Pathological automatism and triggered activity had focal origin. Thus, the treatment has to be aimed at ablation of the arrhythmogenic region. Some arrhythmogenic places can be precisely characterized by analysis of ECG patterns. Among them are foci located close to the pulmonary veins, sinus node, ventricular outflow tracts or mitroaortic commissura. Classical ablation of these loci is highly successful. In other types of focal arrhythmias electroanatomical systems make possible to create 3D map, with activation sequence allowing for identification of the place where the arrhythmia could be eliminated. In reentrant mechanism of the arrhythmia the impulse circulates around the loop via the cardiac muscle. In case of the atrioventricular nodal reentrant tachycardia, atrial flutter or bundle branch ventricular tachycardia the loop can be easily outlined. Ablation can be performed using the anatomical method without induction of the tachycardia. In patients with the ventricular tachycardia with multiple forms or hemodynamically unstable it is possible to perform electroanatomical map with visualization of the scar and the border zones. In this case the proarrhythmic region in the borderline zone is the aim of linear ablation without induction of tachycardias. In the chaotic tachycardias (atrial or ventricular fibrillation), the arrhythmogenic substrate is too much dispersed to destroy them. Therefore, the ablation is aimed at the trigger which is initiating the arrhythmia (for instance the pathological Purkinje fibers). The excitability of the substrate may be also modified by pacemakers (ventricular or atrial resynchronization). In the life-threatening arrhythmias implantable cardioverter-defibrillator is necessary.

Key words: arrhythmia, pathophysiology, radiofrequency ablation, resynchronization therapy, implantable cardioverter-defibrillator
INTRODUCTION

Increasing technological possibilities has changed the understanding of the cardiac arrhythmias and helped in their individualized treatment. Thanks to the ablation procedures enabling precise destruction of the arrhythmogenic focus we could learn about their heterogeneity. The aim of this paper is to present how our knowledge about the mechanisms underlying various types of the arrhythmia influences the choice of the method that should be used for its nonpharmacological treatment.

Physiological activation of the heart starts in the sinus node, which is localized in the upper part of the right atrium. Next, it runs through the atria to the conduction system of the heart. It is the only physiological connection between the atria and ventricles consisting from the atrioventricular node (where the conduction rate is markedly reduced), the bundle of His which is subdivided into the right and left bundle and which runs along the right and the left side of the interventricular septum, respectively. Each of the bundles has many bifurcations which finally create the Purkinje fibers network activating both ventricles within the short time (less than 100ms).

Sometimes this homogenous tract is disturbed. This may cause arrhythmic perturbations if the three main elements are present: the arrhythmia substrate, the trigger and the predisposing factors.

There are three main types of the mechanisms responsible for generation of arrhythmia: the reentry, the pathological automatism and the triggered activity. The most frequent is the reentrant mechanism (1-2). It requires anatomical or physiological substrate for circulation of the activation. The circle must be longer (the length is determined as a time during which the activation runs around) than the duration of the activation wave corresponding to the effective refractory period. In most arrhythmias the circle is elongated due to presence of the slow conducting regions (for example atrioventricular node, the right atrial inferior isthmus, transitional zone in the infarcted area). The final necessary condition is the presence of the unidirectional block which makes possible activation of only one arm of the conducting system or the myocardial muscle in the site in which it divides into two branches. When the trigger (atrial or ventricular premature beat or important change in the sinus rhythm) generates the impulse during the refractory period in one of the arms, so that it can be conducted in the retrograde direction, the activity generated by the trigger can initiate the arrhythmia.

In the physiological state the diastolic depolarization is observed only in the pacemaker cells localized in the sinus node and in the conduction system of the heart. In some conditions (ex. virus infection, ischemia, toxicity, drugs) the normal myocardial cells can acquire this property and the pathological automatism occurs. The pathological automatism causes that activation of the heart starts not in the sinus node but in the region, where the abnormal cells are located. This may be manifested as frequent atrial or ventricular extrasystoles or
as regular tachycardia. These types of arrhythmias cannot be induced using programmed stimulation during the electrophysiological study. Thus, if the spontaneous arrhythmia is absent during examination of the patient it may be very difficult to treat it successfully. If arrhythmia is frequently present during the study it is easier to destroy its substrate using the electroanatomical procedure. However, if the arrhythmia is observed episodically, the classical methods of ablation should be preferred; among which the most successful seems to be the pace-maping (pacing from ablation catheter create the identical morphology of ECG pattern as during tachycardia).

In case of the triggered activity the arrhythmia is induced by the previous excitation. The substrate is created due to an inhomogeneous conduction in the neighbor muscle layers (for example: because of subendocardial ischemia or in genetically caused channelopathies with random distribution of the pathological ion channels). Difference in conduction time between the different layers is responsible for the difference in the electrical potential between them. This is the reason why the electrical current can flow in a circular manner and induce next excitations.

Both, pathological automatism and triggered activity had focal origin, thus the aim of the treatment is the ablation of the pathological region creating the arrhythmia. The information about its origin is based on the ECG pattern. There are same typical places in the heart with strictly characterized ECG patterns where the arrhythmia may originate. Among them are the pulmonary veins ectopy, the sinus node region, right and left ventricular outflow tracts and mitroaortic commissura. In such cases classical ablation procedure may be performed with high success. In case of other localizations of arrhythmic focus the electroanatomical electrophysiological systems are preferred. The most complex are CARTO or EnSite/NavX systems which make possible to create the 3D map of the chamber with activation sequence enabling to expose the place of the earliest initiation of the arrhythmia. The next step is an ablation of the focus using the radiofrequency energy.

In reentrant mechanism there is no “the earliest” activation because the impulse runs around the circle via different cardiac structures. For some of the arrhythmias (example: atrioventricular nodal reentrant tachycardia or atrial flutter or bundle branch ventricular tachycardia) the localization of the reentrant circle can be strictly determined and the ablation can be done using the anatomical method in the precisely defined region (eg. in typical atrial flutter this is the inferior isthmus of the right atrium) so that the induction of the arrhythmia during the ablation procedure is not required. However, in patients with an accessory conducting pathway the precise electrophysiological mapping must be performed as the procedure preceding the successful ablation.

In patients with the postmyocardial VT with multiple forms of arrhythmia or with hemodynamically unstable tachycardia it is possible to perform electranoatomical mapping of the ventricle which allows for visualization of the scar and the borderline (damaged but alive) zones. It is also possible to find out
the anatomical critical portion of the reentrant circle and to destroy it using RF ablation without the induction of the tachycardia. The proarrhythmic region in the borderline zone is the place where the linear ablation should be performed in this group of patients.

The last group among the arrhythmic disorders is the chaotic tachycardia, as exemplified by the atrial or ventricular fibrillations (1, 3 - 5). The pro-arrhythmic substrate is dispersed so that it is impossible to destroy it. Therefore in this group of patient the ablation is aimed at the locus of the trigger initiating the arrhythmia. Sometimes this procedure is combined with simultaneous modification of the arrhythmia substrate (example: pathological Purkinje fibers in patients with ischemic cardiomyopathy, long QT syndrome, Brugada syndrome or idiopathic VF). In this case the arrhythmogenic substrate can be also modified by permanent pacing techniques (mainly ventricular or atrial resynchronization). In life-threatening arrhythmias the hybrid therapy with implantable cardioverter-defibrillator is necessary.

Precise characteristic of each type of the arrhythmia together with various possibilities of the non pharmacological treatments are presented below.

**Atrioventricular nodal reentrant tachycardia**

Anatomically defined substrate is located in the transitional cell zone, which transmits impulses via the posterior and anterior pathways from the right atrium to the compact a-v node. Anterior input is characterized by the fast conduction (the fast nodal pathway). Posterior input (near the coronary sinus) conducts at slower rate (the slow nodal pathway). If the fast pathway has longer effective refractory period than the slow one, the programmed stimulation can cause the jump in the a-v nodal conduction curve. This means that after the extra stimulus with the interval during which the fast pathway is still refractory, we can observe sudden prolongation of PR (AH) interval (more than 40-60ms) (6 - 7). If activation riches the end of the fast pathway, when its refractory period is already finished, it can go back to the atria, and simultaneously via the bundle of His to the ventricles (under physiological conditions only the latter direction is possible). Via the atrium the action potential can be conducted to the slow pathway (which has already recovered from the refractoriness) and initiate thereby the atrioventricular nodal reentrant tachycardia (8). We are able to abolish this type of arrhythmia with the radiofrequency ablation. The aim of the this procedure is to destroy either the slow or the fast pathway, which interrupts the reentrant circle. In many medical centers an ablation of the slow pathway is preferred because of the lower risk of the atrioventricular block during the learning curve (for the slow pathway the risk is about 1-2%, for fast pathway ablation about 10%). However, in experienced hands both procedures have comparable risk (currently <1%). Therefore, both methods are recommended by the current guidelines (2). For highly symptomatic patients the ablation procedure
is the preferred method of treatment whereas in the asymptomatic patients or if tachycardia is present very rarely the patient himself decides how he should be treated (ablation, drugs or interventional pharmacological treatment) (2).

The preexcitation syndromes

The substrate of the arrhythmia in the preexcitation syndrome is the accessory atrioventricular pathway (2, 9). Usually it is caused by presence of the additional fast conducting pathway (for instance the bundle of Kent responsible for the Wolff-Parkinson-White syndrome) but sometimes it has the a-v nodal characteristics (Mahaim fibers). Some accessory pathways conduct only in one direction. If they conduct only from the ventricles to the atria they are not observed during the sinus rhythm. Therefore, they are named the concealed accessory pathways. Conduction via two pathways (physiological and accessory one) predispose to the atrioventricular reentrant tachycardia: if activation runs from the atria to the ventricles via the physiological conduction system, and goes back using the accessory pathway the arrhythmia is classified as the orthodromic atrioventricular reentrant tachycardia, if activation is circling in the opposite direction, it is defined as the antidromic atrioventricular tachycardia. Presence of the accessory pathway predispose to the atrial fibrillation (observed in 40% of cases with the WPW syndrome) with very fast ventricular response (fast conducting via the accessory pathway). Extremely fast ventricular rate may induce ventricular fibrillation. The risk of sudden cardiac death is 0.15% per year (2), however during the whole life it is greater than 10%. The are two main peaks of the atrial fibrillation during the life – the first one occurs when the patient is about 20 and the second one when he (she) is about 40 years old. Ablation of the accessory pathway eliminates the risk of the sudden death, reduces the risk of the AF to the level of that present in the healthy population (if even it occurs, it is not the life threatening arrhythmia) (10), and eliminates the risk of the atrioventricular tachycardia generation. Thus, the ablation procedure is recommended as the method of choice for all symptomatic patients. Because sudden cardiac death may be the first symptom of the WPW syndrome, all patients with preexcitation signs should undergo an evaluation of the accessory pathway possibilities (2). The most sensitive method is the electrophysiological study. One of the catheters used for this examination can be also used for the ablation procedure. Thus only in the asymptomatic patients with the safe accessory pathway (i.e. with long effective refractory period) but localized in the place with high risk of some serious post-ablation complications (e.g. parahisian localization associated with the risk of generation complete a-v block) the procedure is not performed.

Atrial flutter

In typical form (negative flutter wave in ECG leads II, III, aVF) has macro reentrant mechanism in the right atrium. Usually, it is resistant for the antyarrhythmic treatment and therefore the transesophageal atrial pacing or low energy
cardioversion and recommended for its termination (2). Critical area of slow conduction can be precisely localized in the so called inferior isthmus, which is located between inferior part of the tricuspid valve and the inferior vena cava. Linear lesion using radiofrequency current in this region is safe, highly effective and short-lasting procedure and thus recommended as the gold method of treatment (2).

Other forms of the atrial flutter, named as the atypical ones usually also have the reentrant mechanism (usually around the postsurgical or postablation scar). They also can be treated with ablation, however electroanatomical system is more important for localization of the arrhythmogenic substrate (2).

**Atrial fibrillation**

This type of arrhythmia has very complex background. Usually it is chaotic, multireentrant left atrial arrhythmia (5) with functional reentrant circle changing from beat to beat. This arrhythmia increases mortality rate especially because of the cerebral stroke and heart failure (5). For treatment the atrial fibrillation by elimination of the possibility of the reentrant circles in the left atrium, the surgical maze procedure was proposed (11). Electrophysiologists tried to act along the same line. However the procedure was very long, required a lot of fluoroscopy and was often unsuccessful. Haissaguerre et al. (12) focused on elimination of the triggers initiating the arrhythmia, which are usually located in the pulmonary veins. Unfortunately, elimination of the trigger located inside the vein was associated with the high risk of the postprocedural pulmonary vein stenosis (5). Therefore, elimination of the myocardial slew, connecting the focus with the atrium at the pulmonary vein ostium, is considered to be a safer method. Success rate of this method is about 60-75% (5). Probably isolation of the veins using the anatomical systems increased the success rate because of modification of the substrate (destruction of the rotors – the most important reentrant circles necessary for prolongation of the arrhythmia). This method eliminates electrical (and mechanical) function of more than 15% of the left atrium. Because of an increased risk of the post-procedural left atrial flutter some accessory lines are created (ex. in the left atrial roof between the left and the right superior pulmonary vein and in the left atrial isthmus between the left inferior pulmonary vein and the mitral valve) (13 - 15). In our center this method is used for patients with high level of electrical and structural remodeling. In some patients manifesting the high atrial anisotropy as the intra- or interatrial conduction abnormalities the left atrium is activated with a long-lasting delay. This causes that both ventricles can be activated via the conduction system, at the same time at which the left atrium is activated. Consequently, the systole of the left atrium and that of the left ventricle occur at the same time. Therefore the pressure in the left atrium increases causing remodeling of the atria, which results in the higher vulnerability for atrial fibrillation. The biatrial pacing used as a form of the atrial resynchronization restores the normal sequence of the left atrial and left ventricle
excitation and contraction. The left atrial pressure decreases, the electrical remodeling is reversed and the duration and number of atrial fibrillation paroxysms is reduced (16 - 17). However, the success rate of this method is lower than that of the ablation procedures and in contrast to the ablation, the evidence based medicine it is not satisfactory (5). However, it is certainly an interesting component of the so called hybrid therapy (18).

Ventricular arrhythmias

The ventricular arrhythmias are divided into the categories of the malignant and benign arrhythmias (1, 3, 4). Benign arrhythmias usually are not dangerous while the malignant arrhythmias are life threatening. If patient has not organic or electrical heart disease the arrhythmia is defined as a benign. Most frequently it starts from the right ventricle outflow tract and is manifested as frequent ventricular ectopic beats. Sometimes paired ventricular tachycardia is also observed, however it does not increase the risk of the patient. The risk of the patients is also not related to the clinical symptoms. In patients with the benign ventricular arrhythmia only the antyarrhythmic drugs with a low proarrhythmic potential (beta-adrenergic antagonists and calcium channels blockers) can be used. Other types of pharmacological treatments are too dangerous for patients with the benign ventricular arrhythmias. If arrhythmia is symptomatic, radiofrequency ablation is preferred. Other typical places where the benign arrhythmia can start are the left ventricular outflow tract, the sinuses of the Valsalva (aortic valve), and the mitroartic commissura.

In the malignant arrhythmias the most important is to chose a correct treatment of the basic disease. Non pharmacological treatment of ischemic heart disease (PTCA, CABG) or the heart failure (example: ventricular resynchronization therapy) may be the critical options for treatment of this type of ventricular arrhythmia. The other important point is to prevent the sudden cardiac death. The only helpful method is implantable cardioverter-defibrillator (19 - 23). This device may be combined with the resynchronising therapy, if the life threatening arrhythmia occurs it can be terminated by pacing or by direct current (DC) shock. Because the latter intervention is painful the appropriate treatment should reduce the number of DC interventions. The radiofrequency ablation increases the quality of life, however it does not reduce the risk of the sudden cardiac death (24 - 31). Thus, the hybrid therapy seems to be the most optimal in majority of these patients.

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Author’s address: Edward Koźluk MD, PhD, I Chair and Department of Cardiology, Medical University of Warsaw, Banacha 1 str., 02-097 Warsaw, Poland; e-mail: ekozluk@amwaw.edu.pl