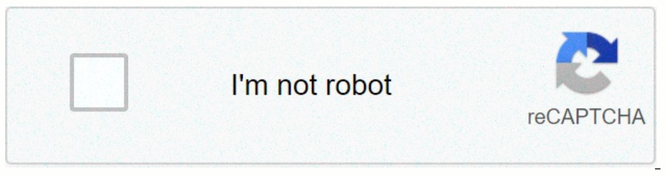

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Atrial flutter guidelines esc

Atrial fibrillation (AF), the most common persistent heart rhythm disorder, increases in prevalence as the population ages. Although it is often associated with heart disease, AF occurs in many patients without detectable disease. Haemodynamic impairment and thromboembolic events result in significant morbidity, mortality and cost. Accordingly, the American College of Cardiology (ACC), the American Heart Association (AHA), and the European Society of Cardiology (ESC) created a committee of experts to establish guidelines for the management of this arrhythmia. The committee consisted of 8 members representing the ACC and AHA, 4 representing the ESC, 1 from the North American Society of Pacing and Electrophysiology (NASPE), and a representative of the Johns Hopkins University Evidence-Based Practice Center representing agencies for health research and quality reports on atrial fibrillation in the elderly. This document was reviewed by 3 official reviewers nominated by the ACC, 3 nominated by the AHA, and 3 nominated by the ESC, as well as the ACC Clinical Electrophysiology Committee, the AHA ECG and Arrhythmia Committee, NASPE, and 25 reviewers nominated by the Writing Committee. The document has been approved for publication by acc, aha and esc managing authorities and officially approved by NASPE. These guidelines will be reviewed annually by the Working Party and will be considered up-to-date if the Working Party does not revise or withdraw them from distribution. The Committee carried out a comprehensive review of the literature from 1980 to June 2000 on AF using the following databases: PubMed/Medline, EMBASE, Cochrane Library (including Cochrane Database of Systematic Reviews and Cochrane Controlled Trials Registry) and Best Evidence. The search was limited to English resources and human subjects.II. DefinitionA. Atrial fibrillationAF is a supraventricular tachyarrhythmia characterized by uncoordinated atrial activation with subsequent deterioration of the mechanical functioning of the atrials. On an electrocardiogram (ECG), AF is described by replacing consistent P waves with rapid oscillations or fibrillation waves, which differ in size, shape and timing, associated with irregular, often rapid ventricular response, when the atrioventricular (AV) conduction is intact (1). The ventricular response to AF depends on the electrophysiological properties of the AV node, the level of the vagal and the sympathetic tone and action of drugs (2). Regular RR intervals are possible in the presence of an AV block or interference by ventricular or connecting tachycardia. Fast, irregular, persistent, wide-QRS-complex tachycardia strongly indicates AF with guidance through the accessory path or AF with the substrate of the bundle-branch block. Extremely fast rates (more than 200 bpm) indicate the presence of runway accessories.B. Related ArrhythmiasAF can be isolated or associated with other arrhythmias, often atrial flutter or atrial tachycardia. flutter may occur during treatment with antiarrhythmic agents prescribed to prevent repeated AF. Flutter attea is more organized than AF, with a saw-tooth pattern of regular attea activation called flutter (f) waves on the ECG, especially visible in leads II, III, and aVF. Untreated, atteam speed usually ranges from 240 to 320 beats per minute (bpm), with f waves reversed in ECG leads II, III, and aVF and upright in lead V1. The right atrium activation wave (RA) can be reversed, resulting in f waves that are upright in leads II, III and aVF and inverted in lead V1. The two-to-one AV block is common, producing a ventricular frequency of 120 to 160 bpm. Flutter atms can degenerate into AF, AF can initiate attea flutter, or ECG pattern can alternate between attea flutter and AF, reflecting changing attea activation. Other atrial tachycardia, as well as AV reentrant tachycardia and AV nodule reentrant tachycardia, can also trigger AF. In other tachycardia ates, P waves are easily identifiable and are separated by an isoelectric baseline for 1 or more ECG conductors. Morphology of P waves can help locate the origin of the atine tachycardia. The unique type of atrial tachycardia comes from the pulmonary veins (3), is usually faster than 250 bpm, and often degenerates into AF. Intracardiac mapping can help distinguish different atrial arrhythmias. III. The AF classification has a heterogeneous clinical presentation that occurs in the presence or absence of detectable heart disease or associated symptoms. For example, the term solitary AF has been variously defined. The prognosis in terms of thromboembolism and mortality is most benign when applied to young individuals (under 60 years of age) without clinical or echocardiographic evidence of cardiopulmonary disease (4). These patients have a favorable prognosis with regard to thromboembolism and mortality. However, due to ageing or the development of cardiac abnormalities, patients move over time from the solitary category of AF, and the risk of thromboembolism and mortality increases. Lone AF differs from idiopathic AF, which means uncertainty about its origin regardless of the patient's age or associated cardiovascular pathology. By convention, the term nonvalvular AF is limited to cases when rhythm disorders occur in the absence of rheumatic mitral stenosis or prosthetic heart valve. The classification scheme recommended in this document represents a consensus based on a desire for simplicity and clinical relevance. The clinician should distinguish the first detected episode of AF, whether symptomatic or limited, in the knowledge that there may be uncertainty as to the duration of the episode and previous undetected episodes (Fig. 1). If the patient has had 2 or more episodes, AF is considered recurrent. After completion, the recurrent AF is marked as paroxysmal and, at permanent, permanent. In the latter case, pharmacological therapy or electrical cardioversion does not change the labelling. Persistent AF can be either the first presentation or the culmination of recurring episodes of paroxysmal AF. Persistent AF includes cases of long-term AF (eg, greater than 1 year) in which cardioversion has not been indicated or attempted, usually leading to permanent AF (Fig. 1). The terminology defined in the preceding paragraph applies to AF episodes that last more than 30 seconds and that are not related to a reversible cause. AF secondary to an accelerating condition, such as acute myocardial infarction, cardiac surgery, myocarditis, hyperthyroidism or acute lung disease, is considered separately. In these settings, treatment of the underlying disorder in parallel with the treatment of an episode of AF usually eliminates arrhythmia. Figure 1. Patterns of atrial fibrillation. 1, episodes that usually last less than or equal to 7 days (most less than 24 h); 2, usually more than 7 days; 3, cardioversion has failed or not tried; and 4, either paroxysmal or persistent AF may be recurrent.IV. Epidemiology and PrognosisAF is the most common clinically significant cardiac arrhythmia. In one series, AF accounted for 34.5% of patients hospitalized with heart rhythm disorders (5). It is estimated that 2.2 million Americans have paroxysmal or persistent AF (6).A. The prevalence of AF is estimated at 0.4% of the total population, increasing with age (7). AF is less common in childhood except after heart surgery. It occurs in less than 1% of those under 60 years of age, but in more than 6% of over-80s (8-10 years of age) (Figure 2). Age-adjusted prevalence is higher in men (10.11). Blacks have less than half the age-adjusted risk of developing AF, which is seen in white (12). The rate of solitary AF was less than 12% of all AF cases in some series (4,10,13,14), but more than 30% in others (15.16). The prevalence of AF increases with the severity of congestive heart failure (HF) or valve heart disease. Figure 2. Prevalence of AF in 2 American epidemiological studies. Framingham presents Framingham Heart Study (9); CHS, Cardiovascular Health Study (10).B. PrognosisMiss of ischemic stroke in patients with non-ref averages 5% per year, which is 2 to 7 times the rate in people without AF (8,9,15,17-19) (Fig. 3) . Patients with AF (20) experience one in 6 strokes. Including transient ischemic attacks and clinically silent vascular brains detected radiographically, the rate of cerebral ischemia accompanying nonvalvular AF exceeds 7% per year (21-25). In the Framingham Heart Study, patients with rheumatic heart disease and AF had a 17-fold increased risk of stroke compared to age-appropriate controls (26), and the risk that could be assigned was 5 times higher than in patients with non-reticular AF (9). Among patients with AF from general procedures in France was the ALFA study (Etude en Activité Liberale sur le Fibrillation Auriculaire) 2.4% of thromboembolism incidence averaged 8.6 months of follow-up (15). The annual risk of stroke caused by AF increased from 1.5% in participants in the Framingham study aged 50 to 59 years to 23.5% in those aged 80 to 89 years (9). Overall mortality is approximately double in patients with AF compared to patients in normal sinus rhythm and is associated with the severity of underlying heart disease (8,11,18) (Figure 3). Figure 3. Relative risk of stroke and mortality in Patients with AF compared to patients without AF. The source data are from framingham heart study (11), regional heart study (8), whitehall study (8) and manitoba study (18).v. pathophysiological mechanismsa. Attea factors1. Pathology of atrium in patients with AFThe atrium of patients with persistent AF display structural abnormalities beyond those caused by underlying heart disease (27). Patches of fibrosis with juxtaposis of normal and sick atms may represent inhomogeneity of at-at-at-house refractory (28.29). Fibrosis or fat infiltration can also affect the sinus node and may be a reaction to inflammatory or degenerative processes that are difficult to detect. The role of inflammation in AF pathogenesis has not yet been evaluated, but histological changes consistent with myocarditis have been reported in 66% of biopsy samples in patients with solitary AF (29). Infiltration of the attic myocardium can occur in amyloidosis, sarcoidosis and hemochromatosis. Atria hypertrophy has been described as the main and sometimes the only histological feature (28). Progressive dilation of the atria has been demonstrated in patients with AF (30) and, like hypertrophy, may be either the cause or result of persistent af.2. AFTheories mechanisms of the AF mechanism include 2 main processes: increased automaticity in 1 or several rapidly depolarizing bearings and reentry involving 1 or more circuits (31.32). Rapid burning of atrial foci found in 1 or more excellent pulmonary veins may initiate AF in sensitive patients (3.33). Outbreaks also occur in RA and rarely in superior vena cava or coronary sinus (3,33,34). Focal origin seems to be more important in paroxysmal AF than in persistent AF. Ablation of these foci can be therapeutic (3). The multiwave hypothesis as an AF reentrant mechanism was advanced by Moe and colleagues (31.35), who suggested that fractionation of wave fronts as it spreads through the atrium leads to self-sustaining daughter ripples. The number of ripples present at any moment depends on the refraction period, weight and speed of the line in different parts of the atn. Although the activation patterns underlying ACEN's irregular electrical activity have traditionally been described as disordered or accidental, recent evidence has emerged that AF is spatially organized. Based on map studies of patients undergoing surgery (WPW), 3 patterns of induced AF (36) have been identified. Type I AF includes one wave of queue propagation over RA. Type II AF includes 1 or 2 wave fronts, and type III AF is characterized by several activation waves that spread in different directions. Finally, a better understanding of electrophysiological mechanisms will lead to the development of effective preventive measures (37).B. AV Conduction1. The general aspects of the Av node are usually a factor that limits the lead during AF. The compact AV node is located in front of the Koch triangle (38), surrounded by transient cells. There seem to be 2 distinct entrances of the atms to the AV node, posteriorly through crista terminalis and the front through the inter-room bulkhead. Studies on rabbit AV nodule preparations show that during AF, the propagation of impulses through the AV node into its bundle depends in part on the relative timing of the antenal and posterior septic activation inputs to the AV node (39). Other factors that affect the conduction through the AV node are its internal conduction and refractority, hidden conduction and autonomic tone.2. AV conduction in WPW syndrome Relevant pathways are muscle connections between the atrium and the ventricle, which have the ability to perform quickly. Guiding through the accessory path during AF can result in a very fast ventricular reaction, which can be fatal (2.40). Drugs such as digitalis, calcium channel antagonists and beta-blockers, which are usually administered to slow conduction through an AV node during AF, do not block the wiring through the accessory pathway and can even increase wiring, leading to hypotension or cardiac arrest (41). Patients who develop AF with a rapid ventricular response associated with hemodynamic instability resulting from guiding through the accessory pathway should undergo immediate electrical cardioversion. In the absence of haemodynamic instability or preexced ventricular response of intravenous prokaaíd and ibutilid, drugs of choice to achieve pharmacological cardioversion or block the conduction through the path of accessories.C. Myocardial and hemodynamic consequences of AFDuring AF, 3 factors can affect hemodynamic function: loss of synchronous attic mechanical activity, irregular ventricular response and inappropriately fast heart rate. A significant decrease in cardiac output can occur with loss of attic contraction, especially in patients with diastolic ventricular filling disorder, hypertension, mitral stenosis, hypertrophic cardiomyopathy (HCM) or restrictive cardiomyopathy. Changing RR intervals during AF can also lead to hemodynamic damage. The consistently fast speed of the atria may adversely affect the mechanical functioning of the atria (tachycardia-induced cardiomyopathy) (2.42). Such changes in atrial tissue may explain the delayed recovery of atrial contractility in patients after cardioversion to the sinus rhythm. Permanently increased (130 beats per m or higher in one study) (43) may cause dilated ventricular cardiomyopathy (2.43-46). It is critically important to recognize tachycardia-induced cardiomyopathy, since control of ventricular velocity can lead to a partial or even complete reversal of the myopathic process. In fact, HF can be the initial manifestation of AF. Various hypotheses have been proposed that explain cardiomyopathy mediated by tachycardia, Which include myocardial energy depletion, ischemia, calcium regulation abnormalities, and remodeling, but the actual mechanisms responsible for this disorder are still unclear (47).D. ThromboembolismAlthough ischemic stroke and systemic arterial occlusion in AF are generally attributed to left atrial embolism (LA), pathogenesis of thromboembolisy is complex (48). Up to 25% of af-related strokes can be caused by internal cerebrovascular disease, other cardiac sources of embolism, or atherotic pathology in the proximal aorta (49.50). Approximately half of elderly patients with AF have chronic hypertension (the main risk factor for cerebrovascular disease) (19) and approximately 12% harbor cervical carotid artery stenosis. However, carotid atherosclerosis is not significantly more common in stroke patients than in patients without AF and is probably a relatively small contributing factor (51).1. Pathophysiology of Thrombus FormationThrombus associated with AF arises most often in the addition of LA (LAA). This cannot be reliably examined by precordial (transtocratic) echocardiography (52), while transesophageal Doppler echocardiography provides a sensitive and specific method for assessing LAA function (53) and for detecting trommical material. Laa flow rates are reduced due to loss of organized mechanical contraction during AF (54.55). This reduced flow substrate within LA/LAA has been associated with spontaneous echo contrast, thrombus formation and embolic events (56-62). LAA flow rates are lower in patients with atteam flutter than what is usually seen with a normal sinus rhythm, but are higher than those with AF. Whether this represents a slightly lower prevalence of LAA thrombus and possibly a lower rate of thromboembolism associated with attic flutter is uncertain. In patients with AF, independent predictors of spontaneous echo contrast include LA size, LAA flow rate (56.63), left ventricular dysfunction (LV), fibrinogen level (62), hematocrit (61.62), and aortic atherosclerosis (61.62,64.65). This phenomenon may be an echocardiographic replacement for regional coagulopathy and, in the case of dense, clinical value for identifying patients with AF with a high risk of thromboembolism (64), but its usefulness for future stratification of the risk of thromboembolism beyond what has been achieved by clinical trials has not been established. Although conventional clinical treatment is based on the assumption that thrombus formation requires the continuation of AF for approximately 48 hours, thrombus echocardiography (TEE) at shorter intervals (66.67). Contrary to the prevailing view that systemic anticoagulation for 4 weeks leads to adherence to the endocardium and laa thrombus organization, TEE studies have verified the disappearance of thrombus in the majority of patients (68). Similar observations defined the transient nature of LA/LAA dysfunction in the conversion of AF, which provides a mechanical justification for anticoagulation for several weeks before and after successful cardioversion.2. Clinical implicationsTh because the pathophysiology of thromboembolism in patients with AF is uncertain, the mechanisms that connect risk factors with ischemic stroke in AF are also incompletely defined. The strong link between hypertension and stroke in AF is likely mediated primarily by an embolism that comes from LAA (49), but hypertension also increases the risk of non-cardiac stroke in AF (49.69). Hypertension in patients with AF is associated with reduced LAA flow rate and spontaneous echo contrast, which predisposes the patient to thrombus formation (63,64,70). Ventricular diastolic dysfunction can be the basis of the effect of hypertension on LA dynamics (71.72). The effect of advancing age to increase the risk of stroke in AF is multifactorial. In patients with AF, aging is associated with LA enlargement, reduced LAA flow rate, and spontaneous echo contrast, each predisposed to the formation of an LA thrombus (30,63,64). In addition, age is a risk factor for atherosclerosis, including complex aortic arch plaque, and is associated with stroke independently of AF (65). Lv systolic dysfunction predicts ischemic stroke in patients with AF who do not receive antithrombotic therapy (73-76).VI. Related conditions, clinical manifestations, and quality of lifeA. CA uses and associated conditions1. Acute causes of AFAF may be related to acute, temporary causes, including alcohol intake, surgery, electric shock, myocarditis, pulmonary embolism, other lung diseases and hyperthyroidism. Successful treatment of the underlying condition can eliminate AF. AF is an early postoperative complication of myocardial infarction and cardiac or

thoracic surgery.2. AF without related cardiovascular diseaseIn younger patients, approximately 30% to 45% of paroxysmal cases and 20% to 25% of persistent cases of AF occur as solitary AF (13,15,16).3. AF Associated with cardiovascular diseasesSpecific cardiovascular conditions associated with AF include valve heart disease (most commonly mitral), coronary artery disease (CAD) and hypertension, especially if LV hypertrophy (LVH) is present. Neurogenic AFSektovatin nervous system can trigger AF in sensitive patients through increased vagal or adrenergic tone. Many patients experience the onset of AF during periods of increased parasympathetic or sympathetic tone. Coumel (77) described a group of patients that he characterized as vagal or adrenergic forms of AF. Patients with Vagal or adrenergic AF are unusual, but if history reveals a pattern of the onset of AF with features of one of these syndromes, the clinician may choose substances more likely to prevent recurrent episodes.B. Clinical manifestationsaf may be symptomatic or asymptomatic, even in the same patient. Symptoms differ with ventricular speed, basic functional state, duration of AF and perception of individual patients. Dysrrhythmia may for the first time represent an embolic complication or exacerbation of HF. Most patients with AF complain of palpitations, chest pain, shortness of breath, fatigue, or head spins. The release of the atrial natriuretic peptide may be associated with polyuria. AF can lead to tachycardia-mediated cardiomyopathy, especially in patients who are not aware of arrhythmia. Syncope is a less common but serious complication that usually indicates sinus node dysfunction, chlopene aortic stenosis, HCM, cerebrovascular disease, or accessory AV pathway.C. Quality of lifeAkher stroke will certainly account for much of the functional disorders associated with AF, rhythm disorders also reduces quality of life. In the Studio Cohort Prevention of Stroke in Atrial Fibrillation (SPAF), the Functional Classification of the New York Heart Association developed for HF was an insensitive quality of life index in patients with AF (78). In another study (79) 47 (68%) 69 patients with paroxysmal AF considered dysrrhythmia disrupting their lives. This perception was not associated with the frequency or duration of symptoms. The AFFIRM (Atrial Fibrillation Follow-up investigation of the rhythm of management), which is still ongoing, compares the maintenance of the sinus rhythm with the speed control in patients with AF, which addresses many aspects of quality of life, as well as a smaller study of PIAF (Pharmacological Intervention in Atrial Fibrillation) (80). In selected patients, radiofrequency catheter ablation of the AV node and pacemaker improved the quality of life score compared to medical therapy (81-86). Long-term oral anticoagulant therapy, which includes frequent blood tests and multiple drug interactions, is another factor with important implications for the quality of life of patients with AF. Analysis of the decision found that only 61% of the 97 patients preferred anticoagulant therapy over any treatment (87), a significantly smaller proportion than those recommended for treatment according to published guidelines. VII. Clinical trial. Minimum assessment of a patient with AF1. Clinical history and physical examinationThe initial assessment of a patient with suspected or proven AF involves characterising the arrhythmia pattern as paroxysmal or persistent, determining its cause and defining related cardiac and extracardial factors (Table 1). Careful history will result in a well-planned, focused examination that serves as an effective guide to therapy (2). An AF patient may usually be examined and treatment may occur initiated in 1 outpatient meeting. The delay occurs when the rhythm has not been specifically documented and further monitoring is required. Table 16135. Minimal and additional clinical trial of patients with atrial fibrillationAF indicates atrial fibrillation; LV, left ventricle; MI, myocardial infarction; RV, right ventricle; LA, left hall; and AV, atrioventricular. Type IC refers to the classification of Vaughan Williams' antiarrhythmic drugs (see Table 2). Minimum rating1. Medical history and physical examination, define• Presence and nature of symptoms associated with AF• Clinical type of AF (first episode, paroxysmal, persistent or permanent)• Onset of the first symptomatic attack or date of discovery of AF• Frequency, duration, accelerating factors and methods of cessation of AF• Reactions to any pharmacological agents that have been administered• Presence of any underlying heart disease or other reversible conditions (e.g. hyperthyroidism or alcohol consumption)2. Electrocardiogram to identify• Rhythm (AF verified)• NN HYPERTROPHY• Duration of P-wave and morphology or fibrillation wave• Preexcetation• Beam branch block• Previous MI• Other atrial arrhythmias• Measurement and observance of RR, QRS and QT intervals in conjunction with antiarrhythmic drug therapy3. Chest X-ray, to evaluate• Pulmonary parenchyma if clinical findings indicate abnormality• Pulmonary vasculature if clinical findings indicate abnormality4. Echocardiogram to identify• Chloústic heart disease• Left and right atrial size• LV size and function• Peak RV pressure (pulmonary hypertension)• NN HYPERTROPHY• LA thrombus (low sensitivity)• Pericardial disease5. Blood tests for thyroid function• For the first episode of AF, when the ventricular ratio is difficult to control, or when AF is unexpectedly repeated after cardioversionPreparation• One or more tests may be required1. Exercise• When it comes to speed control adequacy (permanent AF)• Reproduce exercise-induced af• Elimination of ischemia prior to treatment of selected patients with an antiarrhythmic drug type IC2. Holter monitoring or recording of an event• If a given diagnosis of arrhythmia is called into question• As a means of evaluating speed control3. Transesophageal echocardiography• To identify LA thrombus (in LA encore)• To conduct cardioversion4. Electrophysiological study• To clarify the mechanism of wide-angle complex tachycardia• To identify predisposing arrhythmias, such as atrial flutter or paroxysmal supraventricular tachycardia• Searching for sites for therapeutic ablation or AV conductive block/modificationPhymic examination may indicate AF based on irregular pulse, irregular cervical venous pulsation and variation in the volume of the first heart sound. The examination may also reveal related heart disease, myocardial abnormalities, or HF. The findings at the examination are similar in patients with ated flutter, with the exception of Regular and rapid venous oscillations can occasionally be visible in the cervical pulse.2. AF diagnosis requires ECG documentation with at least one leading ECG record during dysrrhythmia, which may be facilitated by reviewing emergency department records, Holter monitoring, or transtelepopic or telemetry records. A portable ECG recording tool can help establish a diagnosis in cases of paroxysmal AF and provide a permanent record of ECG dysrhythmia. If episodes are frequent, 24h Holtermonitor can be used. If episodes are rare, then an event recorder that allows the patient to transmit an ECG to the recording equipment when arrhythmia occurs may be more useful.B. Further investigations of selected patients with AFAdditional examination may include Holter monitoring and exercise testing, transesophageal echocardiography, and/or electrophysiological studies. See Table 1. VIII for details. ManagementMemotic problems in the treatment of patients with AF are related to arrhythmia itself and to the prevention of thromboembolism. In patients with persistent AF, there are basically 2 ways to manage dysrrhythmias: restore and maintain a sinus rhythm or allow AF to continue and ensure that ventricular speed is controlled.A. Rhythm control vs. Heart rate controlReasons to restore and maintain sinus rhythm in patients with AF include symptom relief, prevention of embolism, and avoidance of cardiomyopathy. B Cardioversion1. The basis for cardioversion of AFCardiversion is often carried out optionally to restore the sinus rhythm in patients with persistent AF. However, the need for cardioversion can be immediate when arrhythmia is the main factor responsible for acute HF, hypotension or deterioration of angina in a patient with CAD. However, cardioversion carries a risk of thromboembolism if anticoagulant prophylaxis is not started prior to the procedure and this risk appears to be greatest if arrhythmia is present for more than 48 h.2. Methods of cardioversionCardioverze can be achieved with the help of drugs or electric shocks. Drugs were commonly used before electric cardioversion became standard procedure. The development of new drugs has increased the popularity of pharmacological cardioversion, although some disadvantages remain, including the risk of drug-induced torsion de pointes ventricular tachycardia or other severe arrhythmias. Pharmacological cardioversion is still less effective than electric cardioversion, but the second requires conscious sedation or anesthesia, while the first does not. The risk of thromboembolism or stroke does not differ between pharmacological and electrical cardioversion. Therefore, the recommendations for anticoagulation are the same for both methods.C. Pharmacological cardioversion seems to be most effective at initiation within 7 days after the onset of AF (88-91). The majority of these patients paroxysmal AF, the first documented episode of AF or an unknown AF pattern at the time of treatment. (See section III of the classification.) A large proportion of patients with recent onset of AF have spontaneous cardioversion within 24 to 48 hours (92-94). This is less likely to occur if AF persists for more than 7 days. The relative effectiveness of different drugs varies for pharmacological cardioversion of AF and attic flutter, but many pharmacotherapy studies for AF involved patients with attic flutter. Efficacy affects dose, route and rate of administration. Reference is made to vaughan Williams' classification of antiarrhythmic drugs (95), which has been modified to include recently available medicines (Table 2). A summary of the recommendation is given in Tables 10-12 (see Section IX-B Recommendations for Pharmacological and Electrical Cardioversion of AF, Tables 10-12). Although the clinical use of listed antiarrhythmic drugs has been approved by regulatory agencies, therapeutic use for AF has not been reported or approved in all cases in each country. The recommendations in this document do not necessarily meet government regulations and labelling requirements. Table 26135. Vaughan Williams Classification of Antiarrhythmic Drug ActionsModified with permission of Vaughn Williams EM. Classification of the antiarrhythmic effect as reassessed after ten years of new drugs. J Clin Pharmacol 1984;24:129-47, © 1984 by Sage Publications Inc. (95) to include compounds introduced after publication of the original classification. Type IADisopyramideProcainamideQuinidineType IBLidocaineMexiletineType ICFlecainideMoricizinePropafenoneType IIBeta-blockers (e.g. propranolol)Type IIIAmiodaroneBretyliumDofetilideIbutilideSotalolType IV Calcium channel antagonists (e.g. verapamil and diltiazem)A common problem related to pharmacological cardioversion is whether the antiarrhythmic drug should be started in a hospital or on an outpatient scale. The main problem is the potential for serious side effects, including torsade de pointes ventricular tachycardia. With the exception of those involving low doses of oral amiodiaron (96), virtually all pharmacological cardioversion studies have been limited to hospitalized patients.D. Electric cardioversion of cardioversionDirect involves electric shock synchronized with internal heart activity. This ensures that no electrical stimulation occurs during the vulnerable phase of the heart cycle (97). Successful cardioversion of AF depends on the nature of the underlying heart disease and the current density delivered to the attic myocardium. This, in turn, depends on the voltage of the capacitor defibrillator, the output course, the size and position of the electrode paddles and the transtoracal impedance. In a randomised controlled trial of 301 subjects undergoing selective external cardioversion, patients were enrolled in anterior-lateral apex and right infracular) or anterior-posterior (thoracic and left scapula) paddle position (98). Energy intensity was lower and overall success was greater with front and rear configuration (87%) than front and side alignment (76%). Cardioversion is carried out with a patient who has fasted and under adequate anesthesia. Short-acting anesthetics are preferred because patients with cardioversion are suitable for daily care and should recover quickly after the procedure (99). An initial shock of 100 J is often too low, and an initial energy of 200 J or higher is recommended for electric cardioversion AF. Devices that supply current with a biphasic course appear to achieve cardioversion at lower energy levels than devices that use a monophase course. The primary success rate measured 3 days after cardioversion in 100 consecutive subjects was 86% (100); this increased to 94% when the procedure was repeated during treatment with quinidine or disopyramide. Only 23% of patients remained in the sinus rhythm after 1 year and 16% after 2 years; In those who relapsed, repeated cardioversion with antiarrhythmic drugs resulted in a sinus rhythm in 40% and 33% after 1 and 2 years. In patients who relapsed again, the third cardioversion resulted in a sinus rhythm in 54% after 1 year and 41% after 2 years. Thus, the sinus rhythm can be restored in a substantial part of patients according to direct cardioversion, but the rate of relapse is high if antiarrhythmic drug therapy is administered simultaneously. Cardioversion of patients with an implanted pacemaker and defibrillator is safe if appropriate measures are taken. The device should be interrogated immediately before and after cardioversion in order to verify the appropriate function. Paddles used for external cardioversion should be placed as far away from the device as possible, preferably in the front and rear configurations. The risks of electric cardioversion are mainly related to embolic events and cardiac arrhythmias. Thromboembolic events have been reported in 1% to 7% of patients who did not recognise anticoagulation prior to cardioversion (101,102). Various short arrhythmias may arise, especially ventricular and supraventricular premature beats, bradycardia and short periods of cavity arrest (103). Ventricular tachycardia and fibrillation may be precipitated in patients with hypocalaemia or digitalis intoxication (104,105). Slow ventricular reaction to AF in the absence of drugs that slow AV nodal conduction may indicate a conduction defect. The patient should be examined before cardioversion with regard to these problems to prevent symptomatic bradycardia (106). After cardioversion (107-108), transient elevation of the ST segment may occur on the ECG, and levels of creatine kinase-MB in the blood may increase even without obvious damage to the myocardium. Prophylactic drug therapy to prevent early recurrence of AF should be assessed individually for each patient. Should (especially early relapse), antiarrhythmic therapy is recommended in conjunction with a second attempt. Another cardioversion has limited value. In highly symptomatic patients, rarely repeated cardioversion can be an acceptable approach.E. Maintaining Sinus Rhythm1. Pharmacological therapy to prevent recurrence of AFMaintenance of sinus rhythm is relevant in patients with paroxysmal AF (in which episodes spontaneously end) and persistent AF (for which electrical or pharmacological cardioversion is required to restore sinus rhythm). Whether paroxysmal or persistent, AF is a chronic disorder, and relapse is likely at some point in most patients (Fig. 4) (109,110). Therefore, prophylactic treatment with antiarrhythmic drugs is often necessary. The aim of maintenance therapy is to suppress symptoms and sometimes prevent cardiomyopathy induced by tachycardia due to AF. It is not yet known whether maintaining a sinus rhythm prevents thromboembolism, HF, or death (80,111). Figure 4. Survival without arrhythmia after electric cardioversion in patients with persistent atrial fibrillation. The lower curve represents the result after a single shock, when no prophylactic drug therapy was administered. The upper curve shows the result with repeated electrical cardioversions in conjunction with antiarrhythmic prophylaxis of drugs. ECV indicates electrical cardioversion; SR, sinus rhythm. Reproduced with permission of van Gelder et al., Arch Intern Med 1996;156:2585–92, © 1996, American Medical Association (110). Repetition of AF is not equivalent to failure of treatment. In several studies (112,113), patients with recurrent AF often decided to continue drug treatment. Sometimes reducing the burden of arrhythmia can be a partial success, while in other patients any recurrence of AF may seem unbearable. However, most patients with AF eventually experience recurrence. Risk factors for frequent recurrence of paroxysmal AF (more than 1 episode per month) include female sex and underlying heart disease (114). In persistent AF, 4-year arrhythmia-free survival was less than 10% after single-shock electric cardioversion without prophylactic drug therapy (110). Recidivology predictors at this interval included hypertension, age greater than 55 years and duration of AF longer than 3 months. In this study, serial cardioversion and prophylactic drug therapy resulted in freedom from recurrent AF in only approximately 30% of patients (110). Relapse predictors included an age greater than 70 years, an AF duration of more than 3 months, and HF (110).2. General approach to antiarrhythmic drug therapyPreceeding administration of any antiarrhythmic agent, reversible cardiovascular and non-cardiovascular af precipitation should be addressed. Prophylactic treatment is rarely indicated in the case of the first detected episode of AF and can also be avoided in patients with less frequent and well-tolerated paroxysmal AF. Similarly, when Uncommon and tolerated, patients experiencing breakthrough arrhythmias may not require a change in antiarrhythmic drug therapy. Beta-blockers can be effective in patients who develop AF only during exercise, but few patients have one specific cause of incitement for all episodes of AF and most do not retain a sinus rhythm without antiarrhythmic drug treatment. In patients with solitary AF, a beta-blocker may be tested first, but flecainid, propaphenone and sotalol are particularly effective. Amiodal and dophetilide are recommended as an alternative treatment. Quinidine, prokaaíran, and disopyramide are not favored if amiodiaron fails or is contraindicated. Anticholinergic activity of long-acting disopyramide is a relatively attractive option for patients with a penchant for vagally induced AF. Propafenone is not recommended in vagally mediated AF because its (weak) internal beta-blocking activity can exacerbate this type of paroxysmal AF. In patients with adrenergic-mediated AF, beta-blockers are the first line of treatment, followed by sotalol and amiocarone. In patients with adrenergically mediated solitary AF, amiocarone should be selected later in the pharmacotherapy sequence due to its potential toxicity (see Fig. 11, Section IX). Overall, when treatment with a single drug fails, combinations of antiarrhythmic drugs can be tried. Useful combinations include beta-blocker, sotalol or amio aaron plus type IC agent. The drug, which is initially safe, can become proarrhythmical when the patient develops CAD or HF or begins to take other drugs that in combination can be arrhythmogenic. Therefore, the patient should be warned about the potential importance of such symptoms as syncope, angina or shortness of breath, and warned against the use of non-cardiatic drugs that can prolong the QT interval. A useful source of information is the website antiarrhythmic drug treatment varies according to the agent and factors of the patient. Prospective data from studies on upper limit increases in QRS duration or prolongation of QT-induced drugs are not available. The following recommendations are consensus writing committee. For IC-type drugs, the QRS extension should not exceed 150% of the QRS duration before treatment. Exercise testing can be useful for detecting QRS extensions that occur only at rapid heart rate (use-dependent line slowdown). For type IA or type III drugs, with the possible exception of amiodarone, the corrected QT interval in the sinus rhythm should remain below 520 ms. During monitoring, plasma levels of potassium and magnesium and kidney function should be regularly checked, since renal insufficiency leads to the addition of the drug and predisposes to proarrhythmia. In individual patients, serial non-invasive tests may be suitable for reassessing LV function, especially if clinical HF develops during treatment with AF.3. Pharmacological substances to maintain Sinus RhythmFourteen drug prophylaxis studies involving patients with paroxysmal AF have been published, and 22 studies of prophylaxis of sinus rhythm drugs have been published in patients with persistent AF. Comparative data are not sufficient to allow subdimatism by the drug or etiology. Individual drugs and doses for maintaining the sinus rhythm are given in Table 3. Table 36135. Typical doses of drugs used to maintain a sinus rhythm in patients with atrial fibrillation**Medication*Daily dosePotential side effectsGI indicates gastrointestinal; AV, atrioventricular; and HF, heart failure.*Medications are listed alphabetically.**The drugs and doses listed here have been determined by consensus based on published studies.†A dose of 600 mg per day is usually given for one month or 1000 mg per day for 1 week.‡The dose should be adjusted for kidney function and QT-interval response during the hospital initiation phase. Amiocarone†100-400 mgPhoto, pulmonary toxicity, polyneuropathy, GI agitation, bradycardia, torsade de pointes (rare), liver toxicity, thyroid dysfunctionDisopyramide400-750 mgTorsade de pointes, HF, glaucoma, urinary retention, dry mouthDofetilide‡500-1000 mcgTorsade de pointesFlecainide200-300 mgVentricular tachycardia, congestive HF, improved av nodal conduction (conversion to atrial flutter)Procainamide100 0-4000 mgTorsade de pointes, lupus-like syndrome, GI symptomsPropafenone450-900 mgVentricular tachycardia, congestive HF , increased AV nodal conduction (conversion to attic flutter)Quinidine600-1500 mgTorsade de pointes , GI agitation, increased AV nodal linesSotalol‡240-320 mgTorsade de pointes, congestive HF, bradycardia, exacerbation of chronic obstructive or bronchospastic pulmonary disease4. Out-of-hospital initiation of antiarrhythmic drugs in patients with AFRežistika for out-of-hospital initiation or intermitary use of antiarrhythmic drugs varies in patients with paroxysmal and permanent AF. In patients with paroxysmal AF, the goal is to stop the attack to prevent recurrence, or a combination of both. In patients with persistent AF, the goal of out-of-hospital initiation of treatment is to achieve pharmacological cardioversion of AF, thereby eliminating the need for electrical cardioversion, or increasing the success of electrical cardioversion and preventing premature recurrence of AF. There is little prospective data on the safety of outpatient initiation of antiarrhythmic drug therapy. Proarrhythmia (Table 4) is rare in patients with normal ventricular function and baseline QT intervals (41,115) without deep bradycardia. Until there is a suspicion of dysfunction of the sinus or AV node, propafenone or flecainid can be started outside the hospital. Sudden death associated with idiopathic ventricular fibrillation in the structurally normal heart may occur in patients with Brugad syndrome, hereditary heart disease characterized by an increase in ST-segment in the right precordial ECG conduction and often accompanied by right Block. Type I antiarrhythmic drugs can unmask this condition (116,117). Before starting treatment with these substances, a beta-blocker or calcium channel antagonist should be administered to prevent rapid AV conduction or 1:1 AV conduction if attic flutter develops (118-122). Since termination of paroxysmal AF with flecainide or propaphenone may be associated with bradycardia due to sinus node or AV node dysfunction, initial conversion should be performed in a hospital before the patient is declared fit for outpatient pill-in-the-pocket use of these substances for relapse conversion. In patients with symptomatic sinus syndrome, AV conduction disorders or bundle branch block, non-hospital discontinuation of treatment should be avoided. Table 46135. Types of proarrhythmia during treatment with various antiarrhythmic drugs for atrial fibrillation or atrial flutter according to Vaughan Williams ClassificationVW suggests Vaughan Williams classification of antiarrhythmic drugs (95). VF refers to ventricular fibrillation; AF, atrial fibrillation.A. Chamber proarrhythmia•Torsade de pointes (VW type IA and type III drugs)•Permanent monomorphic ventricular tachycardia (usually VW IC drugs)•Permanent polymorphic ventricular tachycardia/VF without long QT (types VW IA, IC and III drugs)B. Atrial proarrhythmia•Recidivism provocation (probably VW IA, IC and III drugs)•Conversion of AF to flutter usually with VW IC drugs)•Increase in defibrillation threshold (potential problem with VW IC drugs)C. Impulse conduction or impulse formation abnormalities•Acceleration of ventricular frequency during AF ((VW type IA and IC drugs)•Accelerate the route through the accessory pathway (digoxin, intravenous verapamil or diltiazem)•Dysfunction of sinus nodes and atrioventricular block (almost all drugs)Sotalol can be started in outpatient patients with little or no heart disease if the basic incorrect QT interval is less than 450 ms , serum electrolytes are normal and no risk factors for type III proarrhythmia are present. Safety is greatest when sotalol is triggered when the patient is in a sinus rhythm. Amiotarone can usually be administered safely on an outpatient date, even in patients with persistent AF (123), but hospital loads may be preferable if an earlier restoration of the sinus rhythm is needed, as in patients with HF. Some treatment regimens include administration of either 600 mg daily for 4 weeks (123) or greater than or equal to 1 g per day for 1 week (124) followed by lower maintenance doses. Quinidine, prokainamide, and disopyramide should generally not be started from the hospital. An exception can be made for disopyramide in patients without heart disease who have a normal QT interval. Currently, standards for the use of dophetilide do not allow an out-of-hospital initiation. Transtelephonic monitoring or other ECG monitoring methods may be used to monitor conduction failures as pharmacological antiarrhythmic therapy is patients with AF. Specifically, the PR interval (flecainid, propaphenone, sotalol and amiodalone), the duration of QRS (flecainid and propafenone) and the QT interval (sotalol, amiodalone and disopyramide) should be measured. In general, antiarrhythmic drugs should be started at a relatively low dose with titration up as needed and with each dose change there should be a reassessment of the ECG. The dose of other drugs used to regulate the rate should be reduced approximately 6 weeks after the start of amiodarone and stopped if the speed slows down excessively. Concomitant drug therapy should be closely monitored and both the patient and the doctor should be alerted to possible harmful drug interactions.5. Recurrence of AF After cardioversion: The implications for drug treatment have long been known that most AF relapses occur within the first month after electrical cardioversion, recent research with indoor atteam cardioversion (125) and day-to-day postconversion studies (126) have shown several patterns of recurrence (Figure 5). In some cases, there is a complete failure of anti-current counter-resistance, which would provoke even one isolated sinus or ectopic atrial, which is equal to a high threshold of atrial defibrillation. In others, AF appears within 1 to 2 minutes after the sinus rhythm period (127,128). Sometimes the recurrence is delayed from 1 day to 2 weeks (126) or more after shock. Complete shock failure and immediate recurrence occur in approximately 25% of patients undergoing electrical cardioversion, and subaute relapses occur approximately in the same proportion within 2 weeks (127). Figure 5. A hypothetical illustration of cardioversion failure. Three types of relapses after an electric cardioversion persistent AF are indicated the effectiveness of drugs differs in the strengthening of shock conversion and suppression of ecv relapses indicates external cardioversion; IRAF, the first repetition of AF after cardioversion. Modified with the permission of Van Gelder and Co. (129). Available data suggest that initiating pharmacological treatment before electrical cardioversion (Table 5) may increase immediate success and suppress early relapses. As a result, it seems advisable to determine the therapeutic concentrations of the drug in plasma at the time of cardioversion, and for several weeks thereafter optimize the chance of success. After cardioversion to the sinus rhythm, patients receiving drugs that prolong the QT interval should be monitored in the hospital for 24 to 48 hours to evaluate the effects of slowing heart rate and allow rapid intervention in the event that develops torsade de pointes. Pharmacological agents can be started outside the hospital or in the hospital immediately before the electrical cardioversion. Pre-treatment risks include the possibility of a paradoxical increase in the defibrillation threshold that occurred with flecainide (130); accelerated ventricular speed when handling type IA or IC drugs in the absence of an AN nodule blocker and ventricular proarrhythmias (Table 4). Table 56135. Pharmacological treatment before cardioversion in patients with persistent atrial fibrillation: Effects of various antiarrhythmic drugs on acute and subacute outcome of transtocratic direct stream ShockEnhance Conversion dc shock and prevention of IRAF * Suppress SRAF and maintenance therapy ClassRecommendation ClassLevel evidenceAll drugs (excluding beta-blockers and amiodarone) should be started in the hospital. IRAF indicates immediate recurrence of atrial fibrillation; SRAF, subaute recurrence of atrial fibrillation; and DC, DC.*Drugs are listed alphabetically within each referral class. EffectiveAmiodaroneAll drugs in class I recommendations (except ibutilide) plus beta-blockersIBFlecainideIbutilidePropafenonePropafenone+verapamilQuinidineSotalolUncertain/unknownBeta-blockers DiltiazemIIbBDisopyramideDofetilideDiltiazemVerapamilDofetilideProcainamideVerapamilPretreatment with pharmacological agents is most appropriate in patients who have not previously responded to electrical cardioversion, and in patients who have developed immediate or subaute recurrence of AF. Pre-treatment is optional in patients with late relapse and in patients who have undergone initial cardioversion persistent af (Fig. 10 and 11; see section IX).6. Selection of antiarrhythmic agents in patients with certain heart diseases. Heart failurePatients with congestive HF are particularly susceptible to the ventricular proarrhythmic effects of antiarrhythmic drugs. Randomised studies have demonstrated the safety of amiodarone and dofetilide (given alone) in patients with HF (132,133) and these are recommended drugs for maintaining the sinus rhythm (Fig. 11; see Section IX).b. Coronary artery disease In stable CAD patients, beta-blockers can be considered the first, but their use is supported by only 2 studies (134-135); efficacy in maintaining a sinus rhythm in patients with persistent AF after cardioversion are not conclusive (134). Sotalol has considerable beta-blocking activity and can therefore be chosen as an initial antiarrhythmic agent in patients with AF with coronary artery disease because it is associated with less long-term toxicity than amiodaron (Fig. 11; see Section IX). Both Sotalol and amio aaron have adequate short-term safety profiles and amio aaron may be preferred in patients with HF (136-138). Flecainid (139) and propaphenone are not recommended in these situations. Quinidine, prokaaímin, and disopyramide are considered the third line of treatment of choice in patients with CAD. In view of the results of the DIAMOND-MI study (Danish investigation of arrhythmias and mortality from Dofetilide in myocardial infarction), dophetilide can be considered a second line rather than an antiarrhythmic third-line agent, but this study included selected patients with myocardial infarction for whom the antiranthytmic benefit balanced the risk of prophythmic toxicity.c. Hypertensive heart with LVH are at increased risk of developing torsade de pointes associated with early ventricular subpolarization (140-142). A drug that does not extend the QT interval is therefore preferable as a first-line therapy, and in the absence of CAD or marked ventricular hypertrophy, propafenone and spotetinide are a reasonable choice (Fig. 11; Section IX). Amiobar prolongs the QT interval, but carries a very low risk of ventricular proarrhythmia; Its extracardiatic toxicity profile pushes it into the second line of therapy in these individuals, but amiocarone becomes the first line of treatment if LVH is indicated to be present. When amiodiaron and sotalol either fail or are inappropriate, disopyramide, quinine, or procainamide can be used as alternatives.d. WPW SyndromeRadiofric ablation pathway accessory is the preferred therapy in patients with preexcitation syndromes and AF, but antiarrhythmic drugs can be useful. Digoxin should be avoided due to the risk of paradoxical acceleration of the ventricular frequency during AF. Beta-blockers do not reduce the lead over accessory paths during pre-term periods af.7. Non-pharmacological correction of AFa. Surgical ablation Based on map studies of animals and humans AF, Cox et al (143-146) developed surgery (maze surgery), which controls AF in more than 90% of selected patients. The exact mechanism has not been established convincingly, but the creation of leadership barriers within RA and LA limits the amount of myocardial available to propagate reentrant queue waves, thereby inhibiting sustained AF. Incisions surrounding pulmonary veins may prevent the initiation of AF by isolating potentially arrhythmogenic foci near the pulmonary veins from the rest of the attic or by isolating attic areas with the shortest refractories. Modifications to cox maze surgery include circumnavial vein enveloping surgical incisions in LA and radial incisions in both atriums that connect mitral and tricuspid valve annal (147-149). Surgical operations for AF have been successfully combined with operative correction of various structural heart conditions. In patients with highly symptomatic AF who require open heart surgery for a valve, ischemic or congenital heart defect, concomitant maze surgery for AF or flutter should be considered, even if this entails an additional risk. The mortality rate of isolated maze operations is less than 1%, but mortality is higher when the procedure is combined with other types of surgical corrections. The morbidity associated with maze surgery includes the implications common to moderate sternotomia and cardiopulmonary bypass surgery, as well as the risk of short-term fluid retention (due to reduced release of atrial natriuretic peptide), transient reduction in la and ra transport function, and early postoperative atrial tachyarrhythmia. In addition, when the blood supply to the sinus node is interrupted, dysfunction of the sinus node require permanent implantation of the pacemaker. Progressive iterations of these operations reduced the risk of this complication to less than 10% .b. Catheter ablation Based on the success of surgical approaches to AF, several catheter ablation strategies have been proposed to create similar effects (150-152). Ablation strategies limited to RA produce marginal improvement (150), while linear ablation in LA has been more successful. Recognition that outbreaks of AF triggering often originate in the pulmonary veins has led to ablation strategies that target this zone or electrically isolate the pulmonary vein from LA. Other sites of arrhythmogenic deposits were found in superior vena cava, RA and LA, and coronary sinus. Ablation of these foci eliminates or reduces the frequency of recurrent AF in more than 60% of patients, but the risk of recurrent AF after focal ablation is still 30% to 50% during the first year and even higher if it is more than 1 pulmonary vein. Therefore, many patients continue to require antiarrhythmic drug therapy after ablative AF therapy (153). Potential complications of catheter ablation for AF include systemic embolism, pulmonary venous stenosis, pericardial effusion, cardiac tampowide and phrenic nerve paralysis. Thus, although these procedures have produced promising results, they have not yet been widely applied. Attic flutter can develop not only as a pronounced arrhythmia, but also during antiarrhythmic drug therapy AF, especially in ic type agents. Catheter ablation is more effective than antiarrhythmic drugs for the treatment of attic flutter, reducing the recurrence rate from 93% to 5% when used as a first-line therapy (37). In addition, the risk of developing AF may also be lower after catheter abling of attic flutter than in pharmacological therapy (29% vs. 60% during the first year).c. Suppression of AF pacingAlthough atrium-based pace is associated with a lower risk of AF and stroke than chamber-based switching for patients requiring pacemakers for bradyarrhythmias, using switching as primary therapy to prevent recurrent AF has not been validated.d. Inner atrial Cardiothoracic/ DefibrillatorsThere has been an interest in internal cardioversion of AF for the past 10 years. Attempts have been made to find shock waveforms that reduce the energy requirements for cardioversion, so the shock is awakened awakened. One cardiooverter (154) capable of atteam sensing and cardioversion, as well as sensing and crossing ventricles, was evaluated in 290 patients with AF. The conversion rate to sinus rhythm was 93%. Another device, with dual-chamber sensing, pace, and cardioversion/defibrillation capabilities and a maximum power of 27 J, is a ventricular