The Influence of the VVP pacing on cardiohemodynamics in the critical patients.

Report III.

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In critical patients with acute cardiovascular insufficiency passed by various version of the supraventricular tachycardia, treated by respiratory and routine anti-shock therapy and in experimental setting upon narcotized mongrel dogs with opened chest the influence of the rarefying paired ventricular pacing (St_1 + St_2 delayed impulse; code in the former USSR - VVP) on cardiohemodynamics at intact valves of the heart was studied. Dopplerechocardiography, different modes of cardiac pacing, catheterization of the heart and main vessels, electromagnetic blood flowmeter and angiocardiography were performed. It was proved that VVP pacing in the presence of the intact ventriculo-atrial conduction created functional retrograde blockade and V_1-V_1 > A-A version of the ventriculo-atrial dissociation. VVP pacing application promoted to significant growth of efficiency of a routine anti-shock therapy, increased volume loading on both ventricles, myocardial contractility, its performance and aortal pressure. At the given form of the retrograde dissociation different types of the hemodynamically effective and ineffective atrial cycles, affecting on the process of the filling and contraction of the various chambers of the heart were investigated.

Key words: rarefying paired ventricular pacing, VVP, cardiohemodynamics, retrograde conduction, ventriculo-atrial dissociation, tachycardia.

Introduction

Treatment of the acute cardiovascular insufficiency in critical patients is topical problem. In spite of the fact that the given pathology in critical medicine is pluricausal (hemorrhage, polytrauma, intoxications, stroke, various heart diseases etc), basic efforts at treatment of the shock is focused on correction of the hemodynamic disturbances, such as restoration of circulating blood volume, myocardial contractility and its performance, level of the systemic arterial pressure and interrupting of tachycardia that often appeared during i.v. infusion of adrenoreactive medicaments. Tachycardia completely destroys diastolic function of the ventricles and creates serious problems at treatment of the shock syndrome. Rarefying paired ventricular pacing is one of the effective methods of the correction of the persisting supraventricular tachycardia (1), that often accompany acute cardiovascular insufficiency. Therefore, we investigate the influence of the VVP pacing on intracardiac and central hemodynamics and retrograde conductivity in the presence of acute cardiovascular insufficiency, accompanied by supraventricular tachycardia among critical patients, treated by the routine anti-shock medical supplies.

Material and methods of research.

In clinical conditions 7 critical patients with a various pathology were surveyed, at which at the certain stages of the basic illness were generated various forms of the persistent supraventricular tachycardia complicated by shock, which proceeded with critical reduction of the arterial pressure and oxygen saturation and was not responded to intravenous infusion of routine medicines and respiratory therapy during first 30 - 60 min. Hemodynamics was estimated by Dopplerechocardiography ("Ultramark-ATL" - USA.). It was carried out 24 hours cardiomonitoring. In the right part of the heart transveinsly was inserted multicontact electrode "USCI" (USA). In clinical group were included patients with stable, intact ventriculo-atrial conduction. There was realized short-term diagnostic VVI pacing with simultaneous registration of the atrial electrogrammes. During the short-term VVI pacing retrograde V-A conduction in these patients were detected in 100 % of cases at the frequency of ventricular pacing equal to 142±3,5 imp/min. Average duration of the VA interval was about 164±7,6 msec. Subsequently all patients were treated by rarefying paired ventricular pacing.

In experimental setting 10 narcotized mongrel dogs with open chest and the stable retrograde ventriculo-atrial conduction were examined. " The Rules of the use of experimental animals " and " The international principles of the Helsinki declaration of the humane manipulation with animals " were strictly protected.

After standard premedication and under local anesthesia by Novocainum it was isolated and cannulated peripheral vein. As introduction narcosis it was used Thiopental sodium in a doze of 10 mg/kg. After intubation and i.v. injection of relaxant the lung ventilation (CMV mode) was realized by the device "RO-6"(RF). It was applied Ether Anaesthesicus at a doze about 2 v/%. Respiratory volume was equal to 300-350 ml.

After the left thoracotomy was opened pericard. Pulmonary artery was separated from ascending aorta. On these vessels were implanted volume gauges of electromagnetic blood flowmeter. It was catheterized jugular,caval and pulmonary veins, left carotid and femoral arteries, aorta, left and right atriums and ventricles and pulmonary artery. Into the right part of the heart the multicontact electrode was inserted. Intracardiac pressure, blood flow, electrograms and ECG were registered on "Mingograph-82" (German) at speed of movement of a tape by 100 and 250 mm/sec. X-Ray contrast cinematography research was performed on the device "TUR-700"(German), supplied by the electronic converter and optical allocator. Speed of registration was equal 32 and 80 frame/sec.

The sequence of experimental researches was following: initially it was registered intracardiac electrogrames, primary parameters of hemodynamics and X-ray contrast investigation. It was also scanned the conduction system of the heart and the parameters of the retrograde ventriculo-atrial conduction was estimated. The supraventricular tachycardia and accompanying hemodynamic disturbances was treated conjointly by VVP pacing and i.v. infusion of the adrenoreactive medications. Subsequently, all parameters repeatedly were registered.

