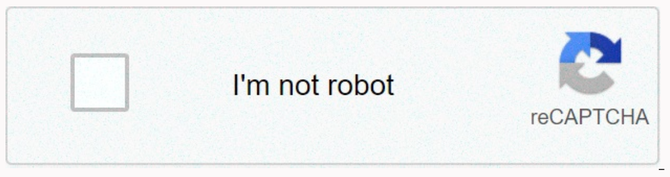

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Guideline af esc

Atrial fibrillation (AF), the most common sustained heart rhythm disorder, increases in prevalence as the population ages. Although often associated with heart disease, AF occurs in many patients with no perceived disease. Hemodynamic impairment and thromboembolic events lead to significant morbidity, deaths and costs. Consequently, the American College of Cardiology (ACC), the American Heart Association (AHA), and the European Society of Cardiology (ESC) have created a committee of experts to establish guidelines for managing this ahythmia. The committee was compiled out of 8 members representing the ACC and AHA, 4 representing the ESC, 1 of the North American Association of Pacing and Electrophysiology (NASPE), and a representative of the Johns Hopkins University Evidence-Based Practice Center representing the Agency for Health Care Research and Quality's report 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 by the ACC Clinical Electrophysiology Committee, the AHA ECG and Athrhythmia Committee, NASPE, and 25 reviewers nominated by the writing committee. The document was approved for publication by the governing bodies of the ACC, AHA and ESC and officially endorsed by NASPE. These guidelines will be reviewed annually by the task force and will be considered current unless the task force reviews or withdraws it from distribution. The committee conducted a comprehensive review of the literature from 1980 to June 2000 that is relevant to AF using the following databases: PubMed/Medline, EMBASE, the Cochrane Library (including the Cochrane Database of Systematic Reviews and the Cochrane-Controlled Trial Law), and Best Evidence. Searches are limited to English language sources and to human subjects.II. DefinitionsA. Atrial FibrillationAF is a supraventricular tachyarrhythmia characterized by uncoordinated atrial activation with consequent deterioration of atrial mechanical function. On the electrocardiogram (ECG) AF is described by the replacement of consistent P-waves through rapid oscillations or fibrillatory waves ranging in size, shape and timing, associated with an irregular, often rapid ventricular reaction when atrioventricular (AV) conduction is intact (1). The ventricular response to AF depends on electrophysiological characteristics of the AV node, the level of vagal and sympathetic tone, and the action of drugs (2). Regular RR intervals are possible in the presence of AV block or interference by ventricular or connector specardy. A quick, irregular, sustained, wide-QRS complex tachycardia strongly suggests AF with conduction across an accessory pad or AF with underlying batch branch block. Extremely fast rates (over 200 bpm) suggest the presence of an accessory .B. Related can be isolated or associated with other athrhythmias, often atrial fluttering or atrial ticardia. ticardia. flutter may arise during antiarritmic drugs treatment prescribed to prevent repetitive AF. Atrial flutter is more organized than AF, with a saw-toothed pattern of frequent atrial activation called flutter (f) waves on the ECG, especially visible in lei II, III, and aVF. Untreated, the atrial rate typically 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 wave of activation in the right atrium (RA) can be reversed, leading to f waves leading upright into II, III, and aVF and flipped into lead V1. Two-on-one AV block is common, producing a ventricular rate of 120 to 160 bpm. Atrial fluttering can degenerate into AF, AF can initiate atrial fluttering, or the ECG pattern can alternate between atrial flutter and AF, reflecting the change of atrial activation. Other atrial tachycardias, as well as AV reentrant tachycardias and AV nodal reentrant staircardia, can also trigger AF. In other atrial tachycardias, P waves are readily identified and are separated by an isotric baseline in 1 or more ECG leads. The morphology of the P waves can help localize the origin of atrial tachycardias. A unique type of atrial tachycardia originates in the pulmonary arteries (3), is typically faster than 250 bpm, and often degenerates into AF. Intracardiac mapping can help distinguish the different atrial arhythmias. III. ClassificationAF has a heterogeneous clinical presentation that occurs in the presence or absence of observable heart disease or related symptoms. For example, the term lone AF is several defined. The prognosis in terms of thrombohybolicism and mortality is most precarious when applied to young individuals (less than 60 years old) without clinical or echocardifiable evidence of cardiopulmonary disease (4). These patients have a favorable prognosis regarding thrombow emboss and deaths. By heads of aging or developing heart abnormalities, however, patients move out of the lone AF category over time, and the risks of thrombowbolicism and deaths are rising. Lone AF is distinguished from idiopathic AF, which implies uncertainty about its origin without reference to the age of the patient or associated cardiovascular pathology. By convention, the term non-valvular AF is limited to cases in which the rhythm disorder occurs in the absence of rheumatic antial stalosis or a prosthetic heart valve. The classification scheme recommended in this document represents a consensus driven by a desire for simplicity and clinical relevance. The clinician must distinguish a first-detected episode of AF, whether it's symptomatic or self-restrained, and acknowledge that there could be uncertainty over the duration of the episode and over previous undetected episodes (Fig. 1). When a patient has had 2 or more episodes, AF is considered repetitive. Once terminated, AF paroxysmally designated, and when persistent, persistent. In the latter case, case, through pharmacological therapy or electric cardiovassion does not align the designation. Persistent AF can be either the first presentation or an excretion of recurring episodes of paroxysmal AF. Persistent AF includes cases of prolonged AF (e.g., greater than 1 year), in which cardiovassion has not been indicated or tried, which usually leads to permanent AF (Fig. 1). The terminology defined in the preceding paragraph applies to episodes of AF lasting more than 30 seconds and which are not related to a reversible cause. AF secondary to a precipitating condition such as acute myocardial infarction, heart surgery, myocarditis, hyperthyroidism or acute pulmonary disease are considered separately. In these settings, treating the underlying disorder simultaneously with the management of the episode of AF usually eliminates the aritmia. Figure 1. Patterns of atrial fibrillation. 1, episodes that generally last less than or equal to 7 days (most less than 24 h); 2, usually more than 7 days; 3, cardioversion fails or does not attempt; and 4, whether paroxysmal or persistent AF can be repetitive.IV. Epidemiology and PrognosisAF are the most common clinically significant cardiac arrhythmias. In one series, AF accounts for 34.5% of patients admitted to hospital with a heart rhythm disorder (5). It has been estimated that 2.2 million Americans have paroxysmal or persistent AF (6).A. AppearanceThe incidence of AF is estimated at 0.4% of the general population, increasing by age (7). AF is unusual in childhood except after heart surgery. It occurs in less than 1% of those under 60 years, but in more than 6% of those over 80 years (8-10) (Fig. 2). The age-adjusted appearance is higher in men (10.11). Blacks have less than half the age-adjusted risk of developing AF seen in whites (12). The frequency of solitary AF was less than 12% of all cases of AF in some series (4,10,13,14) but more than 30% in others (15,16). The incidence of AF increases with the severity of congestive heart failure (HF) or valve heart disease. Figure 2. Prevalence of AF in 2 U.S. epidemiological studies. Framingham marks the Framingham Heart Study (9); CHS, Cardiovascular Health Study (10).B. PrognosisThe rate of iscemic stroke among patients with nonreumatic AF averages 5% per year, which is 2 to 7 times the rate for people without AF (8,9,15,17–19) (Fig. 3) . One of every 6 strokes occurs in patients with AF (20). Including transient iscemic seizures and clinically silent strokes detected radiographically, the rate of brain iscemia associated with non-valvular AF is more than 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 attributable risk was 5 times greater than in those with kidney heumatic AF (9). AF patients from general practices in France, the ALFA Study (Etude and Activité sur le Fibrillation Auriculaire) Auriculaire) A 2.4% incidence of thrombow emboss over an average of 8.6 months of follow-up (15). The annual risk of stroke attributable to AF has increased from 1.5% in Framingham Study participants aged 50 to 59 years to 23.5% for those aged 80 to 89 years (9). The total mortality rate is roughly doubled in patients with AF compared to patients in normal sinus rhythm and has been linked to the severity of underlying heart disease (8,11,18) (Fig. 3). Figure 3. Relative risk of stroke and mortality in patients with AF compared to patients without AF. Source data is from the Framingham Heart Study (11), Regional Heart Study (8), Whitehall Study (8), and Manitoba Study (18).V. Pathophysiological MechanismsA. Atrial Factors1. Pathology of the Atrium in Patients With AFThe atria of patients with persistent AF displays structural abnormalities beyond those caused by underlying heart disease (27). Patch fibrosis with juxtaposition of normal and sick atrial livestock may be responsible for non-comogeneity of atrial birectory (28,29). Fibrosis or fatty infiltration can also affect the sinus node and can be a response to inflammatory or degenerative processes that are difficult to detect. The role of inflammation in the pathogenesis of AF has not yet been evaluated, but histological changes consistent with myocarditis have been reported in 66% of biopsy samples from patients with solitary AF (29). Infiltration of the atrial myocardium can occur in amyloidosis, sarcoidosis, and hemochromatosis. Atrial fiber hypertrophy has been described as a large and sometimes the only histological feature (28). Progressive atrial dilatation is echocardiographically demonstrated in patients with AF (30) and, like hypertrophy, can be either a cause or a result of persistent AF.2. Mechanisms of AFTheories of the mechanism of AF involve 2 main processes: improved automaticity in 1 or several rapidly depolarising foci and re-entry involving 1 or more circuits (31,32). Fast-firing atrial foci, located in 1 or several of the better pulmonary arteries, can initiate AF in susceptible patients (3,33). Foci also occurs in the RA and infreunded in the better vena cava or coronary sinus (3,33,34). The focal origins seem to be more important in paroxysmal AF than in persistent AF. Ablation of such foci can be healing (3). The multiple wavelet hypothesis as the mechanism of reentrant AF was advanced by Moe and colleagues (31,35), who suggested fraction of the gulf fronts as they propagate through the atria leading to self-perceptuating daughter waves. The number of wavelets present at any time depends on the refractory period, mass, and conductivity in different parts of the atria. Although the patterns of activation under the irregular atrial electrical activity of AF have traditionally been described as disorganized or randomized, recent evidence has emerged that AF spatially Is. Based on mapping studies of patients undergoing surgery for the (WPW) syndrome, 3 patterns of induced AF have been identified (36). Type I AF involves single wave fronts that propagate across the RA. Type II AF involves 1 or 2 wave fronts, and type III AF is characterized by various activation wavelets that propagate in different directions. Ultimately, a better understanding of electrophysiological mechanisms will lead to the development of effective preventive measures (37).B. AV Behavior1. General AspectsThe AV node is usually the factor that limits conduction during AF. The compact AV node is anteriorly located in the triangle of Koch (38), surrounded by transition cells. There appear to be 2 separate atrial inputs to the AV node, posteriorly via the crista terminalist and anteriorly via the interattrial septum. Studies on rabbit AV nodal preparations show that during AF, reproduction of impulses by the AV node to the Side bundle partly depends on the relative timing of the anterior and posterior septal activation inputs to the AV node (39). Other factors affecting conductivity by the AV node are its intrinsic conductivity and refractoriness, hidden conductivity and autonomous tone.2. AV Conductivity in the WPW SyndromeAccessory pathways are muscle connections between the atrium and ventricle that have the ability to perform

