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Disorders of cardiac rhythm in patient with syndrome preexitation of ventricles and their pharmacological correction

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The goal of investigation was analysis of possibility of using of different antiarrhythmic agents in patients with syndrome preexitation and disorders of cardiac rhythm.

In normal condition conduction from atriums to the ventricles comes via atrioventricular node (AV)-His-Purkinje system. Reason of pre-excitation is conditioned conductivity of nerve impulses across additional pathway. It is known also as 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.

Anatomic substratum of syndromes of premature excitation is additional anomalous conductive pathways. There are three main additional pathways of the conduction of impulse (Kent bunch, Mahaim bunch and James bunch), which are the reason of preexcitation of ventricles. Syndrome pre-excitation of the ventricles is occurred in result of functional activity of such additional pathways. The Kent bunch is mutated myocardial tissue, which is located in atrioventricular ring. The Kent bunch is capable to conduct impulses from atrium into ventricle (the first way). It is considered that left additional bunch is located in the mitral ring, but the right – in tricuspid ring. The second way is the Jame's bunch, which connects the atrium and sinus node with distal part of atrioventricular node or with His bundle. The third way is the Mahaim bunch, which consists of filaments of conductive

tissue, it connects the upper part of the His bunch or lower part of the atrioventricular node with myocardium of ventricles. The forth way is node-ventricular pathway – Breshenmase pathway, which unites right atrium with common part of His bundle.

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

a) the length of 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 sloped initial part, which has jag (Δ-wave), and with normal final part of QRS complex;

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

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

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

Functional activity of atrial-bundle pathways (James fibers) is reason of 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 fibers), which are need for shunting of conductivity within specialized conductive system; they unite

atrio-ventricular node or brunches of His bundle with ventricular myocardium. Ventricular tachycardia is appeared due to functional activity of such pathways.

Development of cardiac arrhythmias is complication in patient with syndrome preexitation. Most frequent arrhythmias in patients with WPW and CLC syndromes is 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 significant increasing of number of heart contractions up to ventricular fibrillation. ECG signs of paroxysm of atrial fibrillation in patients with syndrome of preextation 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 initial part of these complexes. It is impossible using in preextation 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 attack of tachycardia in patients with syndrome of preexitation may be different clinical symptoms. They can range from mild palpitation to syncope. This tachycardia can be even reason of sudden cardiac death [2,7,8,13]. Main its mechanism is macro-reentrant circuit involving the AV-node, the additional pathway, the atria, the ventricles.

For WPW syndrome is typical the functional and/or anatomical violation in atrioventricular node with development of longitudinal dissociation in atrioventricular system can occured. In patients with WPW syndrome sinus impulses get into ventricles simultaneously by two different ways – through the accessory bunch of conduction (the short way) and through normal atrioventricular pathway. As a result the premature activity of part of ventricles (premature excitation) is occurred. In nearly 50% of patients with WPW syndrome

tachyarrhythmias of different types are appeared. Most widespread tachycardia in patients with syndrome preexitation is such type supraventricular tachycardia as AV reentrant or reciprocating tachycardia (AVRT).

1. Ortodromic reciprocating supraventricular tachycardia: impulse is conducted antero-gradely through AV-node and retro-gradely through Kent bunch;

2. Antidromic supraventricular tachycardia: impulse circulates in opposite direction (antero-gradely) through Kent bunch and retro-gradely through the atrioventricular node.

3. Paroxysmal atrial fibrillation: at raised frequency of the atrial contractions the additional Kent bunch (in difference to AV-node) not capable to slow down the conduction.

As a result velocity of the anterograde conduction through the Kent bunch the frequency of cardiac beats in patients with atrial fibrillation can sometimes exceed 300 beats per 1 minute. This brings about 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 adult people the reasons of 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 ventricle through additional pathways. In ditto time spreading of atrial extrasystole is blocked near entry in AV-node. That is conditioned by shortness of refractory period in additional pathways. The monomorfic 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 mechanism of the development reciprocating (orthodromic) paroxysmal tachycardia. Either as in atrioventricular reciprocating (orthodromic) tachycardia, 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 ventricular wall. Hereafter extrasystole is conducted in retrograde direction to atrium. Then this process is being repeated. Ventricular extrasystoles with 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 auricle. Hereon ventricular extrasystole gets into in AV-node, His-Purkinje system, myocardium of ventricles, in latent additional pathway and come back into atrium [7,8].

