40]. For patients who are or will be treated with amiodarone, thyroid function test can be prescribed as part of the therapy follow-up. Long-term ECG monitoring (Holter or loop-recording) may be useful for detection of asymptomatic episodes or to confirm clinical suspicion of intermittent AF (Level C). Although not mandatory, transthoracic echocardiography (TTE) may confirm clinical suspicion of heart failure and is a sensitive test for detection of systolic or diastolic dysfunction or valve disease (Level C) [38, 41].

Should newly discovered AF always be cardioverted?

In the presence of significant haemodynamic instability or severe hypoperfusion, urgent electrical cardioversion should be performed (Level C). In all other situations the timing and mode of cardioversion should be evaluated on an individual basis (table 2 and figure 1).

When the onset of AF is known and the duration is less than 48 hours, spontaneous cardioversion is very frequent [42] and cardioversion is usually recommended only if spontaneous cardioversion does not occur within the first 48 hours after onset of the arrhythmia (Level C) [43]. Although embolic complications are extremely rare in this

Table 1

Clinical classification of atrial fibrillation (adapted from Levy et al. [16, 17]).

		episodes		cardioversion	
		number	duration	spontaneous	after treatment
Initial event (acute atrial fibrillation)	paroxysmal	unique	<48 h	always	always
	recent onset	unique	48 h to 7 d	possible	possible
	first detected	unique	unknown	rare	possible
Chronic atrial fibrillation	intermittent (recurrent)	relapses	<7 d	frequent	possible
	persistent	relapses	>7 d	impossible	possible
	permanent (accepted)	relapses	>7 d	impossible	impossible

Table 2

Guidelines for cardioversion of atrial fibrillation.

	treatment	Level
Haemodynamic compromise	immediate cardioversion	C
AF of less than 48 hours' duration with stable haemodynamic conditions	therapeutic anticoagulation (INR 2.0–3.0)	C
	treat any precipitating cause	C
	ventricular rate control	C
	cardioversion before 48 hours' duration (immediate or delayed)	A
	therapeutic anticoagulation for 4 weeks after cardioversion	B
AF of more than 48 hours or of unknown duration with stable haemodynamic conditions	immediate therapeutic anticoagulation (INR 2.0–3.0)	C
	evaluate risks and benefit of cardioversion	C
	discuss TEE: if possible, check for contra-indication to cardioversion, and if no, immediate cardioversion	B
	anticoagulation for 3 weeks before cardioversion (INR 2.0–3.0) with ventricular rate control	A
	anticoagulation for 4 weeks after cardioversion (INR 2.0–3.0)	B

INR: international normalized ratio
TEE: transoesophageal echocardiography

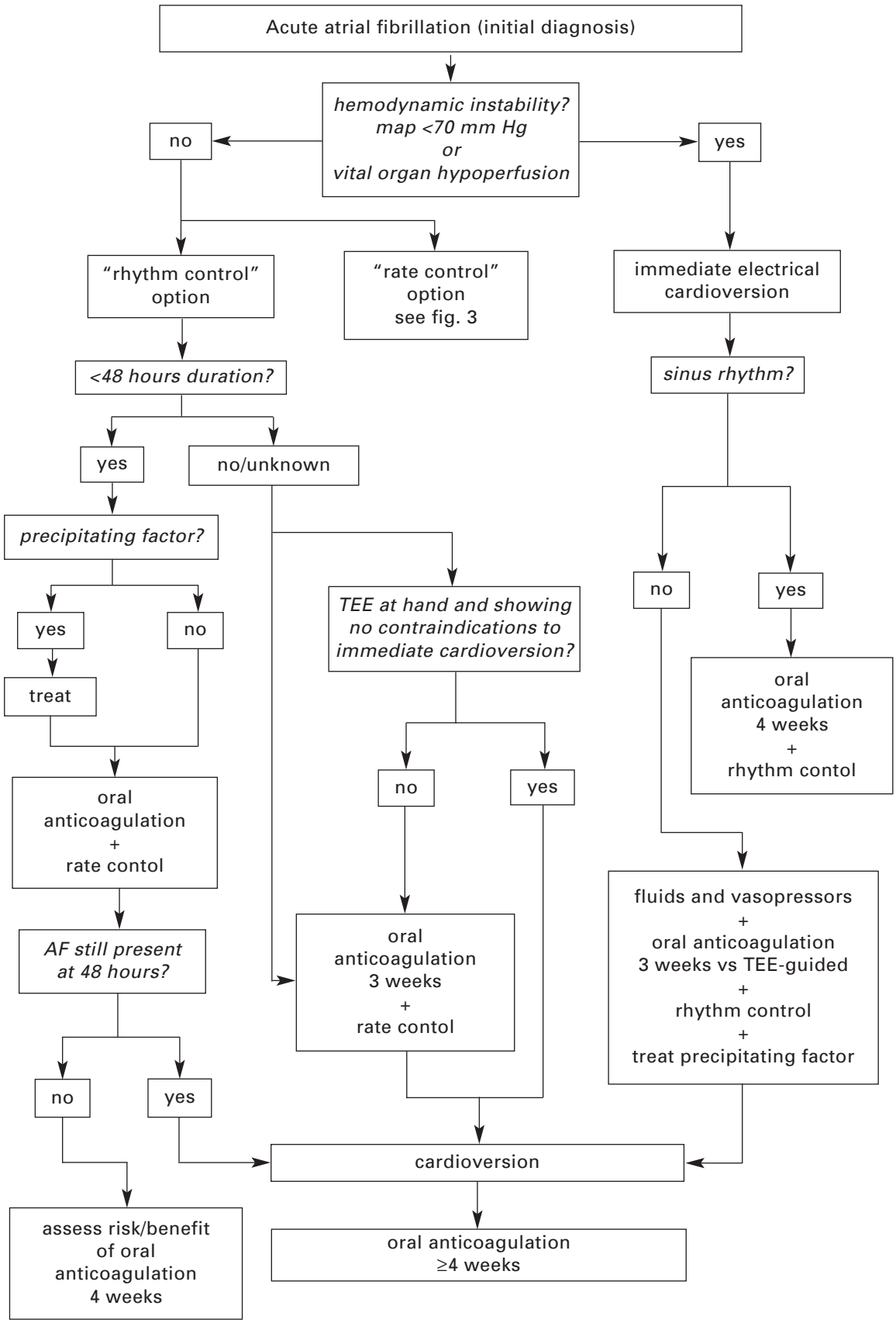


Figure 1

Management of an initial acute episode of AF. MAP: mean arterial pressure; TEE: transoesophageal echocardiography.

situation [44], most experts recommend immediate therapeutic anticoagulation for 3 weeks after cardioversion (Level C) [45, 46].

In all other situations (unknown onset, persistent and intermittent AF), or unknown onset (recent onset or recent diagnosis), cardioversion should be built into a global strategy: the "rhythm control" option, which includes cardioversion and maintenance of sinus rhythm, and the "rate control" option, without cardioversion but with control of ventricular rate (figure 3). Randomised controlled trials have compared these two strategies (PIAF, AFFIRM, RACE and STAF) and shown that they do not influence quality of life and mortality and can be considered equivalent for most patients (Level A) [47–50]. The choice be-