defibrillator with atrial cardioversion capabilities. It was designed to treat attic and ventricular arrhythmias with crossing modality before delivering low-energy shocks. Potential candidates for the attea especially those with occasional episodes of poorly tolerated AF, are often suitable for catheter ablation.F. Rate Control during AF1. Pharmacological approach An alternative to maintaining a sinus rhythm in patients with paroxysmal or persistent AF is the control of ventricular velocity. In a recent randomized study, therapeutic speed versus rhythm control strategies yielded similar clinical results with regard to symptoms in patients with AF, but exercise tolerance was better with rhythm control (111). The results of other studies comparing these 2 strategies are not yet available (80). Speed is generally considered controlled if the ventricular response is between 60 and 80 beats per hour at rest and between 90 and 115 beats per hour during moderate exercise (156,157). Heart rate variability during AF provides additional information about the state of the autonomic nervous system, which can have independent prognostic consequences (158-161). Negative chronotropic therapy AF is mainly based on depression conduction through the AV node. Sinus bradycardia and heart block occur in some patients with paroxysmal AF, especially in elderly patients, as a side effect of pharmacological intervention with beta-blockers, digitalis glycosides or calcium channel antagonists. Table 6 outlines substances that can be administered to control the ventricular response to AF in an emergency environment. Table 7 outlines drugs that block av nodal lines; These drugs can be used to achieve speed control both at rest and during exercise or other types of cardiovascular stress. The combination of drugs is often necessary to achieve speed control in patients with AF in acute and chronic environments. Therapy often requires careful titration of the dose, and in some patients symptomatic bradycardia appears, which requires a constant pace. Table 66135. Intravenous pharmacological agents for heart rate control in patients with atrial fibrillationAssociation*Charging DoseSNavlateMajor side effectsClass HF recommendations indicate heart failure.*Drugs are listed alphabetically in each class of recommendations.**Type I in congestive HF.†Type IIb in congestive HF.Only representative members of the beta-adrenergic antagonist type are included in the table, but other, similar substances could be used for this indication at appropriate doses. Diltiazem0.25 mg/kg IV over 2 min2-7 min5-15 mg per hour of infusionHypotenzion, heart block, HFI†Esmolol‡0.5 mg/kg over 1 min0.05-0.2 $mg \cdot kg^{-1} \cdot min^{-1}$Hypotenzion, heart block, bradycardia, asthma, HFIMetoprolol‡2.5-5 mg IV bolus over 2 min; up to 3 doses5 minNAHypotenzion, heart block, bradycardia, asthma, HFI†Propranolol‡0.15 mg/kg IV5 minNAHypotension, heart block, bradycardia, asthma, HFI†Veramilpa0.075-0.15 mg/kg IV over 2 min3-5 minNAHytenion, heart block, HFI†Digoxin0.25 mg IV every 2 hours, up to 1.5 mg2 h0.125-0.25 mg dailyDigitalis toxicity, heart block, bradycardiaIIb**Table 76135. Recommendations for the use of oral pharmacological products for Speed control in patients with atrial fibrillationDrug*Loading DoseOnsetUsual Maintenance dose**Serious side effectsHF recommendations indicate heart failure; PO, by mouth; NA, not applicable; HF, heart failure; and BID, twice a day.*Drugs are listed alphabetically within each class of recommendations.**Recommended maintenance doses are commonly necessary, but higher doses may be appropriate in some patients.†Style contains representative members of the beta-blocking drug type, but other, similar substances could be used for this indication at appropriate doses. Digoxin0.25 mg PO every 2 hours; up to 1.5 mg2 h0.125-0.375 mg daily Toxicity of prodigitalis, heart block, bradycardiaIDiltiazemNA2-4 h120-360 mg daily in divided doses; possibility of slow releaseHypotenzion, heart block, HFIMetoprolol†NA4-6 h25-100 mg BIDHypotension, heart block, bradycardia, asthma, HFIPropranolol†NA60-90 min80-240 mg daily in divided dosesHypotenion, heart block, bradycardia, asthma, HFIVerapamilNA1-2 h120-360 mg daily in divided doses; slow release availableHypotension, heart block, HF, digoxin interactionIAmiodarone800 mg daily for 1 wk 600 mg daily for 1 wk 400 mg daily for 4-6 wk1-3 wk200 mg dailyPulon toxicity, skin toxicity, skin discoloration, hypothyroidism, corneal foci, optical neuropathy, warfarin interaction, proarrhythmiaIIb2. Non-pharmacological regulation of AV Nodal Conduction and PacingThat the ventricular tempo prolongs the AV nodal refracter period due to hidden retrograde penetration, eliminates ventricular cycles longer than the length of the tempo cycle and reduces the number of short ventricular cycles associated with rapid AV conduction during AF. As a result, the ventricular tempo can be used as a strategy to reduce the irregularity of the ventricular rhythm (162). This modality can be useful for patients with pronounced variability of ventricular rates and for those who develop resting bradycardia during treatment with drugs prescribed to control rapid ventricular rates with exertion. However, the exact role of pacemaker therapy for regulating ventricular velocity in patients with AF remains controversial.3. Nonfarmacological/AV Nodal AblationAV nodal ablationav and permanent implantation of the pacemaker are highly effective means of improving symptoms in patients with AF who develop symptoms associated with rapid ventricular pace during AF that cannot be adequately controlled by antiarrhythmic or negative chronotropic drugs (83,84,86,163). AV nodal ablation is especially useful when excessive ventricular velocity provokes a decrease in ventricular systolic function mediated by tachycardia despite appropriate medical therapy. Limitations of AV nodal ablation include a persistent need for anticoagulation, loss of AV synchrony, and lifelong pacemaker addiction. There is a small but real risk of sudden death, largely due to torsade de pointes (164). In addition, the ablation of the AV management system could exclude or limit the later use of newer Treatment. Patients with diastolic ventricular disorder who are most dependent on AV synchronization to maintain cardiac output (such as HCM or restrictive cardiomyopathy) may experience persistent symptoms after AV nodal ablation and permanent implantation of the pacemaker. Therefore, patients should be instructed on each of these considerations before treating this irreversible treatment. The use of catheter ablation to modify av nodule lines has been reported by removing posterior atial inputs to the AV node that reduce ventricular speed during AF and improve cardiac symptoms without the need for pacemaker implantation (165,166). This technique has several limitations, including the inadvertent induction of a complete AV block and the relatively high risk of increasing ventricular speed during the first 6 months after abation. Thus, AV nodule modification without implantation of a pacemaker is rarely used in patients with rapid ventricular speed during AF.G. Prevention of thromboembolism1. The risk of Stratification. Epidemiological dataDeterrds of stroke in patients with AF are related to co-existing cardiovascular disease (4,19,73,74). In a small, retrospective, population-based study in Olmsted County, Minnesota, over 3 decades, a 15-year cumulative stroke in people with solitary AF (defined as younger than age 60 years with no clinical history or echocardiographic symptoms of cardiopulmonary disease) was 1.3% per year (4). Conversely, in the Framingham study, the age-adjusted stroke rate for an average follow-up period of 11 years was 28.2% in patients with solitary AF, which was more liberally defined to include patients with a history of hypertension or cardiomegaly for chest roentgenography; by comparison, for routine checks it was 6,8 %. In the SPAF study, the annualised rate of ischaemic stroke was similar in those with recurrent (3.2%) and permanent (3.3%) Af (167). Those with a previous stroke or transient ischemic attack have a subsequent stroke rate of 10% to 12% per year despite aspirin use and benefit substantially from treatment with a modified dose of oral anticoagulation (168,169). In addition to previous thromboembolism, independent risk factors for ischemic stroke in nonvalvular AF include HF, hypertension, increasing age, and diabetes mellitus (19,73,170-172). Female sex, blood pressure higher than 160 mmHg and NN dysfunction were associated with stroke (75,170,173). The relative risk of ischemic stroke associated with specific clinical features is shown in Table 8. Almost half of AF-related strokes occur in patients over 75 years of age, and AF is the most common cause of stroke in older women (9,171,173). Elderly people are at an increased risk of anticoagulant-related bleeding (174) and are therefore less likely to be treated with oral anticoagulation, even in situations that have been shown to be effective (175). Special attention to the elderly is therefore entitled to effective Table 86135. Risk factors for ischemic stroke and systemic embolism in patients with non-muscular atrial fibrillation (control group)Relative riskTIA indicates transient ischemic attack. Relative risk refers to comparisons with patients with atrial fibrillation without these risk factors. As a group, patients with non-muscular atrial fibrillation have approximately a 6-fold increased risk of thromboembolism compared to patients in normal sinus rhythm. Data from a joint analysis of five primary preventive studies (75). Previous stroke or TIA2.5Histories of hypertension1.6Polostive heart failure1.4Acrement (continuous, 1.4Diabetes mellitus1.7 Coronary artery disease1.5Patients with AF associated with thyrotoxicosis, which is often associated with congestive HF, are also at risk of stroke (176-178), although the underlying mechanism is unclear (52,179,180). AF is a common complication of HCM (181) and these patients have the incidence of stroke and systemic embolism of 2.4% to 7.1% per year (182-185).b. Role of echocardiography in risk stratificationTransthoracic Echocardiography: In a meta-analysis of 3 randomised antithrombotic therapy studies, moderate to severe LV dysfunction was the only independent echocardiographic predictor of stroke in AF patients when clinical traits were also considered (186). The LA mean was a less useful predictor of ischemic events (186). Transtocratic echocardiography TEE is the most sensitive and specific imaging technique for the detection of thrombus la and LAa (ALA) and far exceeds transtocratic echocardiography (52). This modality also allows excellent evaluation for other causes of cardiogenic embolism (187) and superiority when measuring LAA function (188). Several TEE features have been associated with thromboembomia, including such LA/LAA abnormalities as thrombus, reduced flow rate, and spontaneous echo contrast, as well as complex atheromatous plaques in the aortic (70,189). Detection of TROMB LA/LAA is a contraindication to the selective cardioversion of AF. The absence of a detectable thrombus does not exclude thromboembolism after cardioversion if patients do not receive anticoagulant therapy (190,191). Tee-led strategies for optional AF cardioversion have been reported to result in comparable results for thromboembolism and death compared to conventional anticoagulation for 3 weeks before and 4 weeks after cardioversion (192). Therefore, transtocratic echocardiography is valuable for defining the origin of AF (eg, for the detection of rheumatic mitral valve disease or HCM), and TEE may provide additional information for stratifying thromboembolic risk. Three clinical schemes have recently been proposed to stratify the risk of ischemic stroke in AF patients, based directly or indirectly on analyses of prospectively monitored cohorts of clinical trial participants in which antithrombotic treatment has been controlled One set of criteria is based on a multivariative pooled analysis of 1593 participants enrolled in control or placebo groups of 5 randomised primary preventive studies in which 106 ischemic strokes occurred during an average follow-up of 1.4 years (19). This scheme divides patients into 2 layers that distinguish low-risk patients from patients with moderate or high risk. Echocardiographic features were not initially considered, but a subsequent analysis of 3 of these studies identified abnormal systolic LV function as an independent predictor of stroke (186). The spaf study criteria were based on a multivariation analysis of 854 participants enrolled in clinical trials SPAF-I and -II who were followed for an average of 2.3 years during which 68 ischemic strokes were observed. The third set of criteria was developed on the basis of expert consensus (193) on the basis of consideration of the two above schemes and other available data for the classification of patients into low, medium and high risk groups (Table 9). Table 96135. Published stratification risk systems for primary* thromboembolism prevention in patients with non-muscular atrial fibrillationSourcehigh riskIntermediate RiskLow RiskBP indicates blood pressure. Patients are classified on the basis of the presence or absence of any risk factor. Modified with permission of Am J Med, Vol. 109, Pearce et al., Evaluation of these schemes for stratifying stroke risk in patients with non-muscular atrial fibrillation, p. 45-51, © 2000, with permission, from Excerpta Medica, Inc. (195).*Patients with AF and previous thromboembolism are at high risk of stroke, and anticoagulation is indicated for secondary prevention in such cases.†Did does not distinguish high risk from patients with moderate risk.