The paroxysms of antidromic atrioventricular tachycardia cause seldom the severe disorder of hemodynamics. However, in patients with this disorder of the rhythm there is raised susceptibility of 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 atrioventricular node and only in rare cases interruption of paroxysm can be caused by blockade in additional pathways [7,8].

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

In result of functional activity of Kent additional pathway paroxysmal atrioventricular reciprocating (circular) tachycardia can be developed. Mechanism of such disorders of cardiac rhythm in patients with hidden additional pathways is conduction of the impulse selectively in 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 period of sinus rhythm;

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

3) regularity of rhythm during 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 risk of sudden death is increased because of shortness of the refractory period in filament of the conductive additional 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, atrial arrhythmic 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 is occurred through

additional pathways to the ventricular tract. On the ECG of patients with atrial fibrillation with 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 presence of delta wave. If the impulses are 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 anterograde effective 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. In result of using antyarrhthmic agent positive effect is observed only in 50-85%. However, overall amount of patients with WPW-syndrome is significant. Medicament therapy must be realized with taking in account mechanism of action of medications in respect of provoked factors (exatrasystoles etc.) and paths, which are used for conduction of circulated impulses, that is to say it should be taken in account refractivity and conductivity normal and additional pathways. Beside of, it should be taken in account condition of atrial and ventricular myocardium [8,10].

Treatment of reciprocal tachycardia in patients with syndrome preexitation with narrow and wide QRS complexes has 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 taking in account, that verapamil has no effect in patients with WPW-syndrome and atrial fibrillation. In this case using of verapamil is even dangerous. Due to action of such calcium channel blocker agent causes diminishment of duration of refractory period of additional pathway is occurred in result of restriction hidden retrograde conductivity. Beside of, after administration of verapamil reflector sympathetic effect is developed. This is conditioned by diminishment of arterial pressure.

It should be taken in account, that using of verapamil in patients with WPW-syndrome and atrial fibrillation or atrial flutter can lead to increasing of conductivity across 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 to abbreviate the refractory period in tissue of the additional conductive pathways [2,5,7]. Verapamil causes the increasing of 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 antiarrhythmical preparation of I class accordinning to Williams's classification – procainamide, disopyramide, gilurytmal (ajmaline), allapinin or antiarrhythmical 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 high frequency of the ventricular contractions in connection with high risk of the development of fibrillation of ventricles it is necessary urgently to realize the electric cardioversion;

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

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

Possibility of positive result after using antiarrhythmic agents is sufficiently bigger in case of long duration of effective refractory period of additional pathway. In patients with narrow QRS complex (in its duration $\leq$ 220 ms) there is 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 in combine with atrial fibrillation, since they are capable to raise 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 absent of the effect after administration of agents of I class antiarrhythmic agents of III class is used, in particular amiodarone intravenously as bolus in dose 5 mg/kg of mass of the body during 3-5 minutes. Amiodarone is

capable to lengthen as retrograde, so and anterograde efficient refractory period of the additional pathways. Amiodarone may be administered also as bolus in the same dose during for longer period, having the duration 15-20 minutes. Such bolus administration with slower rate of administration is need for prevention of the reduction of the arterial pressure [11,13,15].

Efficiency of amiodarone in patients with atrioventricular reciprocating paroxysmal tachycardias is conditioned not only 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 antiarrhythmic effect thanks to simultaneous holding up influence on retrograde and anterograde knee of 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 help amiodarone interruption of tachycardia (the orthodromic and antidromic) is occurred in more rare cases in comparison with antiarrhythmic agents of I A subclass. For 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 bring about appearance of arrhythmogenic action. Thanks to arrhythmogenic effect of antiarrhythmic agents of IC subclass in some cases it is possible even the appearance lethal result.