Statistics. Mean and standard deviation were reported. Statistical significance was defined as a p value of < 0.05. Student's paired and unpaired t tests were used as appropriate to compare data at sinus rhythm and VVP pacing.

Results and discussion.

Clinical part.

After installation of adequate parameters of VVP pacing that usually required some minutes at once came stable rarefying of the ventricular rate. The first electrical impulse in pair

was stimulant and formed the contractile act of the ventricles. The second electrical impulse in pair launched by pacemaker with the certain delay promoted to prolongation of the refractory period of the ventricles and infraatrial part of conduction system of the heart. Despite of the presence of the retrograde conductivity both of the electrical impulses were not distributed to atriums owing to what there came the retrograde dissociation - i.e. retrograde blockade of IV degree carrying functional character. For this reason atrial rate was adjusted by supraventricular

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Table 1. M±m. n = 7.

Parameters of hemodynamics	Data in initial state	Data in VVP	P Student
Middle arterial pressure (mm.Hg.)	56±7,2	89±4,2	<0,01
Cardiac index (litre/min/meter^2)	2,02±0,1	2,71±0,03	<0,001
Stroke index (ml/stroke/meter^2)	16,2±0,7	33,2±1,7	<0,001
Stroke work index (Gramme x meter/stroke/meter^2)	11,9±1,3	39,9±2,2	<0,001
Left ventricular end diastolic volume (cm^3)	165,4±7,4	174±5,4	>0,5
Left ventricular ejection fraction (%)	18,6±0,9	36,4±2,7	<0,001
Ventricular rhythm (bpm)	125,1±4,6	82,7±3,9	<0,001
Atrial rhythm (bpm)	125,1±4,6	126,4±2,8	>0,5
Systemic vascular resistance (Dyne x sec x cm-^{5})	1224±193	1402±93,9	>0,5

pacemaking structures and as a rule predominated over ventricular. At testing deactivating of VVP pacing instantaneously restored initial cardiotopic rhythm and disappeared ventriculo-atrial retrograde dissociation.

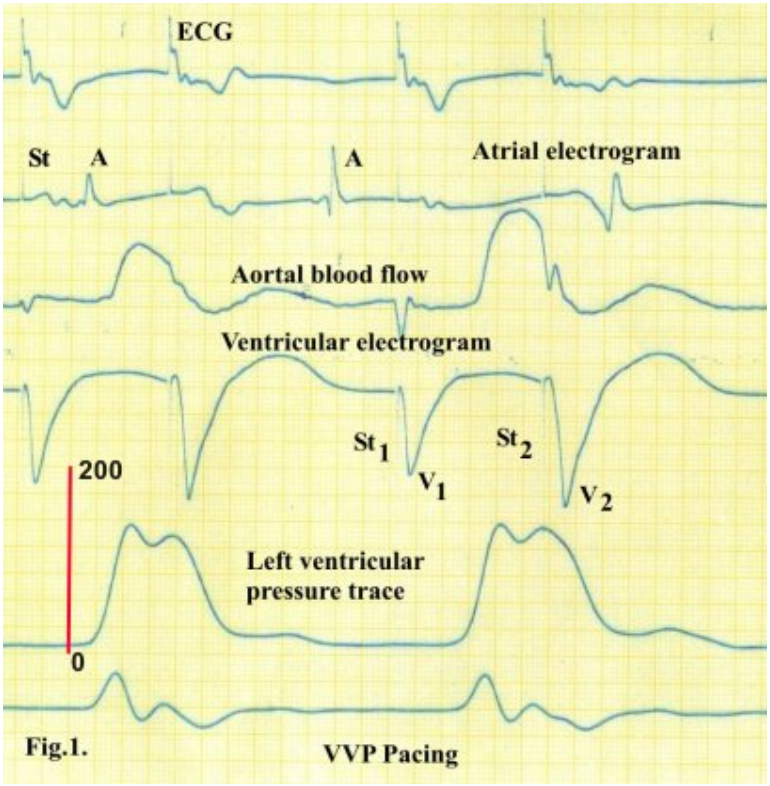
An important feature of the given mode of the ventricular pacing was that it formed such version of the retrograde dissociation at which the frequency of atrial contractions was above ventricular by 34 % ($P < 0.001$). In consequence of uncoordinated activities of atriums and ventricles various chambers of the heart were functioning in a mode return physiological. The process of formation of common cardiocycle had complex design: atrial and ventricular cycles were not electrically interconnected; the versions of the ventriculo-atrial junctions had carried functional, just hemodynamical character, because the structures of the diastolic loading and contraction of various chambers of the heart varied from a cycle to a cycle –there were observed formations of the unique, before unknown compensatory mechanisms ensuring sizeable upgrowth of the initially low stroke volume of the ventricles.