‡Left ventricular dysfunction refers to moderately severe , severe abnormality of wall movement evaluated globally by 2-dimensional echocardiography, decreased ejection fraction, fractional shortening of less than 0.25 according to M-mode echocardiography, or clinical heart failure. Atrial Fibrillation Investigators (194)†Numerate greater than or equal to 65 yearsDíling less than 65 years No high-risk featuresHistory of hypertensionThis is not a high-risk featureCoronary artery diseaseDiabetesAmerican College of Chest Physicians (193)Age greater than 75 yearsPart 65-75 yearsYou want less than 65 yearsHistory of hypertensionDiabet /No risk factorsDiacal ventricular dysfunction‡Coronary artery diseaseMy more than 1 mean risk factorTyrotoxicikosisDetection prevention of atrial fibrillation (186)Women longer than 75 yearsHistory of hypertensionThe highest risk characteristicsSystolic BP greater than 160 mm HgNoho high-risk featuresZ hypertensionSequential dysfunction†c. Therapeutic consequencesRizo thromboembolism in patients with chronic attic flutter is generally estimated to be higher than in patients with sinus rhythm, but less than in patients with permanent or permanent AF. Biblo et al. (196) recently reviewed 8 years of retrospective data on 749 988 hospitalised 17,413 with atteam flutter and 337,428 with AF. The overall risk-of-stroke ratio in patients with attic flutter was 1.4 compared to the control group; patients with AF, the relative risk was 1.6. As chronic arrhythmia, atrial flutter is unusual, and the risk of thromboembolism is not as well established as it is for AF. Until more robust data are available, and although the overall thromboembolic risk associated with attic flutter may be lower than that of AF, it seems reasonable to estimate the risk using similar stratification criteria.2. Antithrombotic strategies for the prevention of ischemic stroke and systemic embolismFé large randomized studies published between 1989 and 1992 evaluated oral anticoagulation, and 2 tested aspirin for primary prevention of thromboembolism in patients with nevalvular AF (24,197-199). The sixth study focused on secondary prevention in patients who survived non-dissibate stroke or transient cerebral ischemic isatic acid (169). A meta-analysis based on the principle of treatment intent showed that modified oral anticoagulation is highly effective for preventing all strokes (ischemic and hemorrhagic), with a risk reduction of 61% (95% CI 47% to 71%) vs. placebo (200) (Fig. 6). This reduction was similar for both primary and secondary prevention. The follow-up time for these studies was usually between 1 and 2 years; the longest was 2.2 years, while in clinical practice the need for antithromatic treatment is usually prolonged for a much longer period. Figure 6. Antithrombotic treatment for the prevention of stroke (ischemic and hemorrhagic) in patients with non-muscular AF: adjusted-dose warfarin compared to placebo. Edited with permission from Hart et al. (170,200) Ann Intern Med 1999;131:492-501. (The American College of Physicians-American Society of Internal Medicine is not responsible for translation accuracy.) Patient age and anticoagulation intensity are the strongest predictors of severe bleeding (201-204). Study participants, aged 69 on average, were carefully selected and managed. It is therefore not clear whether the relatively low rate of severe bleeding also applies to patients with AF in clinical practice who have an average age of approximately 75 years and whose anticoagulant therapy is less regulated (205 206). The target intensity of anticoagulation includes the balance between prevention of ischemic stroke and avoidance of hemorrhagic complications. It is important to focus on the lowest adequate anticoagulation intensity to minimise the risk of bleeding, especially in elderly patients with AF. Maximum protection against ischemic stroke in AF is likely to be achieved with an international standardised ratio (INR) of 2 to 3 (168,207,208), while the INR range of 1.6 to 2.5 appears to be associated with incomplete efficacy, approximately 80% of what was achieved in higher intensity anticoagulation (Figure 7) (207 209). Figure 7. Adjusted course ratios ischemic stroke and intracranial haemorrhage in relation to the intensity of anticoagulation in randomised trials of antithromactic therapy in patients with AF. The data come from Hylek et al (203,207). In patients with AF who do not have mechanical valves, there is a consensus in the written group that anticoagulation can be discontinued for up to 1 week in procedures that carry a risk of bleeding without replacing heparin. In high-risk patients, or if a number of procedures require interruption for more than 1 week, non-fractional or low molecular weight heparin may be administered intravenously or subcutaneously. Heparins with low molecular weight have several pharmacological advantages over nefrakciated heparin. These include longer half-life, more predictable bioavailability (more than 90% after subcutaneous injection), predictable clearance (allowing once or twice a day subcutaneous administration) and a predictable body weight-based antithrombotic response that allows fixed-dose treatment without laboratory monitoring, except in special circumstances such as obesity, renal insufficiency or pregnancy (210). Low molecular weight heparins carry a lower risk of heparin-induced thrombocytopenia than unfractionated heparin (211). Self-use of low molecular weight heparins outside the hospital is a promising approach that can lead to cost savings in conjunction with optional cardioversion (212). Aspirin offers only modest protection against stroke in patients with AF (Fig. 8). The effect is less consistent than that of oral anticoagulation (200,213). Aspirin may be more effective for AF patients with hypertension or diabetes (213,214) and for reducing non-cardiacembolic versus cardioembolic ischemic stroke (49). Cardioembolic strokes are, on average, more preventable than non-cardiacembolic strokes (69). Figure 8. Antithrombotic treatment for the prevention of stroke (ischemic and hemorrhagic) in patients with non-muscular AF: warfarin compared to aspirin and aspirin compared to placebo. Edited with permission from Hart et al. (170,200) Ann Intern Med 1999;131:492-501. (The American College of Physicians-American Society of Internal Medicine is not responsible for translation accuracy.) 3. Conversion to Sinus Rhythm and ThromboembolismRandomized antithromactic therapy studies are absent in patients undergoing cardioversion of AF or atrial flutter, but the risk of thromboembolism was between 1% and 5% in the case-control series (102,215). There is no solid clinical evidence that cardioversion of AF followed by prolonged maintenance of the sinus rhythm effectively reduces thromboembolism. Patients in whom LAA thrombus is identified by TEE appear to be at high risk of thromboembobiism after cardioversion of AF or flutter and should be treated with anticoagulation for at least 3 to 4 weeks before and after pharmacological or electric cardioversion. In multicenter study (192) patients with AF lasting more than 2 days or flutter at the atm and previous AF have been randomized to TEE-guided or conventional strategies. One group was anticoagulant with heparin just before and with warfarin for 4 weeks after cardioversion. After identifying the thrombus, cardioversion was postponed and warfarin was administered for 3 weeks prior to tee recurrence. The second group received anticoagulant treatment for 3 weeks before and 4 weeks after cardioversion. Both approaches were associated with a comparably low risk of stroke (0.8% with tee-guided and 0.5% with conventional approach) after 8 weeks of follow-up. The risk of severe bleeding did not differ significantly. There were no differences in the proportion of subjects in whom the sinus rhythm was successfully restored. The clinical benefit of the TEE approach was therefore limited to saving time before cardioversion. Converting AF to sinus rhythm results in transient mechanical dysfunction of LA and LAA (190), known as stunning. This occurs after spontaneous, pharmacological (216,217), or electrical (217-219) conversion of AF and after radiofrequency catheter ablation of atrial trembling (220). Loss of atteam function may be associated with spontaneous echo contrast (190). Restoration of mechanical function can be delayed by several weeks, partly depending on the length of the AF before the restoration of the sinus rhythm (221-223). This might explain why some patients without a demonstrable LA thrombus on tee before cardioversion subsequently experience thromboembolic events (191). It is believed that the thrombus forms during the period of stun and is excreted after the return of mechanical function, which explains the clustering of thromboembolic events in the first 10 days after

cardioversion (224). Anticoagulation is recommended 3 to 4 weeks before and after cardioversion in patients with AF of unknown duration or in patients with AF of unknown duration or in patients who lasted more than 48 hours. Although LA thrombus and systemic embolism have been documented in patients with a shorter duration of AF, the need for anticoagulation in these patients is less clear. If acute AF creates hemodynamic instability, immediate cardioversion should not be delayed, but first intravenous heparin or low molecular weight heparin should be administered. Protection against late embolism may require the continuation of anticoagulation; The duration of anticoagulation after the procedure depends on the likelihood that the af will be repeated and on the internal risk of thromboembolism of the patient. IX. Proposed management strategiesA. An overview of algorithms for the treatment of patients with AFManagement of patients with AF requires knowledge of its presentation pattern (paroxysmal, persistent, or permanent) and decisions to restore and maintain sinus rhythm, control of ventricular speed and anticoagulation. These problems are addressed in different management algorithms for each AF.1 presentation. Newly discovered or first episode AF (Fig. 9)%It is not always clear whether the initial presentation of AF is actually the first episode of the patient, especially in patients with minimal or no symptoms of dysrrhythmia, so both are considered together. In patients who have self-limited episodes of paroxysmal AF, antiarrhythmic drugs to prevent recurrence are usually unnecessary if AF is associated with severe symptoms associated with hypotension, myocardial ischemia, or HF. Whether these individuals require long-term or even short-term anticoagulation is unclear, and the decision must be individual for each patient based on the internal risk of thromboembolism. Figure 9. Pharmacological treatment of patients with newly discovered AF. AF indicates atrial fibrillation; HF, heart failure. In patients with persistent AF, one option is to accept progression to permanent AF, with an emphasis on antithrombotic therapy and control of ventricular frequency. While it might seem reasonable to make at least 1 attempt to restore the sinus rhythm, it is not in the best interests of all patients. An example is an elderly man without risk factors for thromboembolism, in whom asymptomatic AF is discovered during routine examination, and control of ventricular velocity is easily achieved. Here, the potential toxicity of antiarrhythmic drugs may outweigh the benefits of restoring the sinus rhythm. If it is decided to try to restore and maintain the sinus rhythm, anticoagulation and speed control are important before cardioversion. Although long-term antiarrhythmic therapy may not be needed to prevent repeated AF after cardioversion, short-term treatment may be beneficial. In patients with AF longer than 3 months, early recurrence is early after cardioversion. Antiarrhythmic drugs can be started before cardioversion (after adequate anticoagulation) in such cases to reduce the likelihood of recurrence, and the duration of drug therapy would be short (e.g. 1 month).2. Recurrent paroxysmal AF (Fig. 10 and 11) Figure 10. Pharmacological treatment of patients with recurrent paroxysmal AF. * See Figure 11 for the proposed medicines. Figure 11. Antiarrhythmic drug therapy to maintain sinus rhythm in patients with recurrent paroxysmal or persistent atrial fibrillation. Drugs are listed alphabetically and not in order of proposed use. * For adrenergic atrial fibrillation, beta-blockers or sotalol are the initial medications of choice. †Cond non-pharmacological options to maintain the sinus rhythm if the drug fails. HF indicates heart failure; CAD, coronary artery disease; and LVH, left ventricular hypertrophy. In patients who experience short or minimally symptomatic recurrence of paroxysmal AF, it is reasonable to avoid antiarrhythmic drugs, but unpleasant symptoms generally require suppressive antiarrhythmic treatment. Speed control and thromboembolism prevention are appropriate in both situations. Several different antiarrhytmic drugs may be effective for each patient and selection is therefore based primarily on safety. For people with no or minimal structural heart disease, flecainid, propaphenone, and sotalol is recommended as an initial antiarrhythmic therapy because they are generally well tolerated and are essentially devoid of extracardiac organ toxicity. When one or the other of these drugs is ineffective or is associated with side effects, then the second or third line of choice include amiodo aaron, disopyramide, procainamide, and quinidine, which have greater potential for side effects. For some patients, a non-pharmacological approach is appropriate, which should be considered before starting treatment with amio aaron. On some occasions, a consistent initiation factor can be found. Disopyramide or flecainid can be used in cases of vagally mediated AF, while beta-blockers or sotalol is designed as an initial remedy for adrenergically induced AF. Many patients with organic heart disease can be widely divided into those with HF, CAD, or hypertension, although other types of heart disease may also be associated with AF. In patients with HF, safety data support the selection of amiodarone or dofetilide to maintain a sinus rhythm. Patients with coronary artery disease often require beta-blocker medications. Then sotalol, a drug with beta-blocking activity and primary antiarrhythmic effectiveness, is considered the first if the patient does not have HF. Amiodo aaron and dofetilide are considered secondary substances in this situation. The clinician may consider disopyramide, prokaaírid, or quinine on an individual basis. In patients with HYPERTENSION without LVH, drugs such as flecainid and propaphenone, which do not extend repolarization and QT interval, can offer a safety advantage and are recommended first. If these substances either prove ineffective or produce side effects, then amiodo aaron, dofetilide and sotalol are appropriate secondary options. Disopyramide, procainamide and quinine are considered third-line substances in this situation. Hypertrophied myocardium is prone to proarrhythmic toxicity and development of torsade de pointes type ventricular tachycardia. Amio aaron is proposed as the first line of treatment in patients with LVH (wall thickness greater than or equal to 1.4 cm) based on its relative safety compared to several other substances. Since neither ECG nor echocardiography always detects LVH, as defined by measuring the weight of the myocardium, doctors face a conundrum. The selection of antiarrhythmic drugs for patients with a history of hypertension is compounded by the lack of prospective controlled studies comparing the safety and efficacy of drug therapy for AF. The lack of data from randomized studies of antiarrhythmic drugs for the treatment of patients with AF generally applies to all patient groups. The drug selection algorithm presented here has therefore been developed as a consensus of experts and is particularly under review as further evidence emerges in this area 10).3. Recurrent persistent AF (Fig. 11 and 12)Patients with minimal symptoms who have undergone at least 1 attempt to restore sinus rhythm may remain in AF with treatment for speed control and thromboembolism prevention. Alternatively, those with symptoms prefer a sinus rhythm should be treated with an antiarrhythmic agent (in addition to drugs for speed control and anticoagulation) over cardioversion. The choice of antiarrhythmic drug should be based on the same algorithm used in patients with recurrent paroxysmal AF.4. Permanent AF (Fig. 12)%Permanent AF is a designation for cases where the sinus rhythm cannot be maintained after cardioversion of AF or when the patient and doctor have decided to allow AF to continue without further efforts to restore the sinus rhythm. It is important to maintain control over ventricular velocity and to use antithrombotic therapy, as noted elsewhere in this document, for all patients in this category. Figure 12. Pharmacological treatment of patients with recurrent persistent or permanent AF. *See Fig. Start drug therapy before cardioversion to reduce the likelihood of premature recurrence of AF. Table 106135. Recommendations for pharmacological cardioversion AF less than or equal to 7 days duration ** Drug ** Route of administrationDuction of recommendationsReferences*Drugs are listed alphabetically within each category of recommendations and the level of evidence.**Doses of drugs used in these studies may not be the same as those recommended by manufacturers. Substances with proven efficacyDofetilideOralIA133, 225-229FlecainideOral or intravenousIA8-90, 92, 230-235IbutilideIntravenousIA236-24Propafenoral or intravenousIA90, 93, 94, 230, 233, 235, 242-252AmiodaroneOral or intravenousIaA92, 96, 124, 234, 251, 253-260QuinidineIdinIIb88, 90, 91, 93, 242, 256, 257, 261, 262Ser active or incompletely studied substancesProcainamidIntravenousIIbC237, 238, 263DigoxinOral or intravenousIIA93 233, 244, 259, 264-267SotalolOral or intravenousSIIIA239, 261, 262, 266, 268Paušti 116135. Recommendations for pharmacological cardioversion AF More than 7 days Duration ** Drug * * Route of administrationType of recommendationsReferences * Drugs are listed alphabetically in each category of recommendations and the level of evidence.**The dosages of drugs used in these studies may not be the same as those recommended in Table 3 or manufacturers. Substances proven to be effectiveDofetilideOralIA133, 225-229AmiodaroneOral or intravenousIaA92, 96, 124, 234, 251, 253-260IbutilideIntravenousIaA236-241FlecainideOralIIbB88-90, 92, 230-235PropafenoneOral or intravenousIIbB90, 93, 94, 230, 233, 235, 242-252QuinidineOralIIb88, 90, 91, 93, 242, 256, 257, 261, 262Suniversive or incompletely studied substancesProcainamidIntravenousIIbC237, 238, 263SotalolOral or intravenousII A239, 261, 262, 266, 268DigoxinOral or intravenousIIIC93, 233, 244, 259, 264-267Table 126135. Recommended doses Drugs proven effective for pharmacological cardioversion of atrial fibrillationSsociating*Route of administrationDosage**Potential side effectsReferencesGI indicates gastrointestinal; IV, intravenous; bid, twice a day.*Medicines are sorted alphabetically.**The doses shown in the table may differ from those recommended by manufacturers.†In appropriate data are available on which specific recommendations for the use of one load regimen over another are based for patients with coronary artery disease or impaired left ventricle function, and these drugs should be used with caution or not at all in these patients.‡The use of quinine load to achieve pharmacological conversion of atrial fibrillation is a controversial and safer method available with the alternative substances listed in the table. Quinidine should be used with caution. AmiodaroneOralInpatient: 1.2-1.8 g per day in a divided dose up to 10 g in total, then 200-400 mg daily maintenance or 30 mg/kg as a single doseHypotenzion, bradycardia, qt extension, torsade de pointes (rare), GI agitation, constipation, flebitis (IV)92, 96, 124, 234, 251, 253-260Patient: 600-800 mg daily divided dose up to a total of 10 g, then 200-400 mg daily maintenance therapyIntravenous/oral5-7 mg/kg over 30-60 min then 1.2-1.8 g daily continuous IV or in divided oral doses up to 10 g in total, then 200-400 mg daily maintenanceDofetilideOralCreatinine clearance (mL/min) Dose (mcg BID)Extension QT, torsade de pointes; adjust the dose for kidney function, body size and age133, 225-229deaths than 60,50040-60 25020-40 125 than 20 contraindicatedflecainideOral200-300 mg†Hypotenzion, fast flutter 88-90, 92, 230-235Inttravenous1.5-3.0 mg per kg over 10-20 min†IbutilideIntravenous1 mg over 10 min; repeat 1 mg if necessary ExtensionQT, torsade de pointes236-241PropafenoneOral450-600 mgHypotenzion, fast atteam flutter90, 93, 94, 230, 233, 235, 242-252Intvenous1.5-2.0 mg per kg over 10-20 min†Kinkin‡Oral0.75-1.5 g in divided doses over 6-12 h, usually with speed-slowing drug extension, torsade de pointes, GI agitation, hypotension88, 90, 91, 93, 242, 256, 257, 261, 262Table 136135. Recommendations for the use of pharmacological agents to control the rate of ventricular responses to atrial fibrillationAssociating*Route of administrationAd of recommendationsA level of evidence*Doses of drugs used in these studies may not be the same as those recommended by manufacturers. DiltiazemIntravenousIAEsmololIntravenousIAVerapamilIntravenous or oralIAOthersIntravenous or oralIBDigoxinIntravenous or oralIIaBB. Recommendations for the treatment of patients with AF recommendations are evidence-based and are derived primarily from published data. The weight of evidence was assessed as highest (A) when data were derived from multiple randomised clinical trials and intermediate (B) where data were derived from a limited number of randomised studies, non-domed studies or observational A lower rank (C) was given when the primary basis for the recommendation was expert consensus. The recommendation shall follow the format of the previous ACC/AHA guidelines for the classification of indications, summarising both the evidence and the expert opinion:Class I: Conditions for which there is evidence and/or general agreement that the procedure or treatment is useful and effective. Class II: Conditions for which there is opposed evidence and/or differing views on the usefulness/effectiveness of the procedure or treatment. Class IIa: The weight of evidence or opinion is in favour of the procedure or treatment. Class IIb: Usefulness/efficacy is less demonstrated by evidence or opinion. Class III: Conditions for which there is evidence and/or general agreement that the procedure or treatment is not useful/effective and may in some cases be harmful. The reader is referred to the full-text guidelines available at ACC (www.acc.org), AHA (www.americanheart.org), ESC (www.escardio.org), and NASPE (www.naspe.org) World Wide Web sites and published in the European Heart Journal in mid-October 2001 for a full description of the justification and evidence supporting these recommendations. Recommendations for pharmacological and electrical cardioversion of AFClass IImmediate electrical cardioversion in patients with paroxysmal AF and rapid ventricular response who have ECG evidence of acute IM or symptomatic hypotension, angina or HF that do not respond immediately to pharmacological measures. (Evidence level: C) Cardioversion in patients without haemodynamic instability if symptoms of AF are unacceptable. (Evidence level: C) Class IIaPharmacological or electric cardioversion to accelerate sinus rhythm recovery in patients with the first detected episode of AF. (Evidence level: C) (see Tables 10 to 12 for recommended medicines.) Electrical cardioversion in patients with persistent AF if early recurrence is unlikely. (Evidence level: C) Repeated cardioversion followed by prophylactic pharmacothera in patients who return to AF without antiarrhythmic drugs after successful cardioversion. (Evidence level: C) Class IIbPharmacological substances for cardioversion to sinus rhythm in patients with persistent AF. (Evidence level: C) (see Tables 10 to 12 for recommended medicines.) In addition, hospitalation of pharmacological preparations for cardioversion of the first detected, paroxysmal or persistent AF in patients without heart disease or when verifying the safety of the drug in a particular patient. (Evidence level: C) (see Table 12.) Class IIIElectric cardioversion in patients who show spontaneous alternation between AF and sinus rhythm for a short period of time. (Evidence level: C) Additional cardioversion in patients with short periods of sinus rhythm who return to AF despite multiple cardioversion and prophylactic antiarrhythmic treatment. (Evidence level: C) Recommendations for pharmacological therapy Maintain sinus rhythm (see Table 3)Pharmacological strategies or algorithms for maintaining sinus rhythm in patients with AF (Fig. 9, 10, 11, 12) are based on available evidence and extrapolated from experience with these substances in other situations. Class IBase selection of pharmacological therapy to maintain sinus rhythm in patients with preventable or otherwise unpleasant symptoms during AF predominantly for safety. (Evidence level: B) Treat accelerating or reversible causes of AF before starting antiarrhythmic drug therapy. (Evidence level: C) Class IIaAdminister pharmacological treatment to maintain sinus rhythm to prevent progression of tachycardia-induced cardiomyopathy due to AF. (Evidence level: C) Rarely and well tolerated recurrence of AF can in some cases be considered a successful result of antiarrhythmic drug therapy. (Evidence level: C) Outpatient initiation of antiarrhythmic therapy is appropriate in selected patients. (Evidence level: C) Class IIbAdminister pharmacological treatment to maintain sinus rhythm in asymptomatic patients to prevent attic remodeling. (Evidence level: C) Administer pharmacological treatment to maintain sinus rhythm to prevent thromboembolism or HF in selected patients. (Evidence level: C) Administer combinations of antiarrhythmic agents to maintain a sinus rhythm when single-drug therapy fails. (Level of evidence: C) Class IIIUse of a specific pharmacological agent to maintain a sinus rhythm in patients with well-defined proarrhythmia risk factors for this product. (Evidence level: A) Use of pharmacological therapy to maintain sinus rhythm in patients with advanced sinus node or dysfunction of AV nodes in the absence of a functional electronic pacemaker. (Evidence level: C) Recommendations for heart rate control in patients with AF (see Tables 6, 7 and 13) IMeasure class heart rate response both at rest and during exercise in patients with persistent or persistent AF and control speed with pharmacological agents (using beta-blocker or calcium channel antagonist in most cases) to physiological range. (Evidence level: C) Administer intravenous beta-blockers or calcium channel antagonists (verapamil, diltiazem) in an acute environment to slow the ventricular response to AF in the absence of guidance through the accessory pathway, be careful in patients with hypotension or HF. (Evidence level: B) Perform immediate electrical cardioversion in patients with acute paroxysmal AF and rapid ventricular response associated with acute mycardial infarction , symptomatic hypotension, angina or heart failure that does not respond immediately to pharmacological measures. (Evidence level: C) Class IIaAdminister combination of digoxin and beta-blocker or calcium channel antagonist to control heart rate at rest and during exercise in patients with AF. The choice of the drug should be individualized and the dose modulated to prevent bradycardia. (Level of evidence: non-pharmacological treatment to control heart rate if pharmacological treatment is inadequate. (Evidence level: C) Class IIbAdminister digoxin as the only means to control heart rate at rest in patients with persistent AF. (Evidence level: B) Administer intravenous quinine, prokainamide, disopyramide, ibutilide or amiodaron haemodynamically stable patients with AF involving guidance through the accessory pathway. (Evidence level: B) Immediate cardioversion is required if patients with AF involving guidance through the accessory pathway experience very rapid tachycardia or haemodynamic instability. (Evidence level: B) Class IIIADminister digitalis as the only means to control the rapid response of chambers to AF in patients with paroxysmal AF. (Evidence level: B) Catheter ablation without prior medical therapy to control AF. (Evidence level: C) Recommendations for antithromactic treatment in patients with AF (see Table 146135. Recommendations for antithromactic therapy in patients with thromboembolic fibrillation based on thromboembolic risk StratificationPacient functionAntithrombotic therapyAntithrombotic therapyAntithrombotic recommendations*Risk factors for thromboembolism include HF, 73 ejection fraction smaller than 0.35 and history of hypertension.AF suggests atrial fibrillation; CAD, coronary artery disease; HF, heart failure; INR, international standardised ratio; LV, left ventricle; and TEE, transesophageal echo. Age less than 60 years No heart disease (lonely AF)Aspirin (325 mg per day) or no therapyDeasing less than 60 years Heart disease, but no risk factors*Aspirin (325 mg daily)IAge greater than or equal to 60 years No risk factors*Aspirin (325 mg daily)IAge greater or equal to 60 years With diabetes mellitus or CADOral anticoagulation (INR 2.0-3.0)IAddition aspirin , 81-162 mg per day is optionalIIbAge greater than or equal to 75 years, in particular womenOral anticoagulation (INR ≈2.0)IHF LV ejection fraction smaller than or equal to 0.35 Thyrotoxicosis HypertensionOral anticoagulation (INR 2.0-3.0)IRheumatic heart disease (mitral stenosis) Prosthetic heart valves Previous thromboembolism Persuasion atrial thrombus on TEEOral anticoagulation (INR 2.5-3.5 or higher may be appropriate)IClass IAdminister antithrombotic therapy (oral anticoagulation or aspirin) for all patients with AF , except those that have solitary AF to prevent thromboembolism. (Level of evidence: A) Individualize the selection of an antithrombottic agent based on an assessment of the absolute risks of stroke and bleeding and the relative risk and benefit to a particular patient. (Evidence level: A) Chronic oral anticoagulant therapy at a dose adjusted to reach the target intensity of INR 2 to 3 in patients at high risk of stroke, unless contraindicated. (Evidence level: A)a. The need for anticoagulation should be reassessed at regular intervals. (Level of evidence: A)b. INR should be established at least once a week during the initiation of oral anticoagulant therapy and monthly if the patient Stable. (Evidence level: A) Aspirin at 325 mg per day as an alternative in patients at low risk or in patients with certain contraindications to oral anticoagulation. (Evidence level: A) Oral anticoagulation in patients with AF who have rheumatic mitral valve disease or prosthetic heart valves (mechanical or tissue valves). (Evidence level: B) Base the target intensity of anticoagulation on a specific type of prosthesis, but should not be less than INR 2 to 3. (Evidence level: B) Class IIaTarget lower INR 2 (range 1.6 to 2.5) for primary prevention of ischemic stroke and systemic embolism in patients over 75 years of age who are expected to be at increased risk of bleeding complications but without sincere contraindications to oral anticoagulant therapy. (Evidence level: C) Administration of antithrombotic therapy in patients with attic flutter, in general, as well as in patients with AF. (Evidence level: C) Select antithrombotic therapy according to the same criteria regardless of AF pattern (i.e. in patients with paroxysmal, persistent, or permanent AF). (Evidence level: B) Class IIbInterrupt anticoagulation for up to 1 week for surgical or diagnostic procedures that carry a risk of bleeding without substitutating heparin in patients with AF who do not have mechanical prosthetic heart valves. (Evidence level: C) Administer intravenously or subcutaneously non-fractional heparin or low molecular weight heparin or in selected high-risk patients, or if a number of procedures require discontinuation of oral anticoagulant therapy for more than 1 week. (Evidence level: C) Manage patients with anticoagulant CAD (INR 2 to 3) based on the same criteria used in non-CAD patients. (Evidence level: C) and. A low dose of aspirin (less than 100 mg per day) or clopidogrel (75 mg per day) may be administered in parallel with anticoagulations, but these strategies have not been adequately evaluated and may be associated with an increased risk of bleeding. (Evidence level: C) Aspirin treatment is optional for primary stroke prevention in patients under 60 years of age without heart disease or risk factors for thromboembolism (solitary AF). (Evidence level: C) Class III Long-term anticoagulation for the prevention of stroke in patients under 60 years of age without heart disease (solitary AF) and without risk factors for thromboembolism. (Evidence level: C) Recommendations for antithromactic therapy to prevent ischemic stroke and systemic embolism in patients with AF undergoing cardioversionClass IAdminister anticoagulant therapy regardless of the method (electrical or pharmacological) used to restore sinus rhythm. (Evidence level: B) Anticoaagulation with AF lasting more than 48 h or unknown duration for at least 3 to 4 weeks before and after cardioversion (INR 2 to 3). (Evidence level: B) Perform immediate cardioversion in patients with acute (recent onset) AF accompanied by symptoms or signs of haemodynamic instability angina, myocardial infarction, shock or pulmonary edema without waiting for previous anticoagulation. (Evidence level: C) and. If not contraindicated, administer heparin simultaneously with an initial intravenous bolus injection followed by a continuous infusion at a dose adjusted to extend the time of activated partial thromboplastin at 1,5 to 2 times the reference control value.b. In addition, provide oral anticoagulation (INR 2 to 3) for at least 3 to 4 weeks for patients undergoing selective cardioversion. (Evidence level: C)c. Limited data from recent studies support subcutaneous administration of low molecular weight heparin in this indication. (Evidence level: C) Screening for thrombus in LA or LAA tee is an alternative to routine preanticoagulation in AF cardioversion candidates. (Evidence level: B) and. Patients with anticoagolate in whom thrombus is not identified by intravenous non-fractional heparin by initial bolus injection prior to cardioversion, followed by continuous infusion at a dose adjusted to extend the activated partial thromboplastin to 1,5 to 2 times the reference control value. (Level of evidence: B)b. In addition, provide oral anticoagulation (INR 2 to 3) for at least 3 to 4 weeks for patients undergoing selective cardioversion. (Level of evidence: B)c. Limited data are available to support subcutaneous administration of low molecular weight heparin in this indication. (Evidence level: C)d. Treat patients in whom thrombus is identified by TEE with oral anticoagulation (INR 2 to 3) for at least 3 to 4 weeks before and after the restoration of sinus rhythm. (Evidence level: B) Class IIbCardioverze without TEE guidance within the first 48 hours after onset of AF. (Evidence level: C) a. In these cases, anticoagulation before and after cardioversion is optional, depending on the risk assessment. (Evidence level: C) Anticoagulant patients with at-at-at-risk flutter undergoing cardioversion in the same way as patients with AF. (Evidence level: C) Recommendations for the prevention and treatment of postoperative patients with AFClass ITreat undergoing cardiac surgery with oral beta-blocker to prevent postoperative AF if not contraindicated. (Evidence level: A) In patients who develop postoperative AF, achieve speed control by administration and in nodule blockers. (Evidence level: B) Class IIaAdminister sotalol or amio aaron prophylactically to patients at increased risk of developing postoperative AF. (Evidence level: B) Restore sinus rhythm in patients who develop postoperative AF pharmacological cardioversion with ibutiide or direct current cardioversion as recommended in non-surgical patients. (Evidence level: B) In patients with recurrent or refractor postoperative AF, attempt to maintain sinus rhythm by administering antiarrhythmic drugs as recommended in CAD patients who develop AF. (Evidence level: B) Manage patients who develop postoperative AF as recommended in non-surgical patients. (Evidence level: B) Recommendations for the treatment of patients with AF and acute myocardial infarctionClass IElectric cardioversion in patients with severe haemodynamic compromise or unmanageable ischemia. (Evidence level: C) Intravenous administration of digitalis or amiodarone to slow down rapid ventricular response and improve LV function. (Evidence level: C) Intravenous beta-blockers to slow rapid ventricular response in patients without clinical dysfunction of NN, bronchospastic disease or AV block. (Evidence level: C) Heparin in patients with AF and acute MI, unless contraindications to anticoagulation are present. (Evidence level: C) Class IIIAdminister type IC antiarrhythmic drugs in patients with AF in the setting of acute myocardial infarction. (Evidence level: C) Recommendations for the treatment of AF and ventricular preexcitationClass ICateral ablation of accessory pathways in symptomatic patients with AF who have WPW syndrome, especially those with syncope due to rapid heart rate or patients with short refractory bypass surgery. (Evidence level: B) Immediate electrical cardioversion to prevent ventricular fibrillation in WPW patients experiencing AF with rapid ventricular response associated with haemodynamic instability. (Evidence level: B) Intravenous prokaaíd or ibutilide in an effort to restore the sinus rhythm in patients with WPW who experience AF without haemodynamic instability associated with a broad QRS-based ECG complex (longer than or equal to 120 ms). (Evidence level: C) Class IIbAdminister intravenous quinine, prokaainamide, disopyramide, ibutilide or amiodaron in haemodynamically stable patients with AF involving guidance through the accessory pathway. (Evidence level: B) and. Immediate cardioversion is necessary if patients with AF involving guidance through the accessory pathway experience very rapid tachycardia or haemodynamic instability. (Evidence level: B) Class IIIInside administration of beta-blockers, digitalis glycosides, diltiazem or verapamine in patients with WPW syndrome who have preexcited ventricular activation in AF. (Evidence level: B) Recommendations for the treatment of AF in patients with hyperthyroidismClass IAdminister beta-blocker as necessary to control the rate of ventricular response in patients with AF complicating thyrosoxicosis, if not contraindicated. (Evidence level: B) In cases where the beta-blocker cannot be used, use a calcium channel antagonist (diltiazem or verapamil) to check the ventricular frequency. (Evidence level: B) In patients with AF associated with thyrotoxicosis, use oral anticoagulation (INR 2 to 3) to prevent thromboembolism, as recommended in patients with AF with other stroke risk factors. (Evidence level: C) and. After the restoration of the state of euthyreoid, the recommendations for antithrombotic prophylaxis as in patients without hyperthyroidism. (Evidence level: C) Recommendations for managing AF during pregnancyClass IControl ventricular response rate with digoxin, beta-blocker, or calcium channel antagonist. (Evidence level: C) Electrical cardioversion in patients who become haemodynamically unstable due to dysrhythmia. (Evidence level: C) Administer antithromboline therapy (anticoagulant or aspirin) during pregnancy to all AF patients (except those with solitary AF). (Evidence level: C) Pharmacological cardioversion of Class IIbAttempt by administration of quinidine, prokaaamide or sotalol in haemodynamically stable patients who develop AF during pregnancy. (Evidence level: C) Administer heparin to patients with thromboembolism risk factors during the first trimester and last month of pregnancy. Unfractionated heparin can be administered either by continuous intravenous infusion at a dose sufficient to extend the activated partial thromboplastin to 1.5 to 2 times the control (reference) value, or intermittent subcutaneous injection at a dose of 10,000 to 20,000 U every 12 hours, set to extend the mean interval (6 hours after injection) of activated partial thromboplastin to 1.5 times the control. (Level of evidence: B) a. Limited data are available to support subcutaneous administration of low molecular weight heparin for this indication. (Evidence level: C) Administer oral anticoagulant during the second trimester to patients at high thromboembolic risk. (Evidence level: C) Recommendations for the treatment of AF in patients with HCMClass ITreat patients with HCM who develop oral anticoagulant AF (INR 2 to 3), as recommended in other high-risk patients for the prevention of thromboembolism. (Evidence level: B) Class IIaAntiarrhythmic medications to prevent recurrence. Available data are insufficient to recommend one agent over another in this situation, but disopyramide and amiodo aaron are generally preferred. (Level of evidence: C) Recommendations for the treatment of AF in patients with lung diseasesClass IV patients who develop AF during acute lung disease or exacerbation of chronic lung disease, hypoxemic correction and acidosis are primary therapeutic measures. (Evidence level: C) In patients with obstructive pulmonary disease who develop AF, a calcium channel antagonist (diltiazem or verapamil) is preferred to control the ventricular frequency. (Evidence level: C) Attempted electrical cardioversion in patients with lung disease who become haemodynamically unstable due to AF. (Evidence level: C) Class IIIUse of theophyline and beta-adrenergic agonistic agents in patients with bronchospastic lung disease developing AF. (Evidence level: C) Use of beta-blockers, sotalol, propaphenone, and adenosine in patients with obstructive pulmonary disease who develop AF. (Evidence level: C) This document has been approved by the American College of Cardiology Board of In August 2001, the Scientific Advisory and Coordination Committee of the American Heart Association in August 2001 and the European Cardiology Council and the Committee on Practical Practice in August 2001.Quoting this document by the American College of Cardiology, The American Heart Association and the European Society of Cardiology would appreciate the following citation format: Fuster V, Rydén LE, Asinger RW, Cannom DS, Crijns HJ, Frye RL, Halperin JL, Kay GN, Klein WW, Lévy S, McNamara RL, Prystowsky EN, Wann LS, Wyse DG. ACC/AHA/ESC guidelines for the treatment of patients with atrial fibrillation: summary: report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines and the European Cardiology Commission for Practical Guidelines and Policy Conferences (Committee for the Development of Guidelines for the Management of Patients with Atrial Fibrillation). Circulation. 2001;104:2118-2150.This statement was co-published in the October 2001 issue of the Journal of the American College of Cardiology.The American Heart Association makes every effort to avoid any actual or potential conflicts of interest that may arise as a result of an external relationship or the personal, professional or commercial interest of a panel member. Specifically, all members of the writing group are required to complete and submit a disclosure questionnaire showing any such relationships that could be perceived as actual or potential conflicts of interest. This document is available on the websites of the American College of Cardiology (www.acc.org), the American Heart Association (www.americanheart.org), the European Society of Cardiology (www.escardio.org) and the North American Society of Pacing and Electrophysiology (www.naspe.org). Individual reprints of this document (Summary and Summary of Recommendations) are available at 800-253-4636 (U.S. only) or written by the American College of Cardiology, Educational Services, 9111 Old Georgetown Road, Bethesda, MD 20814-1699. To get a reprint of the full guidelines published in the mid-October issue of the European Heart Journal, call +44 207 424 4200 or +44 207 424 4389, fax +44 207 424 4433, or write to Harcourt Publishers Ltd, European Heart Journal, ESC Guidelines - Reprints, 32 Jamestown Road, London, NW1 7BY, United Kingdom. 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