For therapy of atrial fibrillation (flutter) in patients with WPW syndrome antiarrhythmic agents of 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 help of lidocaine is inefficient [2, 17].

The immediate cardioversion should be used for termination of paroxysmal tachycardia in case of inefficacy of medicinal therapy. The electric cardioversion is for the best also for interruption of this disorder of cardiac rhythm in connection with 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 syndrome of preexitation 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 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 current of part of impulses through the Mahaim filaments premature excitation is appeared in the certain part of ventricle. On ECG Δ-waves in initial part of the QRS complex reflects the premature excitation. The expansion of the QRS complex is typical too [2,3,6-8]. This form of arrhythmia is occurred seldom in comparison with other types of preexitation.

In rear 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 filaments can be a reason of the origin of tachycardia. The combination of the different variants of node-ventricular filaments or the fascicle-ventricular filaments, in particular combination of the Mahaim filaments with James tract can be occurred. In this case the reason of tachycardia is re-entry through the Mahaim bunch and the James bunch.

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 radio-frequency catheter ablation (destruction) is used [12,14]. This destruction is realized 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 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 treatment of this disorder of the rhythm in patients with WPW syndrome. For treatment of the reciprocating tachycardias with wide QRS complexes the main importance has anterograde blockade of the additional pathways [7,8]. Efficiency of therapy by antiarrhythmic agents of I class in accordance with V.Williams classification depends on duration of effective refractory period of the additional pathways.

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 case of 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 possibility of positive result after using of antiarrhythmic agents is depended from duration of effective refractory period the additional pathway.

In patients with premature excitation by Mahaim type the accelerated phase of atrioventricular conduction begins below the upper part of 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 current of part of impulses through the Mahaim filaments premature excitation is appeared in the certain part of ventricle. On ECG Δ-waves in initial part of the QRS complex reflects the premature excitation. Beside of, the significant expansion of the QRS complex is typical. This form of arrhythmia is occurred seldom in comparison with other types of preexitation. In rear cases it is possible the appearance of supraventricular tachycardia in patients with premature excitation by Mahaim type [7,8].

It is considered that re-entry in the Mahaim filaments can be a reason of the origin of tachycardia. The combination of the different variants of node-ventricular

filaments or the fascicle-ventricular filaments, in particular combination of the Mahaim filaments with James tract can be occurred. In this case the reason of tachycardia is re-entry through the Mahaim and the James bunches. Preparations of choosing for treatment 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 preexitation 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. Abovementioned antiarrhythmic agents can be administered orally for preventive purpose for prophylaxis of paroxysmal tachyarrhythmia in patients with syndromes of preexitation.

3. Sufficient antiarrhythmic effect for termination and prevention paroxysmal tachycardia in patients with syndrome of preexitation 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 additional Kent pathway and with narrow QRS complexes, sufficient efficacy has calcium channel blocker agent verapamil.

5. It is impossible using of verapamil for interruption of atrial fibrillation or atrial flutter in patients with WPW-syndrome. In this case using of verapamil is even dangerous since this preparation raises the conductivity through 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 treatment of disorders of cardiac rhythm in patients with syndrome of preexitation of ventricles.

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Резюме

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.

Resume

The goal of investigation was analysis of possibility of using of different antiarrhythmic agents in patients with syndrome preexitation and disorders of cardiac rhythm. Most typical disorders of cardiac rhythm in patient with syndrome preexitation are ortodromic reciprocating supraventricular tachycardia, antidromic supraventricular tachycardia, atrial fibrillation and atrial fibrillation. During attack of tachycardia in patients with syndrome of preexitation can be different clinical symptoms. 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 atrias, the ventricles.

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. Beside of, it should be taken in account that treatment of this type of cardiac arrhythmia cardiac glycosides are forbidden also. 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 syndrome preexitation 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 help these agents quite often bring about appearance of arrhythmogenic action.

It is impossible using in preextation 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). 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, syndrome preexitation, different antiarrhythmic agents, sudden death, arrhythmogenic action, paroxysmal tachyarrhythmias.