At the critical patients the average meaning of a rarefying of the ventricular rate was equal to 34 % ($P < 0,001$) that indicated to the significant growth of a diastolic filling period and end diastolic volume of the left ventricle. The stroke work index of the left ventricle had

increased more than twice (P < 0.001). Considerably raised left ventricular ejection fraction and stroke volume index. Despite of the rarefying of the ventricular rate more than 1/3 from initial level, cardiac output had increased by 34.1%, while mean arterial pressure raised by 59%(P<0.001. **Table 1**.). On the own data, at VVP pacing and presence fixed ventricular rhythm, gradual expansion of a doze of i.v. entered adrenoreactive medicals did not make for increase ventricular rate and formation of malignant arrhythmia.

Experimental settings

VVP pacing (**Fig.1**), accompanied by a delay of the second impulse equal to 227,7±4,5 msec that increasing refractory period of the myocardium of the ventricles, caused beginning of the retrograde ventriculo-atrial dissociation and reduction of the frequency of the ventricular



rhythm up to 107±3 bpm(P < 0,0001) that was equal to 33 % from a starting position. At that ventricles were contracted by the first stimulus generated by the device, while atriums were depolarized by Flack's node in consequence of atrial rhythm was by 32 % (P< 0,001) more ventricular. Such difference between atrial and ventricular rates meant, that on 1 left ventricular cycle came 1,47 atrial's, so some part of the atrial contractions took place at closed atrioventricular valves, and the other share was organized in a physiological sequence. For this reason the absolute number of the effective atrial systoles were equaled to 84,52±2,2 units from

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Table 2. M±m. n=10.

Parameters of hemodynamics	Sinus rhythm	VVP pacing	P
Left ventricular peak systolic pressure (mm.Hg.)	138±4,5	135±4,1	>0,5
Left ventricular end diastolic pressure (mm.Hg.)	3,6±0,17	7,2±0,23	<0,001
dp/dt_{max} of the left ventricle (mm.Hg./sec)	2341±145	2893±131	=0,02
dp/dt_{min} of the left ventricle (mm.Hg./sec)	1405±100	1737±122	>0,5
Stroke volume of the left ventricle (ml/stroke)	14,31±0,5	19,66±0,6	<0,001
Ventricular rate (beat/min)	160±3,15	107±3	<0,001
Cardiac output (litre/min)	2289,6±164	2103±131	>0,5
Mean aortal pressure (mm.Hg.)	103,5±5,1	85±3	<0,01
Peripheral vascular resistance (Dyne x sec x cm^{-5})	3724±150	3146±141	=0,02
Stroke work of the left ventricle (Gramme x meter/stroke)	26±0,56	34,6±0,74	<0,001
Minute work of the left ventricle (kGm/min)	3,23±0,4	2,42±0,21	=0,1
Left atrial reaction	Effective atrial contraction	Ineffective atrial contraction	---
Left atrial pressure at point $\mathbf{a_1}$ (mm.Hg.)	4,5±0,25	7,5±0,22	<0,001
Left atrial pressure at point $\mathbf{a_2}$ (mm.Hg.)	9,6±0,4	24,4±0,53	<0,001
Left atrial dp/dt_{max} (mm.Hg./sec.)	116,3±3,9	218±5,8	<0,001
Mean left atrial pressure (mm.Hg.)	6,3±0,3	11,9±0,48	<0,001
Duration of transmitral pressure gradient (msec)	111,1±5	221,1±8,1	<0,001
Number of the hemodynamically effective atrial contractions (Unit)	160±3,15	84,5±2,2	<0,001

average atrial rhythm and in the percentage relations had made 53,7 % against 100 % at sinus rhythm (P < 0,001). Thus, at VVP pacing approximately 22-23 complexes (about 21.5%) from 107 ventricular cycles were not served by hemodynamically effective atrial contractions.

Rarefying pacing promoted to an increase of the diastolic loading on the left ventricle that was proved by the sizeable rising of the end diastolic pressure (on 100%) and volume (for the asynchronous loading of the ventricles nice calculation of the average data were not statistically explanatory) of the left ventricle, as well as its contractile activity, stroke volume and stroke work on 23,5%, 37,3% и 33% accordingly in comparison with the sinus rhythm (P <0,01-0,001). The increase of the pulse pressure and reduction of the diastolic pressure in aorta was observed, owing to what middle aortal pressure in comparison with sinus rhythm had decreased

by 17,9 % (P < 0,01). Peripheral vascular resistance and minute work of the left ventricle diminished by 15,5 % and 25,1% accordingly (P < 0,001. **Table 2**).

In the presence of the intact valve and contractile apparatuses of the heart the increase of stroke volume in reply to VVP pacing for the most part depended on from a degree of rarefying of the ventricular rate, i.e. from extension of time and degree of the left ventricular diastolic filling - augmentation of volume loading on the left ventricle. Retrograde ventriculo-atrial dissociation had an insignificant role, in the certain sense even negative, as it activated intricate circulatory disturbances in venous segments of the pulmonary and systemic circuits. For example: at VVP pacing accompanied by ventriculo-atrial dissociation the depolarization of the left atrium drived by Flak's node had not extended to ventricles. So, the hemodynamical structure of the left atