ОГЛЯДИ ЛІТЕРАТУРИ

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DISORDERS OF CARDIAC RHYTHM IN PATIENTS WITH PRE-EXCITATION SYNDROME OF VENTRICLES AND THEIR PHARMACOLOGICAL CORRECTION

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Метою дослідження був аналіз можливості використання різних антиаритмічних препаратів для терапії різних порушень ритму у хворих з синдромом передзбудження шлуночків. Найбільш типовими розладами ритму у хворих з синдромом преекситації є ортодромна реципрокна суправентрикулярна тахікардія, антидромна суправентрикулярна тахікардія, фібриляція та тріпотіння передсердь. Під час приступу тахікардії у хворих із синдромом преекситації можуть виникнути різні клінічні симптоми. Розвиток такої тахікардії нерідко є причиною виникнення раптової смерті. Основним механізмом її розвитку є циркуляція хвилі збудження через атріовентрилулярний вузол, додатковий шлях, передсердя та шлуночки, який називається механізмом макрореентрі. Для купірування приступів атріовентрикулярної реципрокної тахікардії у хворих із WPW синдромом та із вузькими комплексами QRS блокатор кальцієвих каналів верапаміл має ефективність у 95% хворих. Однак для лікування хворих з широкими комплексами QRS верапаміл є протипоказаним. Для лікування даного порушення ритму серцеві глікозиди є те ж протипоказаними. Верапаміл та серцеві глікозиди не можна застосовувати у хворих з синдромом WPW та фібриляцією (тріпотінням) передсердь. Для терапії фібриляції (тріпотіння) передсердь у хворих із синдромом WPW можуть бути використані антиаритмічні препарати I A підкласу (хінідин, прокаїнамід, дизопіраміду, пропафенон) та препарати III класу (аміодарон, соталол). Для купірування пароксизмальної тахікардії у хворих із синдромом передзбудження шлуночків антиаритмічні лікарські засоби рослинного походження (гілуритмал і алапінін) мають високу ефективність й тому є препаратами вибору. Для купірування пароксизмальної тахікардії у хворих із синдромом передзбудження шлуночків може бути досягнуте після введення антиаритмічних препаратів IC підкласу, в особливості застосування пропафенону та енкаїниду. Однак застосування цих препаратів достатньо часто призводить до появи аритмогенного ефекту.Застосування серцевих глікозидів та β-адренергічних блокаторів, наприклад пропралололу у хворих із синдромом преекситації є неможливим, оскільки ці лікарські засоби покращують провідність по додатковому провідному шляху. У хворих із пароксизмальною атриовентрикулярною реципрокною (циркулярною) тахікардією препарати дигіталісу та верапаміл повинні бути виключені, тому що вони є небезпечними у хворих із синдромом WPW, оскільки після їх застосування збільшується швидкість провідності по додатковим шляхам.

Ключові слова: аритмії серця, синдром передзбудження, різні анти аритмічні засоби, раптова смерть, аритмогенна дія, пароксизмальні тахіаритмії.

The aim of the research was to analyze the possibility of using different antiarrhythmic agents in patients with pre-excitation syndrome and disorders of cardiac rhythm. Most typical disorders of cardiac rhythm in patients with pre-excitation syndrome are orthodromic reciprocating supraventricular tachycardia, antidromic supraventricular tachycardia, atrial fibrillation and atrial fibrillation. During attack of tachycardia in patients with syndrome of pre-excitation, different clinical symptoms can be observed. They can range from mild palpitation to syncope. This tachycardia can be even reason of sudden cardiac death. Main its mechanism is macroreentrant circuit involving the AV-node, the additional pathway, the atria, the ventricles. To arrest the attacks of atrioventricular reciprocating tachycardia in patients with WPW syndrome and with narrow complexes QRS, calcium channel blocker verapamil has efficacy in 95% of patients. However, for treatment this arrhythmia with wide complexes QRS verapamil is contraindicated. Besides, it should be taken in account that treatment with this type of cardiac arrhythmia cardiac glycosides is also forbidden. Verapamil and cardiac glycosides are contraindicated for termination of arrhythmia in patients with WPW syndrome and such disorders of cardiac rhythm as atrial fibrillation (flutter). For therapy of atrial fibrillation (flutter) in patients with WPW syndrome antiarrhythmic agents of agents of I A subclass (quinidine, procainamide, disopyramide, propafenone) and III class (amiodarone, sotalol) can be useful. For interruption of paroxysmal tachycardia in patients with pre-excitation syndrome

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antiarrhythmical preparations of plant origin (gilurytmal and allapinin) have high efficacy. Termination of paroxysmal tachycardia in patients with premature excitation of ventricles can be achieved after administration of antiarrhythmic agents of IC subclass, in particular after using of propafenone and encainide. However, treatment with the help of these agents quite often leads to appearance of arrhythmogenic action. In pre-excitation syndrome and cardiac arrhythmias, it is impossible to use drugs, which cause the acceleration of conductivity of nerve impulses in additional pathways (cardiac glycosides, β-blocker agents, for example propranolol). In patients with paroxysmal atrioventricular reciprocating (circular) tachycardia digitalis and calcium channel blockers should be avoided. Such agents as digoxin and verapamil in this arrhythmia can turn out to be dangerous in WPW syndrome, since they raise the conductivity through additional conductive pathways.

***Key words**:* cardiac arrhythmias, pre-excitation syndrome, different antiarrhythmic agents, sudden death, arrhythmogenic action, paroxysmal tachyarrhythmias.

Connection of the research to the planned scientific projects. This study is a part of the research work "Pharmacological study of biologically active substances and drugs for correction of homeostasis disorders of different etiology", state registration number 0117 U 004681.

The aim of the research was to analyze the possibility of using different antiarrhythmic agents in patients with pre-excitation syndrome and disorders of cardiac rhythm.

In the normal condition, conduction from the atria to the ventricles comes via the atrioventricular node (AV)-His-Purkinje system. The reason for pre-excitation is the conditioned conductivity of nerve impulses across an additional pathway. It is also known as an accessory pathway. Syndrome of pre-excitation of the ventricles is an electrophysiological phenomenon in which the period of depolarization of the ventricles occurs earlier than during normal impulse conduction.

The anatomic substratum of syndromes of premature excitation is additional anomalous conductive pathways. There are three main additional pathways of the conduction of impulse (Kent bundle, Mahaim bundle, and James bundle), which are the reason for the pre-excitation of ventricles. The pre-excitation syndrome of the ventricles occurs as a result of the functional activity of such additional pathways. The Kent bundle is mutated myocardial tissue, which is located in the atrioventricular ring. The Kent bundle is capable of conducting impulses from the atrium into the ventricle (the first way). It is considered that the left additional bundle is located in the mitral ring, but the right – in the tricuspid ring. The second way is the James bundle, which connects the atrium and sinus node with the distal part of the atrioventricular node or with the His bundle. The third way is the Mahaim bundle, which consists of fibers of conductive tissue; it connects the upper part of the His bundle or lower part of the atrioventricular node with the myocardium of ventricles. The fourth way is a node-ventricular pathway – Breshenmase pathway, which unites the right atrium with a common part of the His bundle.

The functional activity of Kent pathways is the reason for the formation of Wolff-Parkinson-White syndrome (WPW-syndrome). There are three electrocardiographic features of WPW syndrome:

a) the length of the P-R interval is less than 120 milliseconds in case of the sinus rhythm;

b) the length of the QRS complex is above 120 milliseconds with the initial sloped part, which has jag (Δ-wave), and with the normal final part of QRS complex;

c) the secondary change of the S-T segment, which is directed discordantly (in inverse direction) on an attitude to the main vector of the QRS complex.

Kent pathways directly connect the atria and ventricles. Due to the functional activity of Kent pathways bypassing the AV node occur.

ECG in patients with WPW syndrome can remind the blockade of branches of His bundle or pathological changes of ECG, which are typical for myocardial infarction, or ECG in hypertrophy of ventricles.

Supraventricular reciprocal tachycardia is formatted in the result of the functional activity of such pathways. The development of paroxysmal tachycardia in patients with the syndrome of pre-excitation is conditioned by the re-entry mechanism [1,2,4]. The development of the re-entry mechanism is connected with the presence of two different anatomical pathways of AV-conduction – main and additional bundle, which connects the atrium with the ventricle.