quickly. Conduction across an accessory pathway during AF can lead to a very rapid ventricular response that can be deadly (2,40). Drugs like digitalis, calcium channel antagonists, and beta-blockers, which are usually given to slow conduction across the AV node during AF, do not block conduction about the accessory pathway and can even improve conductivity, leading to hypotension or cardiac steepness (41). Patients who developed AF with rapid ventricular response associated with hemodynamic instability that have persisted from conduction across an accessory pathway must undergo immediate electric cardiovassion. In the absence of hemodynamic instability or a leading ventricular reaction, intravenous procaine and ibutilide are drugs of choice to achieve pharmacological cardiovasions or to block conduction across the accessory pathway.C. Myocardial and Hemodynamic Consequences of AFDuring AF, 3 factors may affect hemodynamic function: loss of synchronous atrial mechanical activity, irregularity of ventricular reaction, and inappropriate heart rate A noticeable decrease in heart output may occur with the loss of atrial contraction , especially in patients with impaired diastolic ventricular filling, hypertension, mittral stenosis, hypertrophic cardiomyopathy (HCM), or restrictive cardiomyopathy. The variation in RR intervals during AF can also lead to hemodynamic impairment. A persistently fast atrial rate can adversely affect atrial mechanical function (10-induced atrial cardiomyopathy) (2,42). Such changes in atrial tissue may explain the delayed recovery of atrial contractibility in patients after cardiovassion to sinus rhythm. A increased increased rate during AF (130 bpm or faster in one study) (43) can produce dilated ventricular cardiomyopathy (2,43–46). It is critical to recognize 1ecard-induced cardiomyopathy, because control over the ventricular rate can lead to partial or even complete reversal of the myopathic process. In fact, HF may be the initial manifestation of AF. A variety of hypotheses have been proposed to explain tachycardia-mediated cardiomyopathy that involves myocardial energy depletion, Ischaemia, abnormalities of calcium regulation and remodelling, but the real mechanisms responsible for this disorder are still unclear (47).D. Thrombow embolismAlthough ischaemic stroke and systemic arterial disconnection in AF are generally attributed to embolism from the left atrium (LA), the pathogenesis of thrombo Up to 25% of AF-associated strokes may be due to intrinsic cerebrovascular diseases, other heart sources of embolism, or atheromatous pathology in the proxy aorta (49,50). About half of elderly AF patients have chronic hypertension (a major risk factor for cerebrovascular disease) (19), and about 12% nurture keronic carotidity stenosis. Karotid atherosclerosis is not significantly more common in AF patients with stroke than in patients without AF, however, and is likely a relatively minor contributing factor (51).1. Pathophysiology of Trombus formationThrombus associated with AF originates mostly in the LA Schedule (LAA). It cannot be reliably examined by precordial (transthoracic) echocardiography (52), while transesophageal Doppler echocardiography provides a sensitive and specific method of evaluating LAA function (53) and detecting thrombotic materials. LAA flow speeds decrease due to the loss of organized mechanical contraction during AF (54,55). This substrate of reduced flow within the LA/LAA is associated with spontaneous echo contrast, thrombobe formation, and embolic events (56-62). LAA flow velocities are lower in patients with atrial flutter than is usually seen with normal sinus rhythm, but are higher than with AF. Whether it accounts for the slightly lower incidence of LAA thrombus and perhaps a lower rate of thromboembolicism associated with atrial flute is uncertain. In patients with AF, independent predictors of spontaneous echo contrast include LA size, LAA flow velocity (56,63), left ventricular (LV) dysfunction, fibrinogen level (62), hematocrit (61,62), and aortic atherosclerosis (61,62,65). This phenomenon may be an echocardifiable surrogate for local coagulopathy and, when dense, is of clinical value in identifying AF patients at high risk for thrombow bulbism (64), but its usefulness to prospective risk stratification for thrombohydrasism beyond what has been achieved by clinical assessment has not been established. Although conventional clinical management is based the presumption that trumpet formation requires the continuation of AF for about 48 h, thrombi thrombi identified by transesophageal echocardiography (TEA) at shorter intervals (66,67). Contrary to the most common view that systemic anticoagulation for 4 weeks leads to endocardial compliance and organization of LAA thrombus, TEE studies have verified resolution of thrombe in the majority of patients (68). Similar observations defined the transient nature of LA/LAA dysfunction on converting AF, which provides a mechanistic rationale for anticoagulation for several weeks before and after successful cardiovassion.2. Clinical ImplicationsBecause the pathophysiology of thrombohymanism in patients with AF is uncertain, the mechanisms linking risk factors to ischaemic stroke in AF are also incompletely defined. The strong association between hypertension and stroke in AF is likely primarily mediated by embolism that originates in the LAA (49), but hypertension also increases the risk of noncardymbolic strokes in AF (49,69). Hypertension in AF patients is associated with reduced LAA fluid velocity and spontaneous echo contrast, making the patient susceptible to thrombosis formation (63,64,70). Ventricular diastalic dysfunction may underlying the effect of hypertension on LA dynamics (71,72). The effect of boosting age to increase stroke risk in AF is multifactorial. In patients with AF, aging is associated with LA enlargement, reduced LAA flow velocity and spontaneous echo contrast, each of which is susceptible to LA thrombus formation (30,63,64). In addition, age is a risk factor for atherosclerosis, including complex aorto-arch-plate, and is associated with stroke independently of AF (65). LV systolic dysfunction predicts iskemic stroke in AF patients receiving no antithrombotic therapy (73–76). Associated conditions, clinical manifestations, and quality of LifeA. Ca uses and Associated Conditions1. Acute causes of AFAF can be associated with acute, temporary causes including alcohol intake, surgery, electrocution, myocarditis, pulmonary embolism, other pulmonary diseases and hyperthyroidism. Successful treatment of the underlying condition can eliminate AF. AF is a common early postoperative complication of myocardial infarction and of heart or taracal surgery.2. AF Without Associated Cardiovascular DiseaseIn younger patients, about 30% to 45% of paroxysmal cases and 20% to 25% of persistent cases of AF occur as solitary AF (13,15,16).3. AF With Associated Cardiovascular Disease-specific cardiovascular conditions associated with AF include valve heart disease (mostly mitral), coronary artery disease (CAD), and hypertension, especially when LV hypertrophy (LVH) is present.4. Neurogenic AFThe autonomic nervous system can cause AF in susceptible patients by showing increased vagale or adrenergic. Many patients experience the onset of AF during periods of improved parasympathetic or sympathetic tone. (77) described a group of patients he characterized in terms of a vagal or adrenergic form of AF. Patients with vagal or adrenergic AF is unusual, but if history reveals a pattern of onset of AF with features of one of these syndromes, the clinician agents may choose more likely to prevent recurring episodes.B. Clinical ManifestationSAF can be symptomatic or asymptomatic even in the same patient. Symptoms vary with the ventricular rate, underlying functional status, duration of AF, and individual patient perceptions. The dysrhythm can be present for the first time with an embolic complication or worsening of HF. Most patients with AF complain about palpitations, chest pain, dyspnea, fatigue or lightheadedness. Release of atrial sodium peptide may be associated with polyuria. AF can lead to tachycardia-mediated cardiomyopathy, especially in patients who are unaware of the ahythmia. Syncope is an unusual but serious complication that usually indicates sinus node dysfunction, valve aortic supnose, HCM, cerebrovascular disease, or an accessory AV pathway.C. Quality of LifeAlthough strokes certainly responsible for much of the functional impairment associated with AF, the rhythm disorder also reduces quality of life. In the Stroke Prevention in Atrial Fibrillation (SPAF) study cohort, the New York Heart Association functional classification, developed for HF, was an insensitive index of quality of life in patients with AF (78). In another study (79), 47 (68%) Of 69 patients with paroxysmal AF considered the dysrhythmia disruptive of their lives. This perception is not associated with either the frequency or duration of symptoms. The AFFIRM trial (Atrial Fibrillation Follow-up Investigation of Rhythm Management), which is still ongoing, compares maintenance of sinus rhythm with rate control in patients with AF, addressing many facets of quality of life, as does the smaller PIAF (Pharmacological Intervention in Atrial Fibrillation) study (80). In selected patients, radio frequency catheter ablation of the AV node and pacemaker insertion improved quality-of-life scores compared to medical therapy (81–86). Long-term oral anticoagulant therapy, which involves regular blood testing and multiple drug interactions, is another factor with important implications for the quality of life of AF patients. Decision analysis found that only 61% of 97 patients preferred anticoagulation therapy to no treatment (87), a significantly smaller proportion than that for whom treatment was recommended according to published guidelines. VII. Clinical Evaluationa. Minimum Evaluation of the Patient with AF1. Clinical History and Physical ExaminationThe initial evaluation of a patient with suspected or proven AF includes characterizing the pattern of the arhythmia as paroxysmal or persistent, determining the cause, and defining associated cardiac and extracardia factors (Table 1). A meticulous history will lead to a well-planned, focused update that serves an effective guide to therapy (2). The operation of an AF patient can usually occur and therapy therapy is initiated into 1 outpatient encounter. Delay occurs when the rhythm is not specifically documented and additional monitoring is needed. Table 16135. Minimum and additional clinical evaluation of patients with atrial fibrillationAF indicates atrial fibrillation; LV, left ventricular; MI, myocardial infarction; RV, real ventricular; LA, left atrial; and AV, atrioventricular. Type IC refers to the Vaughan Williams classification of antiarrhythmic drugs (see Table 2). Minimum evaluation1. History and physical examination, to define• The presence and nature of symptoms associated with AF• The clinical type AF (first episode, paroxysmal, persistent or permanent)• The onset of the first symptomatic attack or date of discovery of AF• The frequency, duration, precipitating factors and forms of termination of AF • The response to any pharmacological agents administered• The presence of any underlying heart disease or other reversible conditions (e.g. hyperthyroidism or alcohol consumption). Electrocardiogram, to identify• Rhythm (verify AF)• LV hypertrophy • P-wave duration and morphology or fibrillatory waves• Contraceptives• Bundle branch block• Before MI• Other atrial arrhythmias• To measure and track the RR, QRS and QT intervals in conjunction with antiarrhythmic drug therapy. Breast radiographer, to evaluate• The lung parenchyma, when clinical findings indicate an abnormality• The pulmonary vasculture, when clinical findings suggest an abnormality4. Echocardiogram, to identify• Valvular heart disease • Left and right atrial size• LV size and function • Peak RV printing (pulmonary hypertension)• LV hypertrophy• LA thrombus (low sensitivity)• Pericardial disease5. Blood tests of thyroid function • For a first episode of AF, when the ventricular rate is difficult to control, or when AF unexpectedly goes on to cardiovasionAdditional testing• One or several tests may be necessary1. Exercise testing• If the adequacy of rate control is concerned (permanent AF)• To reproduce exercise-induced AF• To exclude isecathmia prior to the treatment of selected patients with a type of IC antiarrhythmic agent2. Holter monitoring or event survey• If diagnosis of the type of aritmia is involved• As a means of evaluating rate control3. Transesophageal echocardiography• La thrombus (in the LA Schedule)• To guide cardiovasation4. Electrophysiological study• To explain the mechanism of wide-QRS complex tagic carding• To identify a susceptible arrhythmia such as atrial fluttering or paroxysmal supraventricular tagikcardia• Seeking sites for healing ablation or AV conduction block/changeThe physical examination may BE AF based on irregular pulse. irregular jugular negligent pulsations, and variation in the harshness of the first heart sound. Investigation can also disclose associated valve heart disease, myocardial abnormalities or HF. findings when examined are similar in patients with atrial fluttering, except that the Can be frequent and rapid numbed oscillations can sometimes be visible in the jugular pulse.2. InvestigationsThe diagnosis of AF requires ECG documentation through at least single-lead ECG uptake during the dysrhythmia, which can be facilitated by reviewing emergency department records, Holter monitoring or transtephonic or telemetric surveys. A portable ECG survey tool can help establish the diagnosis in cases of paroxysmal AF and provide a permanent ECG record of the dysrhythmia. If episodes are frequent, a 24-h Holter monitor can be used. If episodes are infrecred, an event recorder, which allows the patient to transmit the ECG to a recording facility when the arrhythmia occurs may be more useful.B. Additional examination of selected patients with afditional examination may include Holter monitoring and exercise testing, trans esophagus echocardiography and/or electrophysiological study. See Table 1 for details. VIII. ManagementThe major issues in managing patients with AF are related to the athremia itself and the prevention of thromboa embolism. In patients with persistent AF, there are fundamentally 2 ways to manage the dysrhythmia: to restore and maintain sinus rhythm or to allow AF to continue and ensure that the ventricular rate is controlled.A. Rhythm control vs Heart Rate ControlReasons for the repair and maintenance of sinus rhythm in patients with AF includes relief from symptoms, prevention of embolism and avoidance of cardiomyopathy.B. Cardiovasion1. Base for cardiovassion of AFCardioversion is often performed electively to restore sinus rhythm in patients with persistent AF. The need for cardiovases can be immediate, however, When the arrhythmia is the main factor responsible for acute HF, hypotension, or worsening of angina pectoris in a patient with CAD. Nonetheless, cardiovassion carries a risk of thromboembolism unless anticoagulation prophylaxe is initiated before the procedure, and this risk seems to be greatest when the arrhythmia is present more than 48 h. Methods of CardiovassionCardioversion can be achieved through drugs or electric shocks. Drugs were commonly used before electric cardiovassie became a standard procedure. The development of new drugs has increased the popularity of pharmacological cardiovasies, although some disadvantages persist, including the risk of drug-induced torade de marks ventricular ticardia or other serious arrhythmias. Pharmacological cardiovassion is still less effective than electric cardiovassion, but the latter requires conscious sedation or anesthesia, while the former does not. The risk of thrombo embossism or stroke does not differ between pharmacological and electric cardiversion. Thus, recommendations for anticoagulation are the same for both methods.C. Pharmacological