

THE CHOICE OF EMERGENCY THERAPY FOR ATRIAL FIBRILLATION

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Abstract

This article is a literary review that presents a modern view on the strategy and tactics of relieving AF attacks, indications for pharmacological cardioversion (PCV) and electro-pulse therapy (EIT). Based on the presented material, international recommendations for the management of patients with AF are taken, as well as data from a number of modern studies and our own clinical experience in the treatment of arrhythmias. The article also examines the issues of emergency therapy of AF and expresses the author's point of view on the results of a number of studies in this field of arrhythmology.

Keywords: atrial fibrillation, emergency therapy, electrical cardioversion, electrical pulse therapy, pharmacological cardioversion, antiarrhythmic medicines.

Introduction

Atrial fibrillation is one of the most common tachyarrhythmias, the detectability of which in the general population is 1.0-2.0%. In 20% of cases, it is the cause of all strokes, associated with an increase in mortality and the risk of developing chronic heart failure. It is believed that AF is an independent risk factor for cardiovascular diseases, since cardiac arrhythmia leads to a deterioration in the quality of life of patients, the possibility of thromboembolic complications, leading to sudden cardiac death [18]. The leading place in the formation of thromboembolism with AF is the formation of blood clots in the auricle of the left atrium [19]. Paroxysmal attacks of AF are often accompanied by deterioration of the patient's condition, hemodynamic instability, which requires emergency medical care. The tactics of treatment of emergency treatment of AF are based on the choice of themethod of cardioversion: electrical cardioversion - electric pulse therapy orpharmacological cardioversion [17].

The treatment tactics of emergency AF therapy are based on the choice of the cardioversion method: electric cardioversion - electropulse therapy or pharmacological cardioversion. The alternative to the cardioversion strategy is determined mainly by the severity of the course and duration of tachyarrhythmia, taking into account the technical capabilities of its implementation. At the same time, it is impractical to stop continuously recurrent AF paroxysms, as well as AF paroxysms (in the absence of urgent indications) in patients with a high risk of recurrence. Moreover, short-term asymptomatic (low-symptomatic) AF attacks do not require cardioversion. Our tactics for the relief of AF is to choose the method of EIT, an antiarrhythmic drug and the method of its application.





To begin with, we have to admit the imperfection of modern methods for evaluating the effectiveness of antiarrhythmic therapy based on electrocardiographic criteria (with the exception of implantable loop recorders), as well as their low information content.

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An integral component of the diagnosis of AF is an objective assessment of the severity of symptoms caused directly by AF using the EHRA scale (European Heart Rhythm Association) [16], reminiscent of the classification of the severity of chronic heart failure by the New York Association of Cardiologists (NYHA):

Class I - absence of clinical signs;

Class II - "moderately pronounced" symptoms in which normal daily physical activity does not suffer;

Class III - "severe symptoms" in which normal daily activity is reduced;

Class IV - "disabling" symptoms in which normal daily activity is impossible.

This scale for assessing the severity of symptoms of heart failure caused by the arrhythmia itself is of great practical importance for choosing a treatment strategy: sinus rhythm control or heart rate control. This choice is based not only on the subjective opinion and clinical experience of the doctor, but also on object ive quantitative criteria. So, if the patient scores III or IV points on this scale of assessment of the severity of AF-related symptoms, it is advisable to choose a rhythm control strategy as the initial treatment strategy. If the patient scores I or II on the HRA scale, then the choice of a heart rate control strategy is justified as the initial treatment strategy.

At this stage, it is also extremely important to try to identify the heart disease underlying the occurrence of AF, which will allow the treatment of AF to be directed to its cause.

Thus, the main points of this stage are the registration of AF using available methods, the identification of the disease underlying the occurrence of AF, as well as a clinical assessment of the severity of symptoms associated with the arrhythmia itself (according to the EHRA scale).

The use of an antiarrhythmic drug is determined by its effectiveness and safety in AF, which in turn depends on a number of factors: the main and concomitant diseases, the presence or absence of acute heart failure (OSN), coronary heart disease (CHD), sinus node weakness syndrome (SSSI), intraventricular conduction disorders, left myocardial hypertrophy ventricle (LMHV). Although the relief of tachyarrhythmia is carried out with one drug, but if it is ineffective, these should be used. Despite the fact that in most patients, AF paroxysms stop spontaneously during the first hours or days after development, some of them need PCV or EIT. The difference between KV and EIT is that the former is easier to carry out, since it does not require anesthesia, it is less effective than the latter. During and after pharmacological cardioversion, ECG monitoring is necessary to detect the proarrhythmic effects of an antiarrhythmic drug, intraventricular conduction and impaired atrioventricular sinus node function.

Table 1. Antiarrhythmic drugs for the relief of AF

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Medication	Dose and method of administration	The main side effects
Amiodaronum	Intravenous administration of 5 mg / kg in 60 minutes, followed by prolonged intravenous administration of 50 mg / h (up to 2.0 g / day)	Bradycardia hypotension, prolongation of the QT interval, less often tachycardia "pirouette", thromboembolism
Propafenone	Intravenous administration of 2 mg / kg in 10 minutes or once 450-600 mg orally	QRS complex expansion, hypotension, atrial flutter with rapid AV conduction







As can be seen from Table 1, Class I and III antiarrhythmic drugs are used to restore sinus rhythm in patients with AF. The effectiveness of antiarrhythmic drugs is higher in the case of their early administration after the development of an arrhythmia paroxysm. PCV is ineffective when the duration of an AF attack exceeds 7 days.

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In the case of rapid relief of AF paroxysm, preference should be given to intravenous (IV) administration of class 1C antiarrhythmics (propafenone). However, their use is contraindicated in patients with LV systolic dysfunction (LV ejection fraction <40% or CHF) and in acute coronary syndrome (ACS).

High cupping efficacy and good tolerability both with intravenous and oral administration have been proven in a number of placebo-controlled studies [1-3]. In 60-79% of patients, the administration of propafenone leads to the restoration of sinus rhythm in patients within 30-90 minutes, and 56-83% of patients with oral administration after 2-6 hours [1, 3, 4]. In the first hours of their use with intravenous administration of propagenone, the effectiveness in relieving AF is significantly and statistically significantly higher than the effectiveness of amiodarone. When class 1A and 1C antiarrhythmic drugs are used in patients with AF/atrial flutter (TP), which occur with high frequency, it is necessary to first worsen the conduction in the atrioventricular AV node (βblockers - β-AB, verapamil, diltiazem, cardiac glycosides), since they can significantly increase the frequency of ventricular contractions. This principle also applies to the oral administration of propafenone.

For oral relief, 600 mg of propafenone is taken once (450 mg of the drug is recommended for patients with a body weight of less than 60 kg and elderly patients) or 300 mg of flecainide. This method of relieving cardiac arrhythmias with a single oral administration of the antiarrhythmic pill in your pocket is intended mainly for fairly rare mild but prolonged attacks of AF.

According to the duration of AF for less than 2 days, it is possible to restore the sinus rhythm with a single dose of 600 mg of propafenone in 74.4% of patients, on average within 2.3 ± 1.9 hours, and in the placebo group it was restored only in 37.5% of patients (the follow-up period was 8 hours) [5]. In the vast majority of cases, a single oral intake of 450-600 mg of propafenone is well tolerated. However, the first intake of the drug for the purpose of relieving AF is recommended to be carried out in the presence of a doctor, and in the future, if no side effects are detected, the patient can do it on his own.

The U.S.A. Food and Drug Administration approved amiodarone for intravenous use in 1995. At first, only for life-threatening ventricular arrhythmias (ventricular tachycardia - VT/ventricular fibrillation). Further, it was widely prescribed to patients with supraventricular tachyarrhythmias. A meta-analysis of the main six placebo-controlled studies on amiodarone AF cardioversion showed that a significant difference in the frequency of sinus rhythm restoration between intravenous injections of amiodarone and placebo does not occur earlier than 6-8 hours [6]. Based on this, it becomes clear that amiodarone is not the main drug for emergency treatment of AF, because in most cases there is no rapid relief of tachyarrhythmia with its intravenous administration (with the exception of patients with CHF and ACS, in which other antiarrhythmic agents are contraindicated). With intravenous administration, complications from amiodarone are noted infrequently: arterial hypotension is in the first place, bradyarrhythmia develops less often and polymorphic GI torsades de pointes is extremely rare. When monitored for 24 hours, the effectiveness of amiodarone exceeds the effectiveness of placebo by 30-45% [1]. It can be used in





patients with CHF and coronary heart disease, including unstable angina and myocardial infarction

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The procedure for cupping AF with intravenous administration of amiodarone, recommended in the USA, differs from that used in Europe (it is similar to the scheme for cupping VT): intravenous administration of 150 mg in 10 minutes, then continuation of intravenous administration at a rate of 1 mg/min for 6 hours, if necessary intravenous administration Amiodarone is administered at a rate of 0.5 mg/min for the remaining 18 hours of the first day (or until the end of the arrhythmia paroxysm, if it occurred earlier) [2]. If the intravenous administration of amiodarone lasts more than 24 hours, it is advisable to reduce the rate of its administration to 0.25 mg/min [2]. In addition, unlike the European Society of Cardiology (ESC) [1], experts from the American Heart Association (AHA), the American College of Cardiology (ACC), the Heart Rhythm Society (HRS) -AHA/ACC/HRS [2] do not recommend intravenous cupping with amiodarone AF/TP in patients with Wolf-Parkinson-White syndrome (WPW syndrome), considering it to be as dangerous as digoxin, adenosine and verapamil with diltiazem due to the possibility of a significant increase in the frequency of ventricular contractions (class III: dangerous, evidence level C).

Intravenous administration of propafenone is recommended for cardioversion of recent AF in patients without structural myocardial lesions I And intravenous administration of amiodarone is recommended for cardioversion of recent AF in patients with structural myocardial klass I A.

Single oral administration of large doses of propafenone may be recommended for some patients without structural myocardial lesions to relieve recently developed AF (provided that a doctor must first verify the safety of its use) IIa B Digoxin (evidence level A), verapamil, metoprolol (evidence level B), other β -AB (evidence level C) are not effective for converting AF into a sinus rhythm and therefore are not recommended III A, B, C

If a rhythm control strategy is chosen in patients with hyperthyroidism and AF, then before PCV or EIT, it is necessary to normalize the function of the thyroid gland, since otherwise the arrhythmia does not stop or quickly recurs after restoration of the sinus rhythm.

When undertaking any therapeutic interventions in pregnant women, it is necessary first of all to assess the risk of side effects in both mother and fetus. The main antiarrhythmics recommended by ESC for the relief of AF in pregnant women without organic heart damage are flecainide and propafenone [10]. Amiodarone is not recommended for the relief and preventive therapy of AF/TP in pregnant women due to severe fetotoxicity. In exceptional cases, it can be used in severe arrhythmia and the ineffectiveness or inability to use other antiarrhythmic drugs, as well as EIT (safe for the fetus).

AF is often registered in patients with heart valve defects. It is a fairly early manifestation of mitral valve damage. The presence of paroxysmal or persistent forms of AF may be an indication for percutaneous or surgical intervention on the mitral valve. AF also develops in the late stages of aortic malformation. The most acceptable pharmacotherapy strategy for patients with heart valve damage is frequency control (reduction of heart rate and prevention of thromboembolism), since long-term retention of their restored sinus rhythm is unlikely. To prevent thromboembolism, they should be prescribed warfarin, not new oral anticoagulants.

If AF paroxysm leads to hemodynamic instability, manifested by acute heart failure (cardiac asthma, pulmonary edema), severe symptomatic arterial hypotension or severe anginal pain, emergency EIT is indicated. The implementation of EQV, and not ET in these patients (if there are conditions for its implementation) should be considered as an erroneous action. The initial

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discharge energy of a single-phase current at EIT AF is 150-200 J [11]. After EIT, at least 3 hours of electrocardiographic monitoring and hemodynamic monitoring are required [1]. When performing EIT in patients with an implanted pacemaker (EC) or cardioverter defibrillator (CD), the electrodes should be at least 8 cm away from them (an anterior-posterior arrangement of the electrodes is recommended), and the functioning of the implanted EC and CD should be carefully checked after the procedure.