Functional activity of atrial-bundle pathways (James bundles) is the reason for the formation of additional pathways in patients with Clerc–Levy–Critesco syndrome (CLC – syndrome). For ECG in this syndrome, it is typical shortness of P-R intervals, which are less than 120 milliseconds in case of the sinus rhythm. Functional activity of fascicular-ventricular pathways (Mahaim bundles), which are need for shunting of conductivity within the specialized conductive system; they unite the atrioventricular node or brunches of His bundle with ventricular myocardium. Ventricular tachycardia appears due to the functional activity of such pathways.

The development of cardiac arrhythmias is a complication in patients with pre-excitation syndrome. Most frequent arrhythmias in patients with WPW and CLC syndromes are supraventricular paroxysmal tachycardias, which are characterized by a sudden onset, a frequency of ventricular contractions of 150-220 for 1 min, equal RR intervals, and the disappearance of Δ wave [7,8,10].

Paroxysms of atrial fibrillation are very dangerous, since, along the additional paths, the waves of excitation easily pass into the ventricles, which leads to a significant increase in the number of heart contractions up to ventricular fibrillation. ECG signs of paroxysm of atrial fibrillation in patients with the syndrome of pre-excitation of ventricles (WPW and CLC syndromes) are increasing in the number of ventricular contractions over 200 per 1 minute, shortness of RR interval till 0.3 seconds, deformed QRS complexes due to superimposition Δ wave on the initial part of these complexes. It is impossible using in pre-excitation syndrome and cardiac arrhythmias drugs, which cause the acceleration of conductivity of nerve impulses in additional pathways (cardiac glycosides, β-blocker agents, for example, propranolol) [2,5,7].

During the attack of tachycardia in patients with the syndrome of pre-excitation may be different clinical symptoms. They can range from mild palpitation to syncope. This tachycardia can even be the reason for sudden cardiac death [2,7,8,13]. Its primary mechanism is the

macro-reentrant circuit involving the AV-node, the additional pathway, the atria, the ventricles.

For WPW syndrome, the functional and/or anatomical disruption of the atrioventricular node is typical with the development of longitudinal dissociation in the atrioventricular system. In patients with WPW syndrome, sinus impulses get into ventricles simultaneously in two different ways – through the accessory bundle of conduction (the short way) and normal atrioventricular pathway. As a result of the premature activation of the part of ventricles (premature excitation) occurs. In nearly 50% of patients with WPW syndrome, tachyarrhythmias of different types appear. Most widespread tachycardia in patients with the pre-excitation syndrome is such type supraventricular tachycardia as AV reentrant or reciprocating tachycardia (AVRT).

1. Orthodromic reciprocating supraventricular tachycardia: impulse is conducted anterograde through AV-node and retro-grade through Kent bundle;

2. Antidromic supraventricular tachycardia: impulse circulates in the opposite direction (anterograde) through Kent bundle and retro-grade through the atrioventricular node.

3. Paroxysmal atrial fibrillation: at the raised frequency of the atrial contractions, the additional Kent bundle (unlike the AV-node) not capable of slowing down the conduction.

As a result of the velocity of the anterograde conduction through the Kent bundle, the frequency of cardiac beats in patients with atrial fibrillation can sometimes exceed 300 beats per 1 minute. This leads to severe hemodynamic violations and transformation in ventricular fibrillation. Etiological factors of WPW syndrome are the innate anomaly of the conductive pathways or the acquired syndrome (in myocardial infarction, Ebstein disease, mitral valve prolepses, and cardiomyopathies). In adults, the reasons for this disease are not revealed most often [7,8,10].

In patients with WPW syndrome, antidromic atrioventricular reciprocating (circular) paroxysmal tachycardia can be only in 8-10% patients. Tachycardia begins from atrial extrasystoles, which spreads into the ventricle through additional pathways. In ditto time spreading of atrial extrasystole is blocked near the entry in AV-node. That is conditioned by shortness of the refractory period in additional pathways. The monomorphic wide deformed QRS complexes reflect the maximal premature excitation. The frequency of cardiac beats during tachycardia is from 170 to 260 per minute. The inverted P waves in II, III, αVF leads (if it is possible to recognize these waves on ECG) are situated nearly always with big lateness on attitude to the beginning of the QRS complex [7].

The mechanism of this disorder of the rhythm is practically identical to the mechanism of the development reciprocating (orthodromic) paroxysmal tachycardia. Either as in atrioventricular reciprocating (orthodromic) tachycardia, the paroxysm of atrioventricular reciprocating tachycardia with hidden additional pathways begins in these patients after atrial extrasystoles with "critical" interval of the concatenation. However, deceleration of atrioventricular conduction is not so expressed, as at atrioventricular nodal re-entry. Extrasystole passes consecutively through the atrioventricular node and His-Purkinje system. Then extrasystole reaches the place of the joining of the additional pathway with the ventricular wall. Hereafter, extrasystole is conducted in a retrograde direction to the atrium. Then this process is repeated. Ventricular extrasystoles with the critical interval of the concatenation are blocked in the retrograde direction in the His bundle, but they are conducted through the hidden additional pathway to the auricle. Hereon ventricular extrasystole gets into in AV-node, His-Purkinje system, the myocardium of ventricles, in the additional latent pathway, and comes back into atrium [7,8].

The paroxysms of antidromic atrioventricular tachycardia seldom cause a severe disorder of hemodynamics. However, in patients with this disorder of the rhythm, there is raised susceptibility of the transition of antidromic atrioventricular reciprocating paroxysmal tachycardia in atrial fibrillation, and then it is possible the transformation in ventricular fibrillation. This is more often can be in patients with left-side additional pathways. The spontaneous cessation of the paroxysm of antidromic tachycardia usually can be conditioned by the blockade in the atrioventricular node, and only in rare cases interruption of paroxysm can be caused by the blockade in additional pathways [7,8].

Paroxysmal atrioventricular reciprocating (circular) tachycardias in patients with hidden additional pathways, which conduct the impulse selectively in the retrograde direction (latent WPW syndrome) does not occur in rare cases. It is typical for such cardiac arrhythmia ideal conditions in choosing the antiarrhythmic agents to take into account electrophysiological indices of this agent and its concentration in plasma of blood.

In the result of the functional activity of Kent additional pathway, paroxysmal atrioventricular reciprocating (circular) tachycardia can develop. Mechanism of such disorders of cardiac rhythm in patients with hidden additional pathways is the conduction of the impulse selectively in the retrograde direction (latent WPW syndrome) [7,8].

Electrocardiographic signs of paroxysmal atrioventricular reciprocating (circular) tachycardia are:

1) absence of the manifestations of premature excitation of ventricles during the period of sinus rhythm;

2) the narrow (supraventricular) QRS complex during the period of tachycardia;

3) regularity of rhythm during a paroxysm of tachycardia;

4) wave P during tachycardia have a negative polarity in II, III, αVF leads in case of right-side localization of the additional pathways. Positive P waves come to light in I and αVL leads in case of left-side localization of the additional pathways.

In patients with WPW syndrome, the risk of sudden death increases because of shortness of the refractory period in the filament of the additional conductive pathways between auricles and ventricles as well as in connection with recurrence of paroxysms of atrial fibrillation. In WPW syndrome, there is an increased frequency of atrial fibrillation in comparison with common population of people. Thus, in patients with WPW syndrome, the arrhythmic atrial disease develops more often. This contributes to the violation of intraatrial and intraventricular conduction, shortness of atrial refractoriness, and increasing of its dispersion. All this increases the vulnerability of the atria. The electrical instability of the myocardium contributes to the anomalous pathways themselves and especially the often-repeated retrograde atrial excitation during paroxysms of atrioventricular tachycardia.

In patients with WPW syndrome, atrioventricular reciprocal tachycardia may be transformed into atrial fibrillation or atrial flutter. Multiple impulses entering the atrioventricular node in patients with atrial fibrillation or atrial

flutter cause prolongation of its effective refractory period and functional atrioventricular blockade. As a result, an intensive current of irregular impulses occurs through additional pathways to the ventricular tract. On the ECG of patients with atrial fibrillation with the frequency of heart rate, 220-360 for 1 minute abnormal ventricular rhythm is recorded with different QRS complexes ("false ventricular tachycardia"), which have different shapes, widths and amplitudes. If atrial impulses reach the ventricles only through additional paths, QRS complexes are continuous due to the presence of delta waves. If the impulses spread through an atrioventricular node that has temporarily left the state of refractoriness, then QRS complexes remain narrow [7].