CardiovasionPharmacological cardiovassion appears to be most effective when used within 7 days of the of AF (88–91). Most such patients patients paroxysmal AF, a first documented episode of AF, or an unknown pattern of AF at the time of treatment. (See Section III, Classification.) A large proportion of patients with recent onset AF experience spontaneous cardiovassion within 24 to 48 h (92–94). It is less likely to occur when AF has persisted for more than 7 days. The relative effectiveness of various drugs differs for pharmacological cardiovassion of AF and atrial flute, but many studies of drug therapy for AF included patients with atrial flute. The dosage, route and rapidity of administration affects efficiency. Reference is made to the Vaughan Williams classification of antiarrhythmic drugs (95), which have been modified to include recently available drugs (Table 2). A summary of recommendations is presented in Tables 10through12 (see Section IX-B, Recommendations for Pharmacological and Electric Cardiovassion of AF, Tables 10–12). Although clinical use of the antiaritmic drugs listed by regulatory agencies, therapeutic use for AF has not been mentioned or approved in all cases in each country. The recommendations given in this document do not necessarily meet government regulations and labeling requirements. Table 26135. Vaughan Williams Classification of Antiarrhythmic Drug ActionsModified with permission of Vaughn Williams EM. A classification of antiarrhythmic actions such as rethinking after a decade of new drugs. J Clin Pharmacol 1984;24:129–47, © 1984 by Sage Publications Inc. (95) to include connections established after publication of the original classification. Type IADisopyramideProcainamideQuinidineType IBLidocaineMexiletineType ICFlecainideMoricizinePropafenoneType IIBeta Blockers (e.g. Propranolol)Type IIIAmiodaroneBretyliumDofetilideIbutilideSotalolType IVCalcium channel antagonists (e.g., verapamil and diltiazem)A frequent issue related to pharmacological cardiovasion is whether the antiarrhythmic drug should be started in hospital or on an outpatient basis. The main concern is the potential for serious adverse effects, including torsade de pointes ventricular lacquer cardy. With the exception of those who have low-dose oral amiodarone (96), virtually all studies of pharmacological cardiovassion are limited to hospitalized patients.D. Electric CardiovasionDirect-current cardiovassion involves an electric shock synchronized with the intrinsic activity of the heart. This ensures that electrical stimulation does not occur during the vulnerable phase of the heart cycle (97). Successful cardiovassion of AF depends on the nature of the underlying heart disease and the current density delivered to the atrial myocardium. The latter, in turn, depends on the stress of the defibrillator capacitor, the output waveform, the size and position of the electrode paddles, and transthoracic impedance. In a randomized controlled study of 301 undergoing elective external cardiversion are patients to anterior-lateral anterior lateral apex and right infraclavicular) or anterior-posterior (sternum and left scapular) paddle positions (98). The energy requirement was lower and overall success was greater with the anterior posterior configuration (87%) than with the anterior-lateral alignment (76%). Cardiovassion is carried out with the patient who fasted and under adequate anesthesia. Short-acting anaesthetic drugs are preferred, because cardiovassion patients are well suited for day care and need to recover quickly after the procedure (99). An initial shock of 100 J is often too low, and an initial energy of 200 J or greater is recommended for electric cardioversion from AF. Devices that deliver current with a biphasic waveform appear to achieve cardiovassion at lower energy levels than those using a monophasic waveform. The primary success rate as measured 3 days after cardiverssion in 100 consecutive subjects was 86% (100); it increased to 94% when the procedure was repeated during treatment with quinidine or dysopyramid. Only 23% of patients remained in sinus rhythm after 1 year and 16% after 2 years; In those relapsed, repeated cardioverssion with antiaritmic medications led to sinus rhythm in 40% and 33% after 1 and 2 years respectively. For patients who relapsed again, a third cardiovassion led to sinus rhythm in 54% at 1 year and 41% at 2 years. Thus, sinus rhythm can be restored in a substantial proportion of patients through direct-current cardioversion, but the rate of relapse is high unless antiarrhythmic drug therapy is given simultaneously. Cardiovassion of patients with implanted pacemaker and defibrillator devices is safe when appropriate precautions are taken. The device must be interviewed immediately before and after cardiovassion to verify appropriate function. The paddles used for external cardiovasive should be positioned as far as possible from the device, preferably in the anterior posterior configuration. The risks of electric cardiovassion are mainly related to embolic events and cardiac arrhythmias. Thrombomic events were reported in 1% to 7% of patients who did not receive anticoagulation before cardiovassion (101,102). Several short arrhythmias can arise, especially ventricular and supraventricular premature beats, bradycardia, and brief periods of sinus arrest (103). Ventricular tachycardia and fibrillation can be unleashed in patients with hypokalmia or digitalis intoxication (104,105). A slow ventricular response to AF in the absence of drugs that slow down AV nodal conduction may indicate conduction defect. The patient must be evaluated in mind before cardiovasies with these issues to avoid symptomatic bradycardia (106). Transient ST segment height can appear on the ECG after cardiovassion (107-108) and blood levels of creatine kinase-MB can rise even without apparent myocardial damage. Prophylactic drug therapy to prevent early recurrence of AF should be considered individually for each patient. Have to (especially early relapse) occurs, antiarrhythmic therapy is recommended in conjunction with the second attempt. Further cardiovassion is of limited value. In highly symptomatic patients, infrecalized repeated cardiovasions can be an acceptable approach.E. Maintenance of Sinus Rhythm1. Pharmacological Therapy to prevent repetition of AFMaintenance of sinus rhythm is relevant in patients with paroxysmal AF (in whom episodes spontaneously terminate) and persistent AF (in whom electrical or pharmacological cardiovasion is required to restore sinus rhythm). Whether paroxysmal or persistent, AF is a chronic disorder, and repetition is likely at some point in most patients (Fig. 4) (109,110). Prophylactic treatment with antiarrhythmic drugs is therefore often necessary. The purpose of maintenance therapy is suppression of symptoms and sometimes prevention of lacquer cardiomyopathy due to AF. It is not yet known whether maintenance of sinus rhythm prevents thromboa embossism, HF, or death (80,111). Figure 4. Arrhythmia-free survival after electric cardiversion in patients with persistent atrial fibrillation. The lower curve represents outcome to a single shock when no prophylactic drug therapy was given. The upper curve depicts the outcome with repeated electric cardieversion in conjunction with antiaritmic drug prophylaxes. ECV indicates electric cardioversion; SR, sinus rhythm. Reproduced with permission of Van Gelder et al., Arch Intern Med 1996;156:2585–92, © 1996, American Medical Association (110). Recurrence of AF is not equivalent to treatment failure. In several studies (112,113) patients with recurring AF often chose to continue drug treatment. Sometimes reducing the arrhythmia burden can make up partial success, while to other patients, any repetition of AF may seem intolerable. Nevertheless, most patients with AF eventually experience recurrence. Risk factors for frequent repetition of paroxysmal AF (over 1 episode per month) include female sex and underlying heart disease (114). In persistent AF, the 4-year arrhythmia-free survival was less than 10% after single-shock electric cardioversion without prophylactic drug therapy (110). Predictors of repetitions within that interval included hypertension, age greater than 55 years, and AF lasts greater than 3 months. In that study, serial traditional and prophylactic drug therapy led to freedom of recurrent AF in only about 30% of patients (110). Predictors of recurrence have included age greater than 70 years, AF lasts greater than 3 months, and HF (110).2. General Approach to Antiaritmic Drug Therapy Before administering any antiaritmic drug, reversible cardiovascular and noncardiovascular precipitants of AF should be addressed. Prophylactic drug treatment is rarely indicated in case of a first-proclaimed episode of AF and can also be avoided in patients with infrequent and well paroxysmal AF. Likewise, when when being infrequental and tolerated, patients experiencing breakthrough arrhythmias may not require a change in antiarrhythmic drug therapy. Beta-blockers can be effective in patients developing ONLY during exercise AF, but few patients have a single specific incitement cause for all episodes of AF, and a majority will not sustain sinus rhythm without antiaritmic drug treatment. In patients with solitary AF, a beta blocker can first be tried, but flecainide, propafenone and sotalol are particularly effective. Amiodarone and dofethlyid are recommended as alternative therapy. Quinidine, procaineamide and disopyramide are not favored unless amiodarone fails or con-indicated. The anticholinergic activity of long-acting dysopyramid makes it a relatively attractive choice for patients with a predicament to fleeceally induced AF. Propafenone is not recommended in fleeceally mediated AF because its (poor) intrinsic beta-blocking activity can exacerbate this type of paroxysmal AF. In patients with adrenergic mediated AF, beta-blockers represent first-line treatment, followed by sotalol and amiodarone. In patients with adrenergically mediated lone AF, amiodarone should be selected later in the order of drug therapy 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 a beta-blocker, sotalol or amiodarone, plus a type of IC agent. A drug that is initially safe can become proarrrhythmic when the patient develops CAD or HF or starts taking other medications that may be arrhyogenic in combination. Thus, the patient should be alerted to the potential significance of such symptoms as synopes, angina pectoris, or dyspnea and warned of the use of noncardiac drugs that could prolong the QT interval. A useful source of information is the Internet and antiarrhythmic drug treatment varies with the agent involved and with patient factors. Prospective trial data on upper limits of drug-induced increases in QRS duration or QT extension are not available. The following recommendations are the consensus of the writing committee. With type IC drugs, QRS widening should last no more than 150% of retention QRS. Exercise testing can be useful to detect QRS widening that occurs only at a rapid heart rate (use-dependent conduction slower). For type IA or type III drugs, with the possible exception of amiodarons, the corrected QT interval should remain in sinus rhythm below 520 ms. During follow-up, plasma potassium and magnesium levels and renal function should be checked periodically, because renal adequacy leads to drug congestion and susceptitiveness to proarchy. In individual patients, serial non-invasive tests may be appropriate to evaluate LV function, especially if clinical HF is during the treatment of Develop. Pharmacological Drugs to Maintain Sinus RhythmFourteen Trials of drug prophylaxis involving patients with paroxysmal AF have been published, and there have been 22 published trials of drug prophylaxis for maintaining sinus rhythm in patients with persistent AF. Comparative data is not sufficient to allow subclassification through drug or etiology. Individual drugs and doses for maintenance of sinus rhythm are given in Table 3. Table 36135. Typical doses of drugs used to maintain Sinus Rhythm in patients with atrial fibrillation **Drug*Daily DosePotentialE Adverse EffectsGI indicate gastroinal; AV, atrioventricular; and HF, heart failure.*Drugs are listed alphabetically.**The drugs and doses given here have been determined by consensus based on published studies.†A loading dose of 600 mg a day is usually given for one month or 1000 mg a day over 1 week.‡Dose should be modified for kidney function and QT interval response during in-hospital Amiodarone†100–400 mgPhotosensitivity, pulmonary toxicity, polyneuropathy, GI upset, bradycardia, torade de pointes (rare), hepatic toxicity, thyroid dysfunctionDisopyramide400–750 mgTorse de pointes, HF, glouctomy, urinary retention, dry oraldophide‡500–1000 MCGTorsade de pointesFlecainide200–300 mgVentricular tachycardia, congestion HF, improved AV nodal conduction (conversion to bully flute)Procainamide1000–4000 mgTorse de pointes, lupus Like syndrome, GI symptomsPropafenone450–900 mgVentricular tachycardia, congestive HF, improved AV nodal conduction (conversion to atrial flutter)Quinidine600–1500 mgTorse de pointes , GI upset, improved AV nodal conductivitySotalol‡240–320 mgTorsade de pointes, congestive HF, bradycardia, worsening of chronic obstructive or bronchospastic lung disease4. Out-of-hospital Initiation of Antiaritmic Drugs in Patients With AFRecommendations for out-of-hospital initiation or intermittent use of antiarrhimic drugs differs for patients with paroxysmal and persistent AF. In patients with paroxysmal AF, the goal is to stop an attack, to prevent repetitions or a combination of both. In patients with persistent AF, the objectives of out-of-hospital drug are initiation to achieve pharmacological cardiovasive from AF, thereby familiarizing the need for electric cardioversies, or improving the success of electric cardiovases and preventing early repetition of AF. Little prospective data is available on the safety of outpatient initiation of antiaritmic drug therapy. Proarrrhythmia (Table 4) is rare in patients with normal ventricular function and baseline QT intervals (41,115) without profound bradycardia. As long as sinus or AV node dysfunction is not suspected, propaneone or flecainide can be initiated from the hospital. Sudden death associated with idiopathic ventricular fibrillation in a structurally normal heart can occur in patients with the Brugada syndrome, an heredifty characterized by ST segment height in right precordial ECG leads and is often accompanied by right right Block. Type I antiarrhythmic drugs can unmask this condition (116,117). Before starting therapy with these drugs, a beta-blocker or calcium channel antagonist should be given to prevent rapid AV conduction or 1:1 API conduction if atrial flute develops (118–122). Because ending paroxysmal AF with flecainide or propafenone may be associated with bradycardia due to sinus node or AV node dysfunction, an initial conversion should be undertaken in hospital before a patient is declared suitable for outpatient pill-in-the-pocket use of these agents for converting repetitions. Out-of-hospital drug termination should be avoided in patients with symptomatic ill sinus sinus syndrome, AV conduction disorders, or bundle-branch block. Table 46135. Types of Proarrrhythmia During treatment with various antiarrhythmic drugs for atrial fibrillation or Atrial Flutter According to the Vaughan Williams ClassificationVW indicates the Vaughan Williams classification of antiarrhythmic drugs (95). VF indicates ventricular fibrillation; AF, atrial fibrillation.A. Ventricular prearriction• Torsade de pointes (VW type IA and type III drugs)•Sustained monomorphic ventricular turgicardy (usually VW type IC drugs)•Sustained polymorphic ventricular ventilcardia/VF without long QT (VW types IA, IC, and III drugs Atrial proarrrhythmia•Provocation of repetition (likely VW types of IA, IC, and III drugs)•Conversion from AF to usually flutter VW type IC drugs)•Increasing defibrillation threshold (a potential problem with VW type IC drugs)C. Abnormalities of conduction or impulse formation•Acceleration of • Accelerated conduction on accessory pathway (digoxin Intravenous verapamel or diltiazem)•Sinus node dysfunction and atrioventricular block (almost all drugs)Sotalol can be initiated in outpatients with little or no heart disease as long as the baseline is uncorrected QT interval less than 450 ms , serum electrolytes are normal, and none of the type III drug-related prearrhythmia risk factors are present. Safety is greatest when sotalol is started when the patient is in sinus rhythm. Amiodarone can usually be given safely on an outpatient basis, even in patients with persistent AF (123), but loading in hospital may be more appropriate when earlier recovery of sinus rhythm is needed, as in patients with HF. Some charging regimens involve giving either 600 mg a day for 4 weeks (123) or greater than or equal to 1 g per day for 1 week (124) followed by lower maintenance doses. Quinidine, procaine, and disopyramide generally should not be started from the hospital. An exception can be made for dysopyramid in patients without heart disease who have a normal QT interval. Currently, the standards for the use of dofeththia do not allow out-of-hospital initiation. Transtelphonic monitoring or other ECG surveillance methods can be used to monitor conduction disorders as pharmacological therapies therapies in patients with AF. Specifically, the PR interval (flecainide, propafenone, sotalol, and amiodarone), QRS duration (flecainide and propafenone), and QT interval (sotalol, amiodarone, and disopyramide) should be measured. As a general rule, antiarithmic drugs should be started at a relatively low dose with upward titration as necessary, rethinking the ECG as each dose change is made. The dosage of other medications used for rate control should be reduced about 6 weeks after the onset of amiodarone and stopped if the rate slows excessively. Concurrent drug therapies should be closely monitored, and both the patient and the doctor should be vigilant of possible adverse drug interactions.5. Recurrence of AF After Cardiovassion: Implications for Drug Treatment Although it has long been known that most repetitions of AF occur within the first month after electric cardiovassion, recent research with internal atrial cardiovassion (125) and day-to-day postconversion studies (126) have established several patterns of recurrence (Fig. 5). In some cases, there is complete failure of direct current countershock to elicitation even a single isolated sinus or ectopic atrial beat, tantamount to a high atrial defibrillation threshold. In others, AF reappears within 1 to 2 minutes after a period of sinus rhythm (127,128). Sometimes recurrence is delayed from 1 day to 2 weeks (126) or more after the shock. Complete shock failure and immediate repetitions occur in about 25% of patients undergoing electric cardiversion, and subacute repetitions occur in about an equal part within about 2 weeks (127). Figure 5. Hypothetical illustration of cardiovassion failure. Three types of repetitions after electric cardioverssion of persistent AF are shown The effectiveness of drugs varies in improving shock conversion and suppression of repetitions ECV indicates external cardiovases; IRAF, first recap from AF after cardiovertion. Amended with permission of Van Gelder et al (129). Available data suggests that starting pharmacological therapy before electric cardiversion (Table 5) can improve immediate success and suppress early repetitions. As a corollary, it seems appropriate to establish therapeutic plasma drug concentrations at the time of cardiovassion and for several weeks after that to optimize the chance of success. After cardioverssion after sinus rhythm, patients receiving drugs that extend QT interval should be observed in hospital for 24 to 48 h to allow the effects of heart rate slowdown and to rapid intervention in the event torso de marks developed. Pharmacological drugs can be started immediately before electric cardiovassie from the hospital or in hospital. The risks of retention include the possibility of a paradoxical increase in the defibrillation threshold, as occurred with flecainide (130); accelerated ventricular rate during loading with type IA or IC drugs in the absence of a nodale block agent and ventricular proarmia (Table 4). Table 56135. Pharmacological Treatment Before Cardiovassion in Patients with Persistent Atrial Fibrillation: Effects of Various Antiarrhythmic Drugs on Acute and Subacute Outcome of Transthoracic Direct Current Shock conversion by DC Shock and PreventIRAF * Suppressed SRAF and Maintenance Therapy ClassRecommendation ClassLevel of EvidenceAll drugs (except beta-blockers and amiorone) should be initiated in hospital IRAF indicates immediate recurrence of atrial fibrillation; SRAF, subacute repetition of atrial fibrillation; and D.C., direct current.*Drugs are listed alphabetically within each class of recommendation. EffectiveAmiodaroneAll drugs in recommendation Class I (except ibutilide) plus beta-blockersIBFlecainideIbutilidePropafenonePropafenone + verapamilQuinidineSotalolUncertain/unknownBeta-blockers DiltiazemIIbBDis OpyramideDofetilideDiltiazemVerapamilDofetilideProcainamideVerapamilPretreatment with pharmacological drugs is most suitable in patients who have previously failed to respond to electric cardiovases and in those who have developed immediate or subacute recurrence of AF. In patients with late repetition and those undergoing initial cardioversion from persistent AF, prepayment is optional (Fig. 10 and 11; see Section IX).6. Selection of antiaritmic drugs in patients with certain heart disease. Heart failurePatients with congestive HF are particularly prone to the ventricular proarth rhythmic effects of antiarrhythmic drugs. Randomized trials demonstrated the safety of amiodarons and dofetilide (given separately) in patients with HF (132,133), and these are the recommended drugs for the maintenance of sinus rhythm (Fig. 11; see Section IX).b. Coronary Artery DiseaseIn stable patients with CAD, beta-blockers may first be considered, but its use is supported by only 2 studies (134–135); data on their effectiveness for maintaining sinus rhythm in patients with persistent AF after cardiovassion is not convincing (134). Sotalol has considerable beta-blocking activity and can therefore be chosen as the initial antiaritmic agent in AF patients with ischaemic heart disease because it is associated with less long-term toxicity than amiodarone (Fig. 11; see Section IX). Both sotalol and amiodarone have reasonable short-term safety profiles, and amiodarons may be preferred in patients with HF (136–138). Flecainide (139) and propafenones are not recommended in these situations. Quintine, procaine and disopyramide are considered third-line treatment choices in patients with CAD. Given the results of the DIAMOND-MI trial (Danish investigations of Arrhythmias and Mortality on Dofetilied in Myocardial Infarction), dofetilia may be considered a second-line rather than a third-line antiarrhythmic agent, but that study involves selected post-myocardial infarction patients in whom the antiaritmic benefit balanced the risk of toksisity.c. Hipertensiewe Heart Heart With LVH is an increased risk of developing torade de marks associated with early ventricular postdepolarizations (140–142). Thus, a drug that does not prolong the QT interval is preferable as first-line therapy, and in the absence of CAD or labeled ventricular hypertrophy, propafenone and flecainide are reasonable choices (Fig. 11; Section IX). Amiodarone prolongs the QT interval, but carries a very low risk of ventricular proarmia; its extracardiac toxicity profile relegates it to second-line therapy in these individuals, but amiodarone becomes first-line therapy when labeled LVH is present. When amiodarone and sotalol either fail or are inappropriate, disopyramide, quintarian, or procainamide can be used as alternatives.d. WPW SyndromeRadiofrequency ablation of the accessory pathway is the preferred therapy for patients with pulpitation syndrome and AF, but antiaritmic drugs can be useful. Digoxin should be avoided due to the risk of paradoxical acceleration of the ventricular rate during AF. Beta blockers do not reduce conduction on accessory paths during pre-preoccupated periods of AF.7. Nonphamacological Correction of AFa. Surgical AblationBased on mapping studies of animal and human AF, Cox et al (143–146) developed a surgical procedure (maze operation) that controls AF in more than 90% of selected patients. The exact mechanism has not been conclusively established, but creating barriers to conduction within the RA and LA limits the amount of myocardium available to propagate reentrant wave fronts, thereby inhibiting sustained AF. Incisions that surround the pulmonary arteries can prevent initiation from AF by potentially isolating aritmogenic foci near the pulmonary arteries from the rest of the atria or by isolating atrial regions with the shortest refractory periods. Amendments to the Cox maze operation involve the ambience of the pulmonary arteries by surgical incisions within the LA and radial incisions in both atria joining the mitral and tricuspid valve annuli (147–149). Surgical operations for AF were successfully combined with operative correction of a variety of structural heart conditions. In patients with highly symptomatic AF who require open heart operations for valve, iscemic or congenital heart disease, consideration should be given to concurrent maze operation for AF or flutter, although this involves additional risk. The mortality rate of an isolated maze twerking is less than 1%, but mortality is higher when the procedure is combined with other types of operative recovery. The morbidity associated with the maze operation includes consequences common to byssorious median tailnotomy and cardiopulmonary, as well as a risk of short-term fluid retention (due to reduced release of atrial sodium peptide), transient reduction in LA and RA transport function, and early postoperative atrational tachyarrhythmias. when the blood supply to the sinus node is disrupted, sinus node dysfunction is dysfunction requires permanent pacemaker implantation. Progressive iterations of these operations have reduced the risk of this complication to less than 10% .b operations. Catheter AblationBased on the success of surgical approaches to AF, several catheter ablation strategies are designed to produce similar effects (150–152). Ablation strategies limited to the RA produce marginal improvement (150), while linear ablation in the LA has been more successful. The recognition that foci begured AF often originates within the pulmonary arteries has led to ablation strategies that target this zone or electrically isolate the pulmonary vein of the LA. Other sites of aritmogenic foci have been found in the better vena cava, the RA and LA, and the coronary sinus. Ablation of these foci eliminates or reduces the frequency of recurring AF in more than 60% of patients, but the risk of recurring AF after a focal ablation procedure is still 30% to 50% over the first year and even higher when more than 1 pulmonary vein is involved. Thus, many patients continue to need antiarithmic drug therapy after ablative therapy from AF (153). Potential complications of catheter ablation for AF include systemic embolism, pulmonary vein stunnosis, pericardial effusion, heart tamponade, and phrenic nerve paralysis. Thus, although these procedures have produced promising results, they have not yet been widely applied. Atrial flutter can develop not only as a distinct arrhythmia, but also during antiaritmic drug therapy from AF, especially with type IC agents. Catheter ablation is more effective than antiarrhithmic drugs for the treatment of atrial flute, reducing the recurrence rate from 93% to 5% when used as first-line therapy (37). In addition, the risk of developing AF may also be lower after catheter ablation of atrial flutter than with pharmacological therapy (29% vs 60% over the first year).c. Suppression of AF by PacingAlthough atrium-based pacing is associated with a lower risk of AF and stroke than ventricle-based pacing for patients requiring pacemakers for bradyarrhythmias, using pacing as a primary therapy for preventing recurring AF has not been validated.d. Internal Atrial Cardioverter/DefibrillatorsThere has been an interest in internal cardiovassion from AF for the past 10 years. Efforts have been made to find shockwave forms that reduce the energy requirements for cardiovassion, making the shock tolerable of waking patients up. One cardioverter (154) capable of atrial sensation and cardiovassion as well as ventricular sensation and awing was evaluated in 290 AF patients. The conversion rate to sinus rhythm was 93%. Another device, with double-room sensing, pacing, and cardiovassion/defibrillation capabilities and a maximum output of 27 J, is a ventricular defibrillator with atrial cardiovassion capabilities. It is designed to treat both atrial and ventricular arhythmias with pacing modities before delivering low-energy shocks. kandidate vir atrial atrial mainly those with infrequenal episodes of poorly tolerated AF, are often suitable for catheter ablation.F. Rate control during AF1. Pharmacological ApproachAn alternative to maintaining sinus rhythm in patients with paroxysmal or persistent AF is control over the ventricular rate. In a recent randomized trial, the therapeutic strategies of rate versus rhythm control produced similar clinical results in 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). The rate is commonly considered control when the ventricular reaction is between 60 and 80 bpm rest and between 90 and 115 bpm during moderate exercise (156,157). Heart rate compilability during AF provides additional information about the status of the autonomic nervous system, which may have independent prognostic implications (158–161). Negative chronotropic therapy from AF is based primarily on depression of conduction across the AV node. Sinus bradycardia and heart block occur in some patients with paroxysmal AF, especially the elderly, as an undesirable effect of pharmacological intervention with beta-blockers, digitalis glycosides, or calcium channel ants. Table 6 describes agents who can be administered to control the ventricular response to AF in an emergency setting. Table 7 describes drugs that block AV nodal conduction; These drugs can be used to achieve rate control both at rest and during exercise or other forms of cardiovascular stress. A combination of drugs is often required to achieve rate control in AF patients in the acute and chronic institutions. Therapy often requires careful dose titration, and some patients develop symptomatic bradycardia that requires permanent pacing. Table 66135. Intravenous Pharmacological Drugs for Heart Rate Control in Patients With Atrial FibrillationBack * Load DoseOnsetMaintenance DoseMajor Side EffectsClass RecommendationHF indicates heart failure.*Drugs are suffered alphabetically insides each class of recommendation.**Type I in congestive HF.†Type IIb in congestive HF.‡Only representative members of the type of beta-adrenergic antagonist drugs are included in the table, but others, similar agents can be used for this indication in appropriate doses. Diltiazem0.25 mg/kg IV about 2 min2-7 min5–15 mg per hour infusionHypotension, heart block, HFI†Esmolol‡0.5 mg/kg over 1 min5 min0.05–0.2 mg·kg-1·min−1Hypotension, heart block, bradycardia, asthma, HFIMetoprolol‡2.5–5 mg IV bolus over 2 min; up to 3 doses5 minNAHypotension, heart block, bradycardia, asthma, HFI†Propranolol‡0.15 mg/kg IV5 minNAHypotension, heart block, bradycardia, asthma, HFI†Verapamil0.075–0.15 mg/kg IV over 2 min3–5 minNAHypotension, heart block, HFI†Digoxin0.25 mg IV every 2 h, up to 1.5 mg2 h0.125–0.25 mg dailyDigitalis toxicity, heart block, bradikarialIb **Table 76135. Recommendations for the use of orally administered pharmacological Rate Control in Patients with Atrial FibrillationBack * Load DoseOnsetUsual Maintenance Dosage **Major Side EffectsRecommendationHF Indicates Heart Failure; PO, by mouth; AFTER, not applicable; HF, heart failure; And PRAY, twice a day.*Drugs are listed alphabetically within each class of recommendation.**Recommended maintenance doses are the usual ones needed, but higher doses may be appropriate in some patients.†The table includes representative members of the type of beta-blocker drugs, but others, similar agents may be used for this indication in appropriate doses. Digoxin0.25 mg PO each 2 h; up to 1.5 mg2 h0.125–0.375 mg dailyDigitalis toxicity, heart block, bradycardialIDiltiazemNA2–4 h120–360 mg daily in divided doses; slow release availableHipotence, heart block, HFIMetoprolol†NA4–6 h25–100 mg BIDHypotension, heart block, bradycardia, asthma, HFIPropranolol†NA60–90 min80–240 mg daily in divided doses ofHypotension, heart block, bradycardia, asthma, HFIVerapamilNA1–2 h120–360 mg daily in divided doses; slow release availableHiepotence, 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 dailyPulmonary toxicity, skin discoloration, hypothyroidism, corneal deposits, optical Warfarin interaction, proarchy Nonpharmacological Regulation of AV Nodal Conductivity and PacingBecause ventricular pacing prolongs the AV nodal receptured period due to concealed retrograde penetration, it eliminates ventricular cycles longer than the pacing cycle length and reduces the number of short ventricular cycles associated with rapid AV conduction during AF As a result, ventricular pacing can be used as a strategy to reduce the irregularity of the ventricular rhythm (162). This modity can be useful for patients with marked mobility in ventricular rates and for those who develop resting bradycardia during treatment with medication prescribed to control rapid ventricular rates with inroads. However, the exact role of pacemaker therapy to regulate the ventricular rate in patients with AF remains controversial.3. Nonpharmacological / AV Nodal AblationAV nodal ablation and permanent pacemaker implantation is highly effective way to improve symptoms in patients with AF who experience symptoms associated with rapid ventricular rate during AF that cannot be adequately controlled with antiarrhythmic or negative chronotropic medications (83,84,86,163). AV nodal ablation is especially useful when an excessive ventricular rate causes a lacquer-mediated decrease in ventricular cysticerclic function despite appropriate medical therapy. The limitations of AV nodal ablation include the persistent need for anticoagulation, loss of AV synchronization, and lifelong pacemaker dependence. There is a small but real risk of sudden death, largely as from torsade de pointes (164). In addition, ablation of the AV conduction system may exclude or limit the later use of newer Treatments. Patients with impaired diastolic ventricular compliance most dependent on AV synchronization for maintenance of heart output (such as HCM or restrictive cardiomyopathies) may experience persistent symptoms after AV nodal ablation and permanent pacemaker implantation. Thus, patients should be counseled regarding each of these considerations before proceeding with this irreversible treatment. The use of catheter ablation to alter AV nodal behavior by eliminating posterior atrial inputs to the AV node has been reported to reduce the ventricular rate during AF and improve heart symptoms without affecting pacemaker implantation (165,166). This technique has several limitations, including the unintended induction of complete AV block and a relatively high risk of raising ventricular rate over the first 6 months after ablation. Thus, AV nodal alteration without pacemaker implantation is only rarely used for patients with rapid ventricular rates during AF.G. Prevention of Thrombo embossism1. Risk Stratificationa. Epidemiological DataThe rate of stroke in patients with AF is related to co-existing cardiovascular disease (4,19,73,74). In a small, retrostrosting, population-based study in Olmsted County, Minnesota, more than 3 decades, the 15-year cumulative stroke rate in people with solitary AF (defined as those younger than age 60 years with no clinical history or echocardiographic signs of cardiopulmonary disease) was 1.3% per year (4). Conversely, in the Framingham Study, the age-adjusted stroke rate over an average follow-up period of 11 years was 28.2% in those with solitary AF, who defined more liberally to include patients with a history of hypertension or cardiomegaly on breast roentgeography; By comparison, the rate was 6.8% in normal controls. In the SPAF study, the annualized rate of iscemic stroke was similar in those with recurrent (3.2%) and permanent (3.3%) DOWN (167). Those with previous stroke or transient issemic attack have a rate of subsequent stroke of 10% to 12% per year despite aspirin use and benefit significantly from treatment with modified-dose oral anticoagulation (168,169). In addition to previous thrombohybes, independent risk factors for iscemic stroke include non-valvular AF HF, hypertension, increasing age and diabetes mellitus (19,73,170–172). Female sex, blood pressure greater than 160 mmHg, and LV dysfunction were each linked to stroke (75,170,173). The relative risk for issemic stroke associated with specific clinical features is given in Table 8. Nearly half of AF-associated strokes occur in patients greater than 75 years old, and AF is the most common cause of turning on a stroke in elderly women (9,171,173). Older people are at increased risk for anticoagulant related bleeding (174) and are therefore less likely to to be met with oral anticoagulation, even in situations for which it has been proven effective (175). Special consideration of the elderly is therefore justified for effective effective prophylaxe (171). Table 86135. Risk Factors for Iscemic Stroke and Systemic Embolism in patients with non-valve supplementary atrial fibrillation Chilli Factors (Control groups)Relative RiskTIA indicate transient iscemic attack. Relative risk refers to compared to atrial fibrillation patients without these risk factors. As a group, patients with non-valvular atrial fibrillation have about a 6-fold increased risk of thromboembolicism compared to patients in normal sinus rhythm. Data from collaborative analysis of five primary prevention trials (75). Previous stroke or TIA2.5History of hypertension1.6Congestive heart failure1.4Advanced age (continuous, per decade)1.4Diabetes mellitus1.7Coronary artery disease1.5Patients with AF associated with thyrotoxicosis, which is often associated with congestive HF, are also at increased risk for stroke (176-178), although the underlying mechanism is not clear (52,179,180). AF is a regular complication of HCM (181), and such patients have a prevalence of stroke and systemic embolism amounting to 2.4% to 7.1% per year (182–185).b. Role of Echocardiography in Risk StratificationTransthoracic Echocardiography: In a meta-analysis of 3 randomized trials of antithrombotic therapy, moderate to severe LV dysfunction was the only independent echocardiographic predictor of stroke in patients with AF when clinical characteristics were also considered (186). The diameter of the LA was a less useful predictor of issemic events (186). Transthoracic Echocardiography TEA is the most sensitive and specific imaging technique for detecting LA and LA appendage (LAA) drumbus, far surpassing transthoracic echocardiography (52). This modality also allows better evaluation for other causes of cardiogenic embolism (187) and superiority in the meting of LAA function (188). Several TEA functions have been associated with thrombowbolicism, including such LA/LAA abnormalities as thrombosis, reduced flow velocity, and spontaneous echo contrast, as well as complex atherototic plaques in the aortic (70,189). Detection of LA/LAA thrombosis stands as a contraindication for choice cardioversion from AF. The absence of observable thrombosis does not preclube thrombo thrombomism after cardioversion if patients do not receive anticoagulation therapy (190,191). A TEA-guided strategy for choice cardiovassion from AF has been reported to lead to comparable outcomes for thromboa embossism and death compared to conventional anticoagulation for 3 weeks before and 4 weeks after cardiovassion (192). Therefore, transthoracic echocardiography is valuable for defining the origin of AF (e.g., detecting rheumatic ant rays valve disease or HCM), and TEA may provide additional information for stratifying thrombolic risk. Three clinical schemes have recently been proposed to address the risk of ischemic stroke in AF patients directly or indirectly based on analyses of prospective stratify cohorts of participants in clinical trials in which antithrombotic therapy was controlled. stratify. One set of criteria was based on multivariate pooled analysis of 1593 participants assigned to the control or placebogroups of 5 randomized primary prevention trials in which 106 istemic strokes occurred over an average follow-up of 1.4 years (19). This scheme divides patients into 2 strata, distinguishing low-risk patients from those with intermediate or high risk. Echocardiographic features were initially not considered, but subsequent analysis of 3 of these trials identified abnormal LV synaplical function as an independent predictor of stroke (186). The SPAF study criteria are based on multivariate analysis of 854 participants awarded aspirin in the SPAF-I and -II clinical trials that were followed up for an average of 2.3 years, during which 68 issemic strokes were observed. A third set of criteria were developed by expert consensus (193) based on the consideration of the 2 preceding schemes and other available data to classify patients into low, intermediate and high-risk groups (Table 9). Table 96135. Published Risk Stratification Schemes for Primary * Prevention of Thromboa Embolicism in Patients with Non-Valuable Atrial FibrillationSourceHigh RiskIntermediate RiskLow RiskBP Indicates Blood Pressure. Patients are classified based on the presence or absence of any risk factor. Adapted with permission of Am J Med, Vol. 109, Pearce et al., Assessment of these schemes for stratification of stroke risk in patients with non-valve supplementary atrial fibrillation, pp. 45-51, © 2000, With permission, from Excerpta Medica, Inc. (195).*Patients with AF and previous thrombohymbolism are at high risk of stroke, and anticoagulation is indicated for secondary prevention in such cases.†This does not distinguish high-risk of intermediate-risk patients.‡Left ventricular dysfunction refers to moderate to severe wall movement 2-dimensional echocardiography, reduced ejection fraction, fractional shortening less than 0.25 by M mode echocardiography, or clinical heart failure. Atrial Fibrillation Investigators (194)†Requirement Greater Than or Equal to 65 YearsAge Less than 65 Years No High-Risk FunctionsHistory of Hypertension No High-Risk FeaturesCoRon legendary artery diseaseDiabetesAmerican College of Breast Doctors (193)Age greater than 75 yearsAge 65-75 years Service less than 65 yearsHistory of hyperten 1 Intermediate Risk FactorsLeft Ventricular Dysfunction‡Coronary Artery Disease More than 1 intermediate risk factorThyrotoxicosisStroke Prevention in Atrial Fibrillation (186)Women greater than 75 yearsHistoric of hypertensionNo high-risk featuresSystolic BP greater than 160 mm HgNo hypertensionLeft ventricular dysfunction† c. Therapeutic ImplicationsThe risk of thrombohymbolism for patients with chronic atrial flutter is generally estimated as higher than for patients with sinus rhythm, but less than for those with persistent or permanent AF. Biblo et al. (196) recently 8 reviewed, revised backworking data on 749,988 hospitalized older patients, 17,413 with atrial flutter and 337,428 with AF. The overall stroke risk ratio for patients with atrial flutter was 1.4 compared to the control group; for patients with AF, the relative risk was 1.6. As a chronic arhythmia, atrial flutter is unusual, and the risk of thromboa embossism is not as well established as it is for AF. Until more robust data become available, and although the overall thromboembolic risk associated with atrial flute may be lower than with AF, it seems prudent to estimate risk using similar stratification criteria.2. Antithrombotic Strategies for the Prevention of Issemic Stroke and Systemic EmbolismFive major randomized trials published between 1989 and 1992 evaluated oral anticoagulation, and 2 tested aspirin for primary prevention of thrombohylasism in patients with non-valvular AF (24,197–199). A sixth trial focused on secondary prevention

among patients who survived non-inducing stroke or transient cerebral issemic attack (169). Meta-analysis according to the principle of intention to treat has shown that modified dose of oral anticoagulation is highly effective for preventing all stroke (both ischaemic 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 duration of follow-up in these trials was generally between 1 and 2 years; the longest was 2.2 years, while the need for antithrombotic therapy typically spans much longer periods of time. Figure 6. Antithrombotic therapy for the prevention of stroke (issemic and hemorrhagic) in patients with non-valvular AF: adjusted dose warfarin compared to placebo. Adapted with permission of Hart et al. (170,200) Ann Intern Med 1999;131:492–501. (The American College of Physicies-American Society of Internal Medicine is not responsible for the accuracy of the translation.) Patient age and the intensity of anticoagulation are the most powerful predictors of great bleeding (201–204). Trial participants were carefully selected and managed at an average age of 69 years. It is therefore unclear whether the relatively low rates of large bleeding also apply to AF patients in clinical practice, who have an average age of about 75 years and whose anticoagulation therapy is less carefully regulated (205,206). The target intensity of anticoagulation involves a balance between preventing issemic stroke and avoidance of hemorrhagic complications. It is important to target the lowest adequate intensity of anticoagulation to reduce the risk of bleeding, especially for elderly AF patients. Maximum protection against issemic stroke in AF is likely to be achieved with an international normalized ratio (INR) of 2 to 3 (168,207,208), while an INR range of 1.6 to 2.5 incomplete efficacy is estimated at approximately 80% of that achieved with a higher intensity anticoagulation (Fig. 7) (207,209). Figure 7. Custom Chance Relationships Relationships iskemic stroke and intracranial bleeding involving intensity of anticoagulation in randomized trials of antithrombotic therapy for patients with AF. The data is from Hylek et al (203,207). In patients with AF who do not have mechanical valves, it is the consensus of the writing group that anticoagulation can be interrupted for a period of up to 1 week for procedures that carry a risk of bleeding, without replacing heparin. In high-risk patients, or when a series of procedures require interruption for a period longer than 1 week, unframed or low-molecular weight heparin can be administered intravenously or subcutaneously, respectively. Low-molecular-weight heparins have several pharmacological advantages over unassured heparin. These include a longer half-life, more predictable bioavailability (greater than 90% after subcutaneous injection), predictable cleanup (allowing once- or twice a day subcutaneous administration), and predictable antitropotic reaction based on body weight, Allowing fixed dose treatment without laboratory monitoring, except under special circumstances, such as obesity, kidney adequacy, Low-molecular weight heparins carry a lower risk of heparin-induced thrombowtopenic than infraction heparin (211). Self-application of low-molecular weight heparins from the hospital is a promising approach that can lead to cost savings in conjunction with elective cardiovasive (212). Aspirin provides only modest protection against stroke for 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-cardiomeembolic versus cardiomembic iscemic strokes (49). On average, cardiebolic strokes are more disabled than non-cardiomic strokes (69). Figure 8. Antithrombotic therapy for the prevention of stroke (isxetmic and hemorrhagic) in patients with non-valvular AF: warfarin compared to aspirin and aspirin compared to placebo. Adapted with permission of Hart et al. (170,200) Ann Intern Med 1999;131:492–501. (The American College of Physicies-American Society of Internal Medicine is not responsible for the accuracy of the translation.) 3. Conversion to Sinus Rhythm and ThromboembolicismRandomized studies of antithrombotic therapy lacking for patients undergoing cardiovassion from AF or atrial flute, but the risk of thromboembolicism was between 1% and 5% in case control range (102,215). There is no solid clinical evidence that cardioversion of AF followed by prolonged maintenance of sinus rhythm effectively reduces thromboa thrombolism. Patients in whom LAA thrombobes are identified by TEA appear to be at high risk of thromboa embossing after cardiovassion from AF or flutter, and they should be treated with anticoagulation for at least 3 to 4 weeks before and after either pharmacological or kardiomiversie. In 'n multicenter studie (192), (192), patients with either AF persisting longer than 2 days or atrial fluttering and previous AF have been channelled to a TEA-guided or conventional strategy. One group received anticoagulation with heparin just before and with warfarin for 4 weeks after cardiovassie. When drum boxes were identified, cardiovases were postponed and warfarin was administered for 3 weeks before repeating TEA. The other group received anticoagulant therapy for 3 weeks before and 4 weeks after cardiovases. Both approaches were associated with a comparably low risk of stroke (0.8% with the TEA guided approach and 0.5% with the conventional approach) after 8 weeks of follow-up. The risk of large bleeding did not differ significantly. There were no differences in the proportion of topics in whom sinus rhythm was successfully restored. Thus, the clinical benefit of the TEA approach is limited to saving time before cardiovassion. Converting from AF to sinus rhythm leads to transient mechanical dysfunction of the LA and LAA (190), known as stunning. This happens after spontaneous, pharmacological (216,217), or electric (217–219) conversion from AF and to radio frequency catheter ablation of atrial fluid (220). The loss of atrial function may be associated with spontaneous echo contrast (190). Recovery of mechanical function can be delayed for several weeks, depending on the duration of AF before the recovery of sinus rhythm (221–223). This may explain why some patients with no demonstrable LA thrombus on TEA before cardiovertion subsequently experienced thrombolic events (191). Presumably, thrombosis forms during the period of stunning and is suspended after the return of mechanical function, which explains the clustering of thrombohymbolic events in the first 10 days after cardiovassion (224). Anticoagulation is recommended for 3 to 4 weeks before and after cardiovassion for patients with AF of unknown duration or who have lasted more than 48 h. Although LA thrombus and systemic embolism have been documented in patients with AF of shorter durations, the need for anticoagulation in such patients is less clear. When acute AF produces hemodynamic instability, immediate cardiovassion should not be delayed, but intravenous heparin or low-molecular weight heparin should be administered first. Protection against late embolism may require the continuation of anticoagulation; the duration of anticoagulation after the procedure depends on the likelihood that AF will be repetitive and at the patient's intrinsic risk of thrombow embolicism. IX. Suggested Management StrategiesA. Overview of Algorithms for managing patients with management of patients with AF requires knowledge of its pattern of presentation (paroxysmal, persistent, or permanent) and decisions on the repair and maintenance of sinus rhythm, control of the ventricular rate, and anticoagulation. These issues are addressed in the various management algorithms for each presentation of AF.1. Newly Discovered or First Episode AF (Fig. 9)%It's not always clear whether the initial presentation of AF is actually the patient's first episode, especially in those with minimal or no symptoms of the dysrhythmia, so both are considered together. In patients who have self-limited episodes of paroxysmal AF, antiaritmic drugs to prevent repetition are usually unnecessary unless AF is associated with severe symptoms associated with hypotension, myocardial isaemia or HF. Whether these individuals require long-term or even short-term anticoagulation is not clear, and the decision should be individually for each patient based on the intrinsic risk of thrombohybolicism. Figure 9. Pharmacological management of patients with newly discovered AF. AF indicates atrial fibrillation; HF, heart failure. In patients with persistent AF, one option is to assume progression to permanent AF, paying attention to antitropotic therapy and control of the ventricular rate. Although it may seem reasonable to make at least 1 attempt to restore sinus rhythm, it is not in the best interests of all patients. An example is the older man without risk factors for thromboembolism in whom asoptomatic AF is discovered on routine examination and control over the ventricular rate is readily achieved. Here, the potential toxicity of antiarrhythmic drugs may outweigh the benefit of restoring sinus rhythm. If the decision is made to restore and maintain sinus rhythm, anticoagulation and rate control are important before cardiovassie. Although long-term antiaritmic therapy may not be necessary to prevent repetitive AF after cardiovertion, short-term therapy can be beneficial. In patients with AF of more than 3 months' duration, early recurrence is common after cardiovassion. Antiaritmic medications can be initiated before cardiovassion (after adequate anticoagulation) in such cases to reduce the likelihood of recurrence, and the duration of drug therapy will be short (e.g., 1 month).2. Recurring Paroxysmal AF (Fig. 10 and 11) Figure 10. Pharmacological management of patients with recurrent paroxysmal AF. *See Figure 11 for proposed drugs. Figure 11. Antiaritmic 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 drugs of choice. † Oversides nonfonacological options to maintain sinus rhythm if drug failure occurs. HF indicates heart failure; CAD, coronary artery disease; and LVH, left ventricular hypertrophy. In patients experiencing short or minimally symptomatic repetitions of paroxysmal AF, it is reasonable to avoid antiarrhythmic drugs, but troublesome symptoms generally call for oppressive antiaritmic therapy. Rate control and prevention of thromboa embossism is appropriate in both situations. In any given patient can have several different antiarrhythmic be effective, and the selection is therefore primarily based on safety. For individuals with no or minimal structural heart disease, flecainide, propafenone, and sotalol is recommended as initial antiarrhythmic therapy because they are generally well tolerated and are essentially without extracardia organ toxicity. When some kind of these drugs are ineffective or are associated with side effects, then second or third line choices include amiodarone, dysopyramide, procaine, and quinidine, which have a greater potential for adverse reactions. A nonpharmacological approach is suitable for some patients, and it should be considered before starting amiodarone therapy. Sometimes a consistent initiation factor can be found. Disopiradide or flecainide can be used in cases of fleeceally mediated AF, while beta-blockers or sotalol are suggested as the initial agent for adrenergously induced AF. Many patients with organic heart disease can be broadly categorized into those with HF, CAD, or hypertension, although other forms of heart disease may also be associated with AF. For patients with HF, safety data supports the choice of amiodarone or dofetilide to maintain sinus rhythm. Patients with iscemic heart disease often require beta-blocker medications. Then sotalol, a drug with both beta-blocking activity and primary antiaritmic efficacy, is first considered, unless the patient HF. Amiodarone and dofetilide are considered secondary agents in this situation. The clinician may consider disopyramide, procaine or cainid on an individual basis. In patients with hypertension without LVH, drugs such as flecainide and propafenone, which do not extend repolarisy and the QT interval, can provide a safety benefit and are first recommended. If these agents either prove ineffective or produce side effects, then amiodarone, dofetilia, and sotalol represent appropriate secondary choices. Disopyramide, procainamide, and climbing are considered third-line agents in this situation. Hypertrophyde myocardium is prone to proarrhythmic toxicity and development of the torso de marks type ventricular lacquer. Amiodarone is proposed as first-line therapy in patients with LVH (wall thickness greater than or equal to 1.4 cm) based on its relative safety compared to several other agents. Because neither ECG nor echocardiography detects LVH invariably as defined by the metesting of myocardial mass, clinicians face a conundrum. The choice of antiarithmic drugs for patients with a history of hypertension is exacerbated by the dearth of prospective controlled trials comparing the safety and effectiveness of drug therapy for AF. The scarcity of data from randomized trials of antiaritmic medications for the treatment of patients with AF generally applies to all patient groups. Consequently, the drug-selection algorithm presented here has been developed as a consensus of experts and is especially on review as additional evidence emerges in this field 10).3. Recurrent persistent AF (Fig. 11 and 12)Patients with minimal symptoms who have at least 1 attempt to restore sinus rhythm may remain in AF with therapy for rate control and prevention of thrombo embolism. Alternatively, those with symptoms that favor sinus rhythm should be treated with an antiarrhythmic drug (in addition to medications for rate control and anticoagulation) before cardiovasies. Choosing an antiaritmic drug should be based on the same algorithm used for patients with recurrent paroxysmal AF.4. Permanent AF (Fig. 12)%Permanent AF is the designation given to cases where sinus rhythm cannot be sustained after cardiversion of AF or when the patient and doctor have decided to allow AF to proceed without further attempts to restore sinus rhythm. It is important to maintain control over the ventricular rate and use antithrombotic therapy, as outlined elsewhere in this document, for all patients in this category. Figure 12. Pharmacological management of patients with recurrent persistent or permanent AF. *See Figs. 11. Initiate drug therapy before cardiovertion to reduce the likelihood of early recurrence of AF. Table 106135. Recommendations for Pharmacological Cardiovasies of AF Less than or Equal to 7 Days Duration **Drug*Route of AdministrationType of RecommendationSLevel of EvidenceReferences * Drugs are listed alphabetically within each category of recommendation and level of evidence.