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The discharge during EIT should be synchronized with the heart's own electrical activity. Preference should be given to devices with a biphasic discharge, because their efficiency is higher than with a monophasic discharge [12]. EIT is contraindicated in patients with digitalis intoxication and hypokalemia. Repeated EIT is not recommended in cases where AF quickly recurred after the previous restoration of the sinus rhythm, despite preventive antiarrhythmic therapy. R.Sam et al. [13] it is believed that EIT in AF is inappropriate or contraindicated in hyperthyroidism, digitalis intoxication, acute infectious diseases, hypokalemia, decompensated CHF (with the exception of urgent conditions), contraindications to general anesthesia, paroxysmal AF with very frequent recurrence of arrhythmia, the predicted life expectancy of the patient is less than 1 year (asymptomatic or low-symptomatic AF).

To this can be added attacks of AF with a high risk of recurrence: sinus node weakness syndrome, especially in the absence of an implanted EX; a significant increase in heart chambers; a history of refractory to preventive antiarrhythmic therapy. In all these patients, frequency control may be the optimal treatment strategy.

The success of EIT depends not only on the discharge power, the shape of the current, but also on the position of the electrodes and the resistance of the chest. The electrodes should be pressed tightly against the chest, and the discharge should be applied at the moment of exhalation. Data from some studies (G.Botto et al.) [14] indicate that with the anteroposterior arrangement of the electrodes (right subclavian region and the area under the left scapula), the efficiency of EIT AF is higher than with their anterolateral position (right subclavian region and the area along the midmuscular line at the level of the apex of the heart). Our clinical experience supports this point of view. Multifactorial analysis showed that independent predictors of successful recovery and retention of sinus rhythm are: not very pronounced duration of AF (at least no more than 1 year), small size of the left atrium (diameter within 5 cm), absence of cardiomegaly, young age [1].

When AF paroxysm lasts for more than 48 hours or the time of its onset is unknown, and there is no possibility of transesophageal echocardiography (TPHOCG) before sinus rhythm restoration, oral anticoagulants (OAC) should be prescribed for three weeks before and at least four weeks after electrical cardioversion or pharmacological cardioversion, regardless of the presence or absence of risk factors for thromboembolic complications on the CHA2DS2-VASC scale. According to ESC experts [1], patients with at least one of these risk factors (the exception is female) should take UAC after cardioversion indefinitely (for life). As an antithrombotic therapy, the AHA/ACC/HRS recommendations [2] suggest warfarin (maintaining an international normalized ratio at 2.0-3.0) or new UAC - dibigatran, apixaban, rivaroxaban. However, it should be noted that at the time of writing this article, only dabigatran and rivaroxaban had clinical evidence of successful (not inferior to warfarin) use during EIT and PCV. Mandatory three-week use of UAC before cardioversion can be avoided if no intracardiac blood clots have been detected using EchoCG (in more than 90% of cases they are localized in the auricle of the left atrium).



In patients with AF paroxysm duration <48 h, cardioversion can be performed quickly under the guise of intravenous infusion of unfractionated heparin (UPH) or subcutaneous administration of low molecular weight heparin (MWH) [1]. In the presence of risk factors for the development of ischemic stroke and systemic thromboembolism, patients are recommended to use UAC for life in the future. Given the rapid onset of action of new oral anticoagulants (within 2-4 hours) [15,16], the appointment of these drugs in this clinical situation is possible before or immediately after cardioversion without prolongation of the administration of UPH or MWH [2]. In cases of complicated course of AF, urgent EIT is naturally not postponed until therapeutic hypocoagulation is achieved, even if the duration of arrhythmia is more than 48 hours. EIT is performed against the background of intravenous infusion of NG or subcutaneous administration of MWH with further appointment of UAC for at least 4 weeks.

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