More often, atrial fibrillation (atrial flutter) is detected in patients with additional left-sided pathways. During atrial flutter on the ECG, the correct ventricular rhythm with wide cattle complexes (large delta waves) can be recorded. Such an ECG simulates attacks of ventricular tachycardia. If a retrograde block occurs in an additional 2:1 pathway, the number of ventricular complexes decreases to 140-160 for 1 minute. Each additional flutter wave (1:1) through an additional path increases the number of ventricular contractions to 280–320 for 1 minute. The duration of the effective anterograde period additionally is a factor that determines the maximum frequency of the ventricular rhythm, which can be achieved with atrial fibrillation or atrial flutter. A short effective refractory period leads to frequent ventricular contractions with even shorter RR intervals. Frequent and irregular activation of the ventricles in an unusual sequence leads to ventricular fibrillation. The long anterograde effective refractory period of the accessory pathway prevents the occurrence of lethal ventricular arrhythmias.

Pharmacological therapy in patients with WPW-syndrome is not specific. When using an antiarrhythmic agent, a positive effect is observed only in 50-85%. However, the overall amount of patients with WPW-syndrome is significant. Medicament therapy must be implemented with taking into account the mechanism of action of medications in respect of provoked factors (extrasystoles, etc.) and paths, which are used for conduction of circulated impulses, that is to say, one should take into account refractivity and conductivity of normal and additional pathways. Besides, one should take into account the condition of atrial and ventricular myocardium [8,10].

Treatment of reciprocal tachycardia in patients with the pre-excitation syndrome with narrow and wide QRS complexes has a difference. Attacks of atrioventricular reciprocating tachycardia, having re-entry mechanism with participation additional Kent pathway and with narrow QRS complexes after using of verapamil are interrupted in 90-95% of patients [2,3,5]. Bolus intravenous administration of verapamil is administered in dose 10 mg (4 ml of 0,25% solution). It should be taken into account that verapamil has no effect in patients with WPW-syndrome and atrial fibrillation.

In this case, using verapamil is even dangerous. Due to the action of such calcium channel blocker agents, causes diminishment of the duration of the refractory period of the additional pathway occurs in the result of restriction hidden retrograde conductivity. Besides, after the administration of verapamil reflector, a sympathetic effect develops. This is conditioned by diminished arterial pressure.

It should be taken into account that using verapamil in patients with WPW-syndrome and atrial fibrillation or atrial flutter can lead to increasing of conductivity across an additional pathway. This can cause the transformation of atrial fibrillation or atrial flutter in ventricular flutter or ventricular fibrillation [2,5,6, 16]. Thus, intravenous administration of verapamil is allowed only for the treatment of patients with atrioventricular reciprocal tachycardia with narrow complexes QRS. In patients with paroxysmal atrioventricular reciprocating (circular) tachycardia, digitalis, and calcium channel blockers should be avoided [2,5,17]. Such agents as digoxin and verapamil in this arrhythmia can turn out to be dangerous in WPW syndrome since they raise the conductivity through additional conductive pathways. These agents caused the limitation of the retrograde conduction through them. Digoxin is also capable directly of abbreviating the refractory period in the tissue of the additional conductive pathways [2,5,7]. Verapamil causes an increase in reflex sympathetic effect. This is conditioned the shortness of the refractory period in the additional conductive pathways.

For interruption of paroxysm of tachycardia in patients with WPW syndrome, intravenous administration of antiarrhythmic preparation of I class according to Williams's classification – procainamide, disopyramide, gilurytmal (ajmaline), allapinin or antiarrhythmic preparation of III class – amiodarone or sotalol are used. [2,3,5,17].

In case of the paroxysm of atrial fibrillation in patients with WPW syndrome:

a) in the high frequency of the ventricular contractions in connection with a high risk of the development of fibrillation of ventricles it is necessary urgently to realize the electric cardioversion;

b) in the moderate increase of the frequency of ventricular contractions for termination of the paroxysm procainamide is administered intravenously. This antiarrhythmic agent slows down the conductivity of additional pathways.

For prevention of tachyarrhythmia paroxysms, oral administration of preparations of I class (procainamide, quinidine, disopyramide, allapinin, gilurytmal) or III class – amiodarone is used. For preventive maintenance of paroxysms, other antiarrhythmic agents of the A class are used.