**The doses of medications used in these studies may not be the same as those recommended by the manufacturers. Agents with proven efficiencyDevetehideOralIA133, 225–229FlecainideOral or intravenousIA88–90, 92, 230–235IbutilideIntravenousIA236–241PropafenoneOral or intravenousIA90, 93, 94, 230, 233, 235, 242–252AmiodaroneOral or intravenousIIaA92, 96, 124, 234, 251, 253–260QuinidineOralIIbB88, 90, 91, 93, 242, 256, 257, 261, 262Lose effective or incomplete studied agentsProcainamideIntravenousIIbC237, 238, 263DigoxinOral or intravenousIIIA93, 233, 244, 259, 264–267SotalolOral or intravenousIIIA239, 261, 262, 266, 268Table 116135. Recommendations for Pharmacological Cardiovasies of AF More than 7 Days Duration **Drug*Route of AdministrationType of RecommendationsLevel from EvidenceReferences * Drugs are listed alphabetically within each category of recommendation and level of evidence.**The doses of medication used in these studies may not be the same as those recommended in Table 3 or by the manufacturers. Agents prove effectiveDophilecilideOralIA133, 225–229AmiodaroneOral or intravenousIIaA92, 96, 124, 234, 251, 253–260IbutilideIntravenousIIaA236–241FlecainideOralIIbB8 92, 230–235PropafenoneOral or intravenousIIbB90, 93, 94, 230, 233, 235, 242–252QuinidineOralIIbB88, 90, 91, 93, 242, 256, 257, 261, 262Lose effective or incomplete studied agentsProcainamideIntravenousIIbC237, 238, 263SotalolOral or intravenousIIIA239, 261, 262, 266, or intravenousIIC93, 233, 244, 259, 264–267Table 126135. Recommended dosages dosages Drugs Proven Effective for Pharmacological Cardiovassion of Atrial FibrillationDrug * Route of AdministrationDosage **Potential Adverse EffectsReferencesGI indicates gastroinal; IV, intravenous; PRAY, twice a day.*Drugs are listed alphabetically.**Dosages given in the table may differ from those recommended by the manufacturers.†Insynical data are available on which to base specific recommendations for using one charging regimen over another for patients with iscemic heart disease or impaired left ventricular function, and these drugs should be used carefully or not at all in such patients.‡Thorbillion use of clonal salad to achieve pharmacological conversion of atrial fibrillation is controversial, and safer methods are available with the alternative agents listed in the table. Quintiles should be used with caution. AmiodaroneOralInpatient: 1.2–1.8 g per day in divided dose up to 10 g total, Then 200–400 mg daily maintenance or 30 mg/kg as single diocypotence, bradycardia, QT extension, torade de pointes (rare), GI upset, constipation, phlebitis (IV)92, 96, 124, 234, 251, 253–260Outpatient: 600–800 mg per day split dose to 10 g total, then 200–400 mg a day maintenanceIntravenous/anywhere5–7 mg/kg over 30–60 min, then 1.2–1.8 g per day continuous IV or in divided oral doses up to 10 g total, then 200–400 mg daily maintenanceDofetiliedOralCreatinine cleanup (mL/min) Dosage (MCG BID)QT extension, torsade de pointes; adjust dosage for kidney function, body size and age 133, 225–229greater as 60 50040–60 25020–40 125loos as 20 ContraindicatedFlecainideOral200–300 mg†Hipotence, rapidly exporting atrial flutter88–90, 92, 230–235Intravenous1.5–3.0 mg per kg over 10-20 min†IbutilideIntravenous1 mg over 10 min; repeat 1 mg when necessaryQT extension, torade de pointes236–241PropafenoneOral450–600 mgHypotension, rapidly performing atrial flutter90, 93, 94, 230, 233, 235, 242–252Intravenous1.5–2.0 mg per kg over 10-20 min†Quinidine‡Oral0.75–1.5 g in divided doses about 6-12 h, usually with a rate-delayed drugQT extension, tors GI Hypotension88, 90, 91, 93, 242, 256, 257, 261, 262Table 136135. Recommendations for the use of Pharmacological Drugs to control the rate of Ventricular Responses to Atrial FibrillationDrug * Route of AdministrationType of Recommendational Skin of Evidence * The doses of medication used in these studies may not be the same as those recommended by the manufacturers. DiltiazemIntravenousIAEsmololIntravenousIAVerapamilIntravenous or oralIAOther beta-blockersIntravenous or oralIBDigoxinTravenous or oralIIaBB. Recommendations for managing patients with AFRecommendations are evidence-based and are primarily derived from published data. The weight of evidence was ranked the highest (A) when the data was derived from several randomized clinical trials and intermediates (B) when the data was derived from a limited number proewe, nie-geandomiseerde studies of waarnemingsstudies, of waarnemings waarnemings A lower rank (C) was given when the primary basis for the recommendation was expert consensus. Recommendations follow the format of previous ACC/AHA guidelines for classifying indications, summary of both the evidence and 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 conflicting evidence and/or a deviation of opinion on the use/effectiveness of a procedure or treatment. Class IIa: The weight of evidence or opinion is in favour of the procedure or treatment. Class IIb: Utility/efficiency is less well established 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 on the ACC (www.acc.org), AHA (www.americanheart.org), ESC (www.escardio.org) and NASPE (www.naspe.org) Worldwide Sites and published in the European Heart Journal in mid-October 2001 for a full description of the rationale and evidence supporting these recommendations. Recommendations for Pharmacological and Electric Cardiovassion of OEKlas IImmediary electric cardiovases in patients with paroxysmal AF and rapid ventricular reaction showing ECG evidence of acute MI or symptomatic hypotension, angina, or HF that do not immediately respond to pharmacological measures. (Level of Evidence: C) Cardiovassion in patients without hemodynamic instability when symptoms of AF are unacceptable. (Level of Evidence: C) Class IIaPharmacological or electric cardiovassion to accelerate the recovery of sinus rhythm in patients with a first-detected episode of AF. (Level of Evidence: C) (See Tables 10 to 12 for recommended drugs.) Electric cardiovertion in patients with persistent AF when early recurrence is unlikely. (Level of Evidence: C) Repeated cardiovases followed by prophylactic drug therapy in patients relapsing to AF without antiarrhythmic medication after successful cardiovassion. (Level of Evidence: C) Class IIbPharmacological agents for cardiovassion to sinus rhythm in patients with persistent AF. (Level of Evidence: C) (See Tables 10 to 12 for recommended drugs.) Out-of-hospital administration of pharmacological drugs for cardiovassion of first-detected, paroxysmal, or persistent AF in patients without heart disease or when the safety of the drug in the specific patient is verified. (Level of Evidence: C) (See Table 12.) Class III Electric cardiovassion in patients displaying spontaneous alternation between AF and sinus rhythm over short periods of time. (Level of Evidence: C) Additional cardiovases in patients with short periods of sinus rhythm that retreat to AF despite various cardiovassion procedures and prophylactic antiarrhythmic drug treatment. (Level of Evidence: C) Recommendations for Pharmacological Therapy Maintain Sinus Rhythm (See Table 3)Pharmacological management strategies or algorithms to maintain sinus rhythm in patients with AF (Figs. 9, 10, 11, 12) is based on available evidence and from experience with these agents in other situations. Class IBase selection of pharmacological therapy to maintain sinus rhythm in patients with the turn on or otherwise troublesome symptoms during AF mainly at safety. (Level of Evidence: B) Treat precipitating or reversible causes of AF before starting antiarrhythmic drug therapy. (Level of Evidence: C) Class IIaAd Minister pharmacological therapy to maintain sinus rhythm to prevent the progression of lacquer cardiomyopathy due to AF. (Level of Evidence: C) Infrequered and well tolerated recurrence of AF may in some cases be considered a successful outcome of antiarrhythmic drug therapy. (Level of Evidence: C) Outpatient initiation of antiaritmic drug treatment is appropriate in selected patients. (Level of Evidence: C) Class IIbAd Minister pharmacological therapy to maintain sinus rhythm in asymptomatic patients to prevent atrial remoting. (Level of Evidence: C) Apply pharmacological therapy to maintain sinus rhythm to prevent thromboa embossism or HF in selected patients. (Level of Evidence: C) Administers combinations of antiarrhythmic drugs to maintain sinus rhythm when single-drug therapy fails. (Level of Evidence: C) Class IIIUse of a specific pharmacological drug to maintain sinus rhythm in patients with well-defined proarrhythmia risk factors for that agent. (Level of Evidence: A) Use of pharmacological therapy to maintain sinus rhythm in patients with advanced sinus node or AV node dysfunction in the absence of a functioning electronic heart pacemaker. (Level of Evidence: C) Recommendations for Heart Rate Control in Patients With AF (See Tables 6, 7 and 13)Class IMeasure heart rate response both restfully and during exercise in patients with persistent or permanent AF and control the rate with pharmacological drugs (using a beta-blocker or calcium channel antagonist in most cases) to the physiological range (Level of Evidence: C) Administer intravenous beta-blockers or calcium channel anthagonists (verapamil, diltiazem) in the acute environment to slow the ventricular response to AF in the absence of conduction across an accessory pathway, exercise caution in patients with hypotension or HF. (Level of Evidence: B)Perform immediate electric cardiovassion in patients with acute paroxysmal AF and rapid ventricular reaction associated with acute myocardial in , symptomatic hypotension, angina, or heart failure that does not immediately respond to pharmacological measures. (Level of Evidence: C) Class IIaAd administers a combination of digoxin and a beta-blocker or calcium channel antagonist to control the heart rate at rest and during exercise in patients with AF. The choice of medication should be individualised and the modulated to avoid bradycardia. (Level of Evidence: Evidence: nonpharmacological therapy to control heart rate when pharmacological therapy is inadequate. (Level of Evidence: C) Class IIbAd Minister's Dense as the only agent to control heart rate rests in patients with persistent AF. (Level of Evidence: B) Administers intravenous quinidine, procaineamide, dysopyramide, ibutilide, or amiodarone to hemodynamically stable patients with AF involving conduction across an accessory pathway. (Level of Evidence: B) Immediate cardiovasions are required when very rapid tropycardias or hemodynamic instability occur in patients with AF entailing conduction across an accessory pathway. (Level of Evidence: B) Class IIIAdminister digitalis as the only agent to control a rapid rate of ventricular response to AF in patients with paroxysmal AF. (Level of Evidence: B) Catheter ablation without prior medical therapy to control AF. (Level of Evidence: C) Recommendations for Antithrombotic Therapy at Patients with AF (see Table 14)Table 146135. Recommendations for Antithrombotic Therapy in Patients with Atrial Fibrillation Based on Thromboembolic Risk StratificationPatient FunctionsAntithrombotic TherapyGrade of Recommendation * Risk Factors for thromboemboism include HF, LV ejection fraction less than 0.35, and history of hypertension.AF indicates atrial fibrillation CAD, coronary artery disease; HF, heart failure; INR, international normalized relationship; LV, left ventricular; and TEA, transesophageal echo. Age less than 60 years No heart disease (solitary AF)Aspirin (325 mg daily) or no therapyIAge 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 than or equal to 60 years With diabetes mellitus or CADOral anticoagulation (INR 2.0–3.0)IAddition of aspirin , 81–162 mg daily is optionalIIbAge greater than or equal to 75 years especially womenOral anticoagulation (INR ≈2.0)IHF LV ejection fraction less than or equal to 0.35 Thyrotoxicosis HypertensionOral Anticoagulation (INR 2.0–3.0)IRheuma Prosthetic Heart Disease (Mitral Stalks) Prosthetic Heart Valves Before thrombo embossingism Persistent atrial thrombosis on TEAOral anticoagulation (INR 2.5–3.5 or higher may be appropriate)IClass IAdminister antithrombotic therapy (oral anticoagulation or aspirin) to all patients with AF , except those with solitary AF, to prevent thrombow embossism. (Level of Evidence: A) Individualize the choice of the antithrombotic drug based on assessment of the absolute risks of stroke and bleeding and the relative risk and benefit to a particular patient. (Level of Evidence: A) Chronic oral anticoagulant therapy in a dose adjusted to achieve a target intensity of INR 2 to 3 in patients at high risk of stroke unless con off-indicated. (Level of Evidence: A)a. The need for anticoagulation should be evaluated at regular intervals. (Level of Evidence: A)b. INR must at