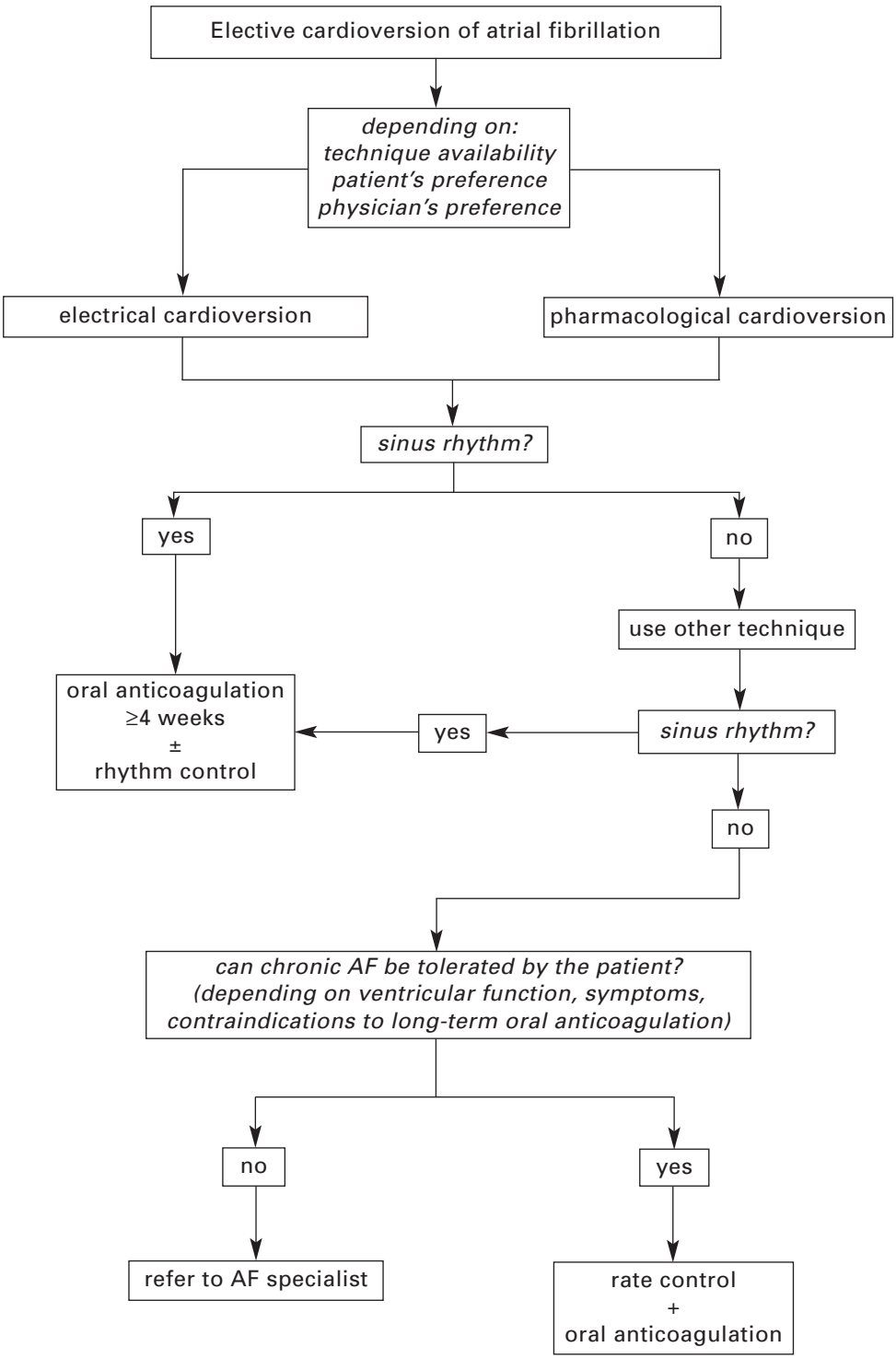


Figure 2
Elective cardioversion of AF.