The possibility of a positive result after using antiarrhythmic agents is sufficiently stronger in case of the long duration of the effective refractory period of an additional pathway. In patients with narrow QRS complex (in its duration ≤ 220 ms), there is a high-risk transformation of atrial fibrillation in ventricular fibrillation. In case of its duration > 220 < 250 ms there is relative risk such transformation, in such index > 250 < 300 ms there is possible risk and in > 300 ms – small risk [2,17].

It is impossible using of verapamil and digoxin in patients with WPW syndrome combined with atrial fibrillation since they are capable of raising the frequency of cardiac contractions and hereunder to enlarge the risk of the transformation of atrial fibrillation in ventricular fibrillation. Diltiazem and β-blocker drugs should be avoided too.

In case of absence of the effect after administration of agents of I class antiarrhythmic agents of III class is used, in particular, amiodarone intravenously as a bolus in dose 5 mg/kg of mass of the body during 3-5 minutes. Amiodarone is capable of lengthening as retrograde, so and anterograde efficient refractory period of the additional pathways. Amiodarone may also be administered as a bolus in the same dose for a longer period, having the duration 15-20 minutes. Such bolus administration

with a slower rate of administration is needed for the prevention of the reduction of the arterial pressure [11,13,15].

The efficiency of amiodarone in patients with atrioventricular reciprocating paroxysmal tachycardias is conditioned not only by its influence upon AV-node. In some patients (in nearly 50% of patients), amiodarone causes the lengthening of the retrograde effective refractory period of the additional pathways. Amiodarone has an antiarrhythmic effect thanks to simultaneous holding up influence on the retrograde and anterograde knee of the re-entry loop. In general, in the case of single intravenous administration of amiodarone, paroxysms of atrioventricular reciprocating tachycardia were interrupted in 75-80% of patients [2,11,15].

After therapy with the help of amiodarone, interruption of tachycardia (the orthodromic and antidromic) occurs in more rare cases in comparison with antiarrhythmic agents of I A subclass. For the treatment of reciprocating paroxysmal tachycardias, preparations of plant origin – gilurytmal and allapinin have high efficacy.

Termination of paroxysmal tachycardia in patients with premature excitation of ventricles can be achieved after administration of antiarrhythmic agents of IC subclass, in particular after using of propafenone and encainide. However, treatment with help these agents quite often lead to the appearance of arrhythmogenic action. Due to the arrhythmogenic effect of antiarrhythmic agents of IC subclass, in some cases, even the lethal outcome is possible.

For therapy of atrial fibrillation (flutter) in patients with WPW syndrome antiarrhythmic agents of I A subclass (quinidine, procainamide, disopyramide) and III class (amiodarone, sotalol) can be useful [2,3,11,15]. Combined therapy with help the antiarrhythmic agents of I class and III class simultaneously is not used. Treatment with the help of lidocaine is inefficient [2, 17].

The immediate cardioversion should be used for termination of paroxysmal tachycardia in case of the inefficacy of medicinal therapy. The electric cardioversion is for the best also for interruption of this disorder of cardiac rhythm in connection with the risk of development of deterioration of the circulatory dynamic.

For warning of recurrence of paroxysm of atrial fibrillation in patients with WPW syndrome amiodarone is the best antiarrhythmic agent. Sufficient antiarrhythmic effect for termination and prevention paroxysmal tachycardia in patients with the syndrome of pre-excitation of ventricles is achieved after using such agents as allapinin and gilurytmal, which have plant origin [2,3].

In patients with premature excitation by Mahaim type, the accelerated phase of atrioventricular conduction begins below the upper part of the atrioventricular node. The upper part of AV-node provides the slow conduction of nerve impulses. In this connection, the P-R interval remains normal. In consequence of the current part of impulses through the Mahaim bundles, premature excitation appears in a certain part of the ventricle. On ECG Δ-waves in the initial part of the QRS complex reflects premature excitation. The expansion of the QRS complex is also typical [2,3,6-8]. This form of arrhythmia seldom occurs in comparison with other types of pre-excitation.