least determined during the onset of oral anticoagulation therapy and monthly when the patient patient Stable. (Level of Evidence: A) Aspirin in a dose of 325 mg daily as an alternative in low-risk patients or in those with certain contraindications to oral anticoagulation. (Level of Evidence: A) Oral anticoagulation for patients with AF who have rheumatic mittral valve disease or prosthetic heart valves (mechanical or tissue valves). (Level of Evidence: B) Base the target intensity of anticoagulation on the specific type of prosthesis, but it should not be less than INR 2 to 3. (Level of Evidence: B) Class IIaTarget a lower INR of 2 (series 1.6 to 2.5) for primary prevention of iskemic stroke and systemic embolism in patients over 75 years old are considered an increased risk of bleeding complications, but without honest contraindications to oral anticoagulant therapy. (Level of Evidence: C) Manage antithrombotic therapy for patients with atrial fluttering, in general, as for those with AF. (Level of Evidence: C) Choose antithrombotic therapy through the same criteria regardless of the pattern of AF (that is, for patients with paroxysmal, persistent or permanent AF). (Level of Evidence: B) Class IIbInterrupt anticoagulation for a period of up to 1 week for surgical or diagnostic procedures that carry a risk of bleeding, without replacing heparin in patients with AF who do not have mechanical prosthetic heart valves. (Level of Evidence: C) Apply unframed or low-molecular weight heparin intravenously or subcutaneously, respectively, in selected high-risk patients or when a series of procedures require interruption of oral anticoagulant therapy for a period longer than 1 week. (Level of Evidence: C) Manage patients with CAD with anticoagulation (INR 2 to 3) based on the same criteria used for patients without CAD. (Level of Evidence: C) a. A low dose of aspirin (less than 100 mg a day) or clopidogrel (75 mg a day) may be given simultaneously with anticoagulation, but these strategies have not been adequately evaluated and may be associated with an increased risk of bleeding. (Level of Evidence: C) Treatment with aspirin is optional for primary prevention of stroke in patients under the age of 60 years without heart disease or risk factors for thromboa embossism (solitary AF). (Level of Evidence: C) Class IIILong-term anticoagulation for stroke prevention in patients under 60 years of age without heart disease (lone AF) and without risk factors for thromboabolicism. (Level of Evidence: C) Recommendations for Antithrombotic Therapy to prevent ischaemic stroke and systemic embolism in patients with AF undergo cardiovassionClass IAd minister anticoagulation therapy, regardless of the method (electrical or pharmacological) used to restore sinus rhythm. (Level of Evidence: B) Anticoagulate patients with AF lasting more than 48 h or unknown duration for at least 3 to 4 weeks before and after cardiovertion (INR 2 to 3). (Level of Evidence: B) Perform immediate cardioversion in patients with acute onset) AF accompanied by symptoms or signs of hemodynamic instability instability in angina pectoris, myocardial infarction, shock, or pulmonary edema, without waiting for past anticoagulation. (Level of Evidence: C) a. If not contraindicated, administer heparin simultaneously by an initial intravenous bolus injection followed by a continuous infusion in a dose adjusted to extend the activated partial tromboplastin time at 1.5 to 2 times the reference control value.b. Next, provide oral anticoagulation (INR 2 to 3) for a period of at least 3 to 4 weeks, such as for patients undergoing elective cardiovassion. (Level of Evidence: C)c. Limited data from recent studies support subcutaneous administration of low-molecular weight heparin in this indication. (Level of Evidence: C) Screening for the presence of thrombombing in the LA or LAA by TEA is an alternative to routine preanticoagulation in candidates for cardiovassion from AF. (Level of Evidence: B) a. Anticoagulating patients in whom no thrombosis is identified with intravenously unphrased heparin by an initial bolus injection before cardiomyversion, followed by a continuous infusion in a dose tailored to extend the activated partial thrombosis time at 1.5 to 2 times the reference control value. (Level of Evidence: B)b. Next, provide oral anticoagulation (INR 2 to 3) for a period of at least 3 to 4 weeks, such as for patients undergoing elective cardiovassion. (Level of Evidence: B)c. Limited data is available to support the subcutaneous administration of low-molecular weight heparin in this indication. (Level of Evidence: C)d. Treat patients in whom thrombos is identified by TEA with oral anticoagulation (INR 2 to 3) for at least 3 to 4 weeks before and after recovery of sinus rhythm. (Level of Evidence: B) Class IIbCardioversion without TEA guidance during the first 48 h after starting AF. (Level of Evidence: C) a. In these cases, anticoagulation before and after cardiovassion is optional, depending on the assessment of risk. (Level of Evidence: C) Anticoagulate patients with atrial flutter who undergo cardiovassion in the same way as for patients with AF. (Level of Evidence: C) Recommendations for the Prevention and Management of Postoperative AFClass ITreat patients undergo heart surgery with an oral beta-blocker to prevent postoperative AF unless contraindicated. (Level of Evidence: A) In patients developing postoperative AF, rate control achieves by administration of AV nodal blocking agents. (Level of Evidence: B) Class IIaAd Minister sotalol or amiodarone prophylactic to patients at increased risk of developing postoperative AF. (Level of Evidence: B) Restores sinus rhythm in patients developing postoperative AF through pharmacological cardiovassion with ibutilide or direct current cardiovassion, as recommended for nonsurgical patients. (Level of Evidence: B) In patients with recurrent or refractory postoperative AF, try maintenance of sinus rhythm by admissing medications, as recommended for patients with CAD who develop AF. (Level of Evidence: B) Administer Administer medications in patients developing postoperative AF, as recommended for nonsurgical patients. (Level of Evidence: B) Recommendations for managing patients with AF and Acute Myocardial InfarctionClass IElectric Cardiovassion for patients with severe hemodynamic compromise or intractable ischaemia. (Level of Evidence: C) Intravenous administration of digitalis or amiodarons to slow down a rapid ventricular response and improve LV function. (Level of Evidence: C) Intravenous beta-blockers to delay rapid ventricular response in patients without clinical LV dysfunction, bronchospastic disease or AV block. (Level of Evidence: C) Heparin for patients with AF and acute MI unless contraindications to anticoagulation are present. (Level of Evidence: C) Class IIIAdminister type IC antiarrhythmic drugs in patients with AF in setting up acute myocardial infarction. (Level of Evidence: C) Recommendations for managing AF and Ventricular PreexcitationClass ICatheter ablation of the accessory pathway in symptomatic patients with AF who have WPW syndrome, especially those with synops due to rapid heart rate or those with a short bypass tract refract (Level of Evidence: B) Immediate electric cardiovassion to prevent ventricular fibrillation in patients with WPW in whom AF occurs with rapid ventricular reaction associated with hemodynamic instability. (Level of Evidence: B) Intravenous procaine or ibutilide in an effort to restore sinus rhythm in patients with WPW in whom AF occurs without haemodynamic instability in conjunction with a wide QRS complex on the ECG (greater than or equal to 120 ms in duration). (Level of Evidence: C) Class IIbAd minister intravenous quinidine, procaineamide, disopyramide, ibutilide, or amiodarons to hemodynamically stable patients with AF who are conducted across an accessory pathway. (Level of Evidence: B) a. Immediate cardiovases are required when very rapid tropycardias or hemodynamic instability occur in patients with AF entailing conduction across an accessory pathway. (Level of Evidence: B) Class IIIIntravenous administration of beta-blocking agents, digitalis glycosides, diltiazem, or verapamel in patients with WPW syndrome who have prescribed ventricular activation in AF. (Level of Evidence: B) Recommendations for managing AF in patients with hyperthyroidismClass IAd apply a beta-blocker as necessary to control the rate of ventricular reaction in patients with AF complicating thyrotoxicosis unless contraindicated. (Level of Evidence: B) In circumstances when a beta-blocker cannot be used, a calcium channel antagonist (diltiazem or verapamil) administers to control the ventricular rate. (Level of Evidence: B) In patients with AF associated with thyrotoxicosis, use oral anticoagulation (INR 2 to 3) to prevent thrombohybemolism, as recommended for AF patients with other risk factors for stroke. (Level of Evidence: C) a. Once a euthanasia state recovers recommendations for antithrombotic prophylaxe prophylaxe same as for patients without hyperthyroidosis. (Level of Evidence: C) Recommendations for managing AF During PregnancyClass IControl the rate of ventricular reaction with digoxin, a beta-blocker, or a calcium channel antagonist. (Level of Evidence: C) Electric cardiversion in patients who become hemodynamically unstable due to the dysrhythmia. (Level of Evidence: C) Administer antithrombotic therapy (anticoagulant or aspirin) throughout pregnancy to all patients with AF (except those with solitary AF). (Level of Evidence: C) Class IIbAttempt pharmacological cardiovassion by administration of quintiers, procaine, or sotalol in hemodynamically stable patients who develop AF during pregnancy. (Level of Evidence: C) Administers heparin to patients with risk factors for thromboa embossism during the first trimester and last month of pregnancy. Unphrased heparin can be administered either by continuous intravenous infusion in a dose adequate to extend the activated partial thrombublastin time to 1.5 to 2 times the control (reference) value or by intermittent subcutaneous injection in a dose of 10,000 to 20,000 You every 12 h, adjusted to prolong the mid-interval (6 h after injection) activated partial thromboplastin time to control 1.5 times. (Level of Evidence: B) a. Limited data is available to support the subcutaneous administration of low-molecular weight heparin for this indication. (Level of Evidence: C) Apply an oral anticoagulant during the second trimester to patients with a high thrombohybolic risk. (Level of Evidence: C) Recommendations for managing AF in patients with HCMClass ITreat patients with HCM who develop AF with oral anticoagulation (INR 2 to 3), as recommended for other high-risk patients for the prevention of thrombow embossism. (Level of Evidence: B) Class IIaAntiaritmic medications to prevent repetitions. Available data is insufficient to recommend one agent over another in this situation, but disopyramide and amiodarons are generally preferred. (Level of Evidence: C) Recommendations for managing AF in patients with pulmonary diseaseClass IIn patients who develop AF during an acute pulmonary disease or worsening of chronic pulmonary disease, correction of hypoxia and acidosis are the primary therapeutic measures. (Level of Evidence: C) In patients with obstructive pulmonary disease developing AF, a calcium channel antagonistic drug (diltiazem or verapamil) is preferred for ventricular rate control. (Level of Evidence: C) Try electric cardiversion in patients with pulmonary disease that becomes hemodynamically unstable due to AF. (Level of Evidence: C) Class IIIUse of theophylline and beta-adrenergic agonist drugs in patients with bronchospastic lung disease developing AF. (Level of Evidence: C) Use of beta-blockers, sotalol, propafenone and adenosine in patients with obstructive lung disease developing AF. (Level of Evidence: C) This is goedgekeur deur die American College of Cardiology Board of of In August 2001, the American Heart Association Science Advisory and Coordination Committee in August 2001, and the European Association of Cardiology Council and Committee for Practice Guidelines and Policy Conferences in August 2001.When this document is quoted, the American College of Cardiology, the American Heart Association, and the European Society of Cardiology will 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 AND, Wann LS, Wise ACC/AHA/ESC guidelines for managing patients with atrial fibrillation: executive Summary: a report from the American College of Cardiology/American Heart Association Task Force on Practice Guidelines and the European Society of Cardiology Committee for Practice Guidelines and Policy Conferences (Committee to Develop Guidelines for Managing Patients with Atrial Fibrillation). Circulation. 2001;104:2118–2150.This statement was co-published in the October 2001 edition of the Journal of the American College of Cardiology.The American Heart Association makes every attempt to avoid any real or potential conflicts of interest that could arise as a result of an out-of-proportion or a personal, professional or business interest of a member of the writing panel. Specifically, all members of the writing group are expected to complete and submit a Disclosure Questionnaire that shows all such relationships that may be considered actual or potential conflicts of interest. This document is available on the Worldwide 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). Single reprints of this document (the Executive Summary and Summary of Recommendations) are available by calling 800-253-4636 (U.S.ONLY) or writing the American College of Cardiology, Educational Services, 9111 Old Georgetown Road, Bethesda, MD 20814-1699. To obtain a reprint of the full guidelines published in the mid-October edition of the European Heart Journal, call +44 207 424 4200 or +44 207 424 4389, fax +44 207 424 4433, or write Harcourt Publishers Ltd, European Heart Journal, ESC Guidelines - Reprints, 32 Jamestown Road, London, NW1 7BY, UK. To purchase additional reprints, specify version and reprint number (Executive Summary 71-0208; Voltex 71-0209): up to 999 copies, call 800-611-6083 (US only) or fax 413-665-2671; 1000 or more copies, call 214-706-1466, fax 214-691-6342; or Email *Former Task Force member during this writing attempt. References 1 Bellet S. Clinical Disorders of the Heart Rate. 3rd ed. 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