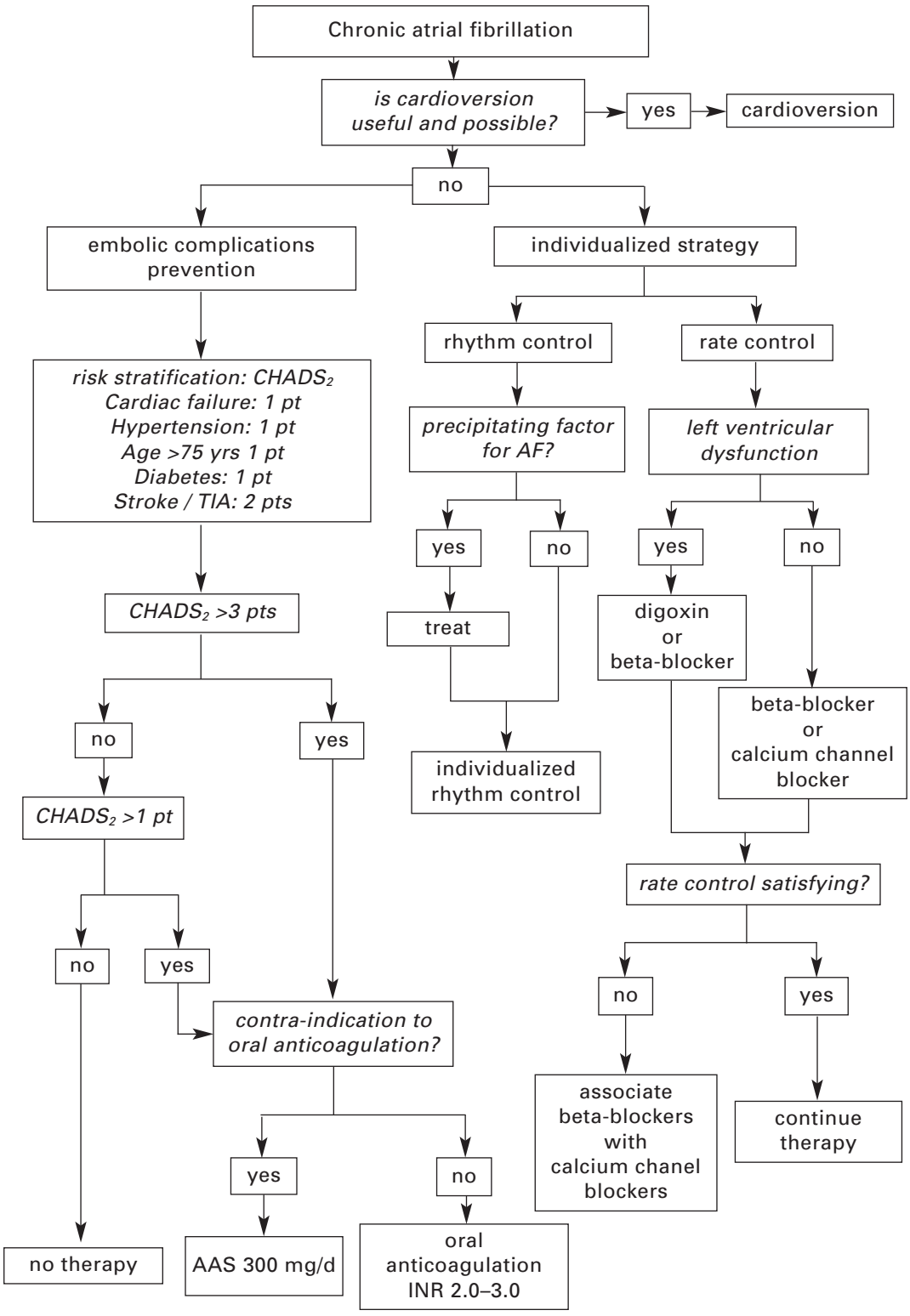
tween "rate control" and "rhythm control" should therefore be made after thorough individual evaluation of the risk and benefit of both strategies. The physician should therefore assess subjective tolerance and symptoms associated with the arrhythmia, the presence of heart failure or other cardiovascular diseases, the patient's preference and compliance with therapy, and the risk/benefit ratio of drug therapies before any choice is made. The relatively low success rate of "rhythm control" should also be considered.

What is the preferred cardioversion technique?

The choice between electrical and pharmacological cardioversion is mainly influenced by their availability and the physician's and patient's preference (figure 2). Both techniques require a short hospital stay for temporary rhythm monitoring [51], and are burdened with potential complications, associated with sedation and analgesia for electrical cardioversion, and with rhythmic and hypotensive complications for pharmacological cardioversion [52]. The combination of both techniques, starting with drug administration, followed in the event of failure by electrical cardioversion, could be an interesting and cost-effective way of increasing the success rate [53–56]. The success rate of cardioversion essentially depends on the duration of the arrhythmia and the patient's age; the longer the duration and the older

Figure 3

Management of chronic AF
AAS: aspirin



the patient, the lower the rate of cardioversion [57, 58].

Electrical cardioversion is the most widespread and probably the most effective mode of restoring sinus rhythm [37, 43, 51, 59]. Biphasic mode is about to replace the standard monophasic mode since it is equally effective but requires lower energy levels [60–63]. Anterior-posterior positioning of the electrode for DC cardioversion appears to be more effective than anterior-lateral positioning [64].

Many factors influence the effectiveness of pharmacological cardioversion, such as the duration of AF or the presence of cardiac or valvular diseases: the highest conversion rates are reported for AF of short duration in otherwise 'healthy' patients. Drug comparisons are flawed by the inadequate methodology of trials [65]. Nonetheless, Vaughan-Williams class Ic and III drugs, such as flecainide, propafenone, ibutilide, dofetilide and amiodarone, are usually considered effective drugs [37, 65–68] (table 3). A recent meta-analysis showed that amiodarone was as effective as IC drugs for cardioversion in acute AF at 24 hours, although cardioversion was slower (Level A) [68]. All

Table 3
Drugs for AF cardioversion.

Drug	class	dose range	contraindications	conversion rate	time to conversion
Propafenone	Ic	oral: 150–600 mg one dose	ventricular dysfunction severe asthma	40–75%	3–8 hours
Flecainide	Ic	oral: 100–400 mg one dose	ventricular dysfunction active ischaemia diuretic therapy	70–90%	1–8 hours
Amiodarone	III	intravenous: 150–300 mg in 20 min oral: 30 mg/kg or 0.8–1.6 g/d (total 10 g)	bradycardia	40–90% less effective in acute AF	1–24 hours
Ibutilide	III	intravenous: 0.1 mg/kg in 10 min repeat after 10 minutes if no response	left ventricular dysfunction prolonged Q-T interval	30–70%	1 hour

Class: Vaughan-Williams classification

other drugs are either ineffective or of unproven efficacy, and should therefore not be used as first-line treatment.

How should anticoagulation be prescribed before and after cardioversion?

Therapeutic anticoagulation with intravenous unfractioned heparin (UFH) should always be prescribed at the time of AF diagnosis and its effectiveness assessed, for example with the aPTT or an ACT. Low molecular weight heparins are as effective, easier to use, and could shorten the hospital stay (Level B) [69, 70]. Heparin anticoagulation must be rapidly replaced by oral anticoagulation with warfarin or derivatives, within 3–5 days. The duration of treatment thereafter depends on the therapeutic strategy (table 2).

If cardioversion is the preferred option, it should be performed only after a three-week period of controlled anticoagulation (INR 2.0–3.0, with twice weekly monitoring), the only exception being AF of less than 48 hours' duration. During this period the INR should not fall below 2.0, otherwise a new 3-week period of oral anticoagulation should be started. This could explain a longer than recommended duration of anticoagulation in real-life medical practice [71]. The ACUTE study has shown that immediate cardioversion can be safely performed in the absence of auricular thrombi or spontaneous auricular contrast in transoesophageal echocardiography (TEE) (Level B), thus reducing the rate of haemorrhagic complications [72, 73]. However, post-cardioversion anticoagulation is still mandatory, though the absence of a cost-effectiveness analysis and the restricted availability of TEE in clinical practice limit its widespread use.

Transient atrial mechanical dysfunction is frequent after cardioversion and usually lasts only a few hours, in rare cases over a week [74–76]. After 4 weeks of sinus rhythm, atrial mechanical function should be normalised in most patients [77]. Effective anticoagulation must therefore be maintained for at least 4 weeks after cardioversion (Level B). The intensity of anticoagulation should be regularly monitored (INR 2.0–3.0).

How should the risk of embolic complications of AF be evaluated?

Risk stratification for the development of stroke can be based on clinical factors [33, 78]. Many stratification systems have been validated which combine similar items such as age, hypertension, cardiac failure, history of stroke or diabetes [3, 33, 79–84]. The $CHADS_2$ (Cardiac failure, Hypertension, Age >70, Diabetes, history of Stroke or transient ischemic attack) score (table 4) is easy to use [33]. The recently published Framingham score is more complicated but may be useful in case of doubt [84]. A minority of patients are at increased risk of thromboembolic complications in the absence of clinical risk factors, but systematic use of TEE for risk assessment is not recommended (Level B) [83].

How should embolic complications of AF be prevented?

Oral anticoagulation (INR of 2.0–3.0) is recommended for all chronic forms of AF (persistent, permanent, and recurrent) in the presence of a significant risk of stroke and in AF associated with rheumatic valvular diseases (INR 3.0–4.0) [25, 26, 46]. It can be very difficult to prove the maintenance of sinus rhythm even with continuous ambulatory ECG monitoring, and anticoagulation should therefore be prescribed indefinitely for the majority of patients (Level C). This is particularly true of recurrent asymptomatic AF episodes, which are very difficult to detect but are associated with a similar risk of stroke to permanent or persistent AF [14, 27, 49]. Moreover, AF relapses are very frequent and often asymptomatic in patients treated with anti-arrhythmic drugs [49].

Eleven randomised trials of primary (I) or secondary (II) prevention [78] (SPAF 1, 2, and 3 (I) [81, 85–87], CAFA (I) [88], SPINAF (I/II) [89], AFASAK 1 and 2 (I) [90, 91], BAATAF (I) [92], EAFT (II) [93], and PATAF (I) [94]) have shown, despite methodological drawbacks, the benefit of antithrombotic prevention [78, 95–98], with a 60% reduction (47 to 71%, 95% confidence interval) in the relative risk of stroke associated with anticoagulation compared to placebo. This compares favourably with the 20% (4–36%) reduction ob-

Table 4

Stratification and prevention of thromboembolic risk with the $CHADS_2$ score.

Cardiac failure		1 point	
Hypertension		1 point	
Age over 75 years		1 point	
Diabetes mellitus		1 point	
History of stroke or transient ischaemic attack		2 points	
	Risk of stroke (per 100/yr)	**Prevention of thromboembolic complications**	**Level**
$CHADS_2$ >3	8.5 to 18.2%	Oral anticoagulation (INR 2.0–3.0)	A
$CHADS_2$ 2–3	4.0 to 5.9%	Oral anticoagulation (INR 2.0–3.0) or aspirin (300 mg/d)	A
$CHADS_2$ 0–1	1.9 to 2.8 %	<55 years old: *nil* >55 years old: aspirin (300 mg/d)	A

tained with aspirin, and anticoagulation reduces the risk of stroke by 33% (16–50%) compared with aspirin, with an increase in the risk of hemorrhage [97]. Several recent "outcome studies" have confirmed these results over longer periods of time in unselected outpatients [99–102].

What risk is involved in anticoagulation for AF?

Even though the proportion of treated patients has quadrupled since 1990, oral anticoagulation is still underprescribed in AF, mainly because of fear of haemorrhagic complications [103–108]. To limit this risk an INR between 2.0 and 3.0 is recommended, with close monitoring [109, 110]. Recently published data suggest that the rate of complications is not increased in cases with previous episodes of upper gastrointestinal tract bleeding, predisposition to falling and old age, and there is only conflicting evidence that alcoholism, the presence of a bleeding diathesis or non-compliance with monitoring increase this risk [108]. Fear of haemorrhagic complications is therefore often greater than the real risk. The haemorrhagic risk should nevertheless be assessed, but there are no validated tools for such evaluation in patients with AF. For the vast majority of patients with AF the benefit of thromboembolic prophylaxis largely outweighs the risk of hemorrhagic complications [108, 111]. The Landefeld-Beyth score is based on 4 independent factors (age >65 years, history of stroke, of gastro-intestinal bleeding and of serious co-morbidity such as myocardial infarction or renal failure), but has only been validated for patients with venous thromboembolic diseases [112–114]. Its usefulness for patients with AF is less evident, since most AF patients with a clear indication for anticoagulant prophylaxis are also at high risk of bleeding complications, as predicted by this score. Close monitoring of the level of anticoagulation is therefore the key to safe prescription in most patients, e.g. the very elderly (Level C) [115]. The patient can participate in the decision to introduce thromboembolic prevention therapy, thus improving his quality of life and reducing costs compared with systematic prescription (Level B) [116, 117].

How can "rhythm control" be obtained?

One year after cardioversion more than two thirds of patients present a recurrence of AF [37, 49, 50, 118]. The main risk factors for recurrence are functional NYHA class before cardioversion and non-rheumatic origin of AF [119]. Clinical trials have shown that most drugs are more efficient than placebo in maintaining sinus rhythm, but with such poor methodology that recommendations are difficult to formulate [37, 65]. The individual risks and benefits of each therapeutic agent must be evaluated, and co-morbidities should guide the choice of drug.

With amiodarone (100 to 200 mg/d) sinus rhythm is maintained after one year in more than 50% of patients [37, 49, 120, 121]. Its side effects are generally tolerable, except for the rare hyperthyroidism and lung toxicity. Its proarrhythmogenic effect is low, but high-degree heart block is frequent in older patients, particularly women [122]. Dofetilide is a promising drug, particularly for heart failure patients [123]. Sotalol is less effective than amiodarone in this situation [120, 121]. Class I drugs (flecainide, quinidine, disopyramide and propafenone) are effective but their side effects may outweigh their antiarrhythmic properties, particularly in patients with structural heart disease [121, 124, 125].

How can "rate control" be obtained?

In acute episodes of AF, rate control (90–100 per minute) should be rapidly obtained, intravenous administration being the route of choice (table 5). Calcium channel blockers and beta-blockers are more rapidly effective than digoxin, and combination is sometimes necessary [126–129]. Intravenous administration of calcium channel blockers and beta-blockers may be associated with significant hypotension, and patients should therefore be closely monitored during the procedure.

In chronic forms of AF pharmacological control of ventricular rate is the first choice (table 5), and interventional therapies should be considered only after treatment failure [130]. A ventricular rate of 90–100 per minute is generally recommended, but must be individualised on the basis of

Table 5
Drugs for ventricular rate control.

Drug	class	acute AF dosing	chronic AF dosing
Diltiazem	IV	0.25 mg/kg intravenous bolus in 2 min followed by 5–15 mg/h under blood pressure monitoring	180–270 mg/d orally
Verapamil	IV	5–10 mg intravenous dose in 3 min followed by 0.1–0.5 mg/kg/min under blood pressure monitoring	120–480 mg/d orally
Esmolol	II	0.5 mg/kg intravenous bolus in 1 min followed by 50–100 mg/kg/min	–
Metoprolol	II	5 mg intravenous bolus in 1 min repeat up to 3 times if necessary	25–200 mg/d orally
Propranolol	II	0.15 mg/kg intravenous bolus in 20 min followed by 3 mg/h	10–100 mg/d orally
Digoxin	-	0.5 mg intravenous bolus followed by 0.25 mg after 6 and 12 h	0.125–0.250 mg/d orally

AF: atrial fibrillation
min: minutes
h: hours

symptoms and signs, particularly during exercise [131, 132]. Digoxin is more effective than placebo, but rate control is rarely satisfactory during exercise and should therefore be prescribed only to patients with concomitant systolic dysfunction [130, 131, 133, 134]. Beta-blockers are very effective alone or in combination [134, 135]; their side effects, such as symptomatic bradycardia and heart blocks, are rare, though more frequent in elderly patients [131]. The beneficial effect of beta-blockers in patients with chronic heart failure makes them an alternative to digoxin [136–138]. Non-dihydropyridine calcium channel blockers, such as diltiazem and verapamil, are more effective than digoxin [130, 134, 135]. A significant fall in blood pressure is frequent but well tolerated in most patients [127], and heart blocks only rarely occur in older patients or in association with beta-blockers or digoxin [134]. Amiodarone for rate control should be restricted to first-line drug failure or contraindications [134].

What is the place of interventional therapies?

Such therapies were initially proposed years ago, but never imposed themselves as relevant therapeutic options. Many new procedures have recently been further developed, and a large body of data now shows that in the near future interventional therapies may become a satisfactory option for the management of many patients with AF. However, due to their recent development and their relatively limited availability they nowadays only apply to complex situations in which "standard" treatments have failed. These therapies can be divided into palliative, preventive and curative strategies. Palliative approaches, such as complete or selective ablation of the atrioventricular conduction pathways, associated with definitive ventricular pacing, are indicated when the ventricular rate is not under control despite optimal medical therapy [139]. Preventive techniques, centred on surgical or catheter-based modifications of intra-atrial conduction, have the potential to prevent recurrence of AF and maintain sinus rhythm in more than 80% of patients at 6 months [140]. Curative techniques, such as selective catheter ablation of auricular or para-auricular ectopic foci [141], or pulmonary vein isolation [142], may cure the arrhythmia in more than 70% of paroxystic AF. In a recent controlled non-randomised trial, pulmonary vein isolation was associated with an decrease in mortality and morbidity compared with anti-arrhythmic therapy [143]. However, the data are preliminary and this technology is still limited to a few centres whose clinical research will assess its indications and long term complications [144]. An implantable atrial cardioverter is a potentially interesting device, but its development is hampered by most patients' poor tolerance of painful shock deliveries [145]. Finally, the role of various pacing techniques in the management of AF is currently under evaluation in several trials [146].

Conclusions

AF is an increasingly frequent cardiac arrhythmia. Thanks to progress in therapies and extensive clinical research, its management can nowadays be tailored to the patient's individual characteristics and the patient's and physician's preferences. Decision algorithms and recommendation tables can assist the physician in his decision-making, particularly in diagnostic and therapeutic areas such as global strategy, choice of cardioversion mode or thromboembolism prevention treatment. In all complex cases, such as treatment failure or unusual presentations, the patient should be referred to an AF specialist.

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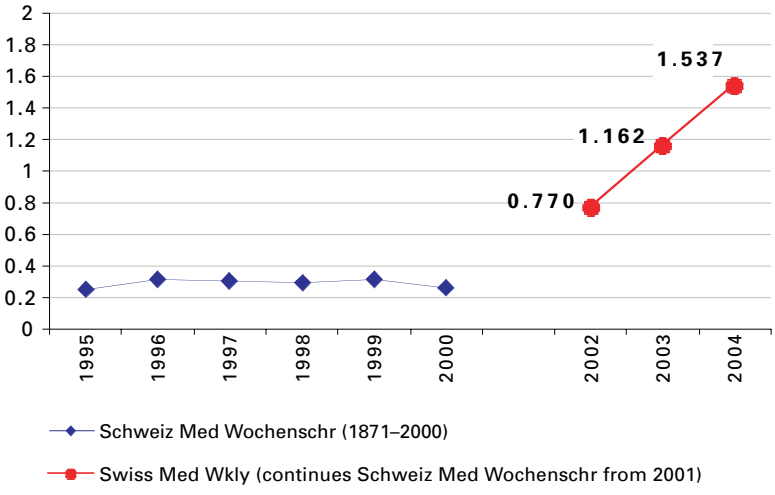

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