In rare cases, it is possible the appearance of supraventricular tachycardia in patients with premature excitation by Mahaim type. It is considered that re-entry in the Mahaim bundles can be a reason for the origin of tachycardia. The combination of the different variants of node-ventricular bundles or the fascicle-ventricular bundles, in particular, a combination of the Mahaim bundles with James tract can occur. In this case, the reason of tachycardia is re-entry through the Mahaim bundle and the James bundle.

In case of absence of effect after using antiarrhythmic for interruption of the current of impulses through additional pathways of conduction, the method of their radiofrequency catheter ablation (destruction) is used [12,14]. This destruction is implemented during the electrophysiological study by means of electrodes, which were introduced transcutaneously. The indications for this way of the treatment are the steady supraventricular tachycardia, tolerance to the medicinal treatment, the bad bearableness of the antiarrhythmic agents, the high risk of sudden death in case of atrial fibrillation with a high frequency of the cardiac contractions [6-8].

The treatment of paroxysmal tachycardia with narrow QRS complexes in patients with CLC syndrome is similar to the treatment of this disorder of the rhythm in patients with WPW syndrome. For the treatment of the reciprocating tachycardias with wide QRS complexes, the main importance has anterograde blockade of the additional pathways [7,8]. The efficiency of therapy by antiarrhythmic agents of I class in accordance with V.Williams classification depends on the duration of the effective refractory period of the additional pathways.

The efficiency of treatment with help procainamide, allapinin, and gilurytmal is 80-90% if the anterograde refractory period of the additional pathway is < 270 milliseconds (ms). In the case of a short refractory period of the additional pathway, ≤ 270 ms blockade of the additional pathway by means of these antiarrhythmic agents was caused only in 5-10% of patients. That is why the possibility of a positive result after using antiarrhythmic agents depends on the duration of the effective refractory period of the additional pathway.

In patients with premature excitation by Mahaim type, the accelerated phase of atrioventricular conduction begins below the upper part of the atrioventricular node. The upper part of AV-node provides the slow conduction of impulses. In this connection, the P-R interval remains normal. In consequence of the current part of impulses through the Mahaim bundles, premature excitation appears in a certain part of the ventricle. On ECG Δ-waves in the initial part of the QRS complex reflects the premature excitation. Besides, the significant expansion of the QRS complex is typical. This form of arrhythmia seldom occurs in comparison with other types of pre-excitation. In rare cases, the appearance of supraventricular tachycardia in patients with premature excitation by Mahaim type is possible [7,8].

It is considered that re-entry in the Mahaim bundles can be a reason for the origin of tachycardia. The combination of the different variants of node-ventricular bundles or the fascicle-ventricular bundles, in particular, a combination of the Mahaim bundles with James tract can occur. In this case, the reason of tachycardia is re-entry through the Mahaim and the James bundles. Preparations of choice for treatment of patients with premature excitation by Mahaim type and paroxysmal supraventricular tachycardia are amiodarone and gilurytmal.

Conclusions

1. For termination of disorders of cardiac rhythm in patients with syndromes of pre-excitation of ventricles, intravenous administration of antiarrhythmic preparation

of IA subclass (procainamide, disopyramide, gilurytmal, allapinin) or antiarrhythmical preparation of III class – amiodarone can be useful.

2. The abovementioned antiarrhythmic agents can be administered orally for prophylaxis of paroxysmal tachyarrhythmia in patients with syndromes of pre-excitation.

3. Sufficient antiarrhythmic effect for termination and prevention paroxysmal tachycardia in patients with the syndrome of pre-excitation of ventricles is achieved after using such agents as allapinin and gilurytmal, which have plant origin.

4. For suppression of attacks of atrioventricular reciprocating tachycardia, having re-entry mechanism with participation of additional Kent pathway and with narrow QRS complexes, calcium channel blocker agent verapamil has a sufficient efficacy.

5. It is impossible to use of verapamil for interruption of atrial fibrillation or atrial flutter in patients with WPW-syndrome. In this case, using verapamil is even dangerous since this preparation raises the conductivity through the additional conductive pathway.

6. Other drugs, which cause the acceleration of conductivity of nerve impulses in additional pathways (cardiac glycosides, β-blocker agents, for example, propranolol) are contraindicated for the treatment of disorders of cardiac rhythm in patients with the syndrome of pre-excitation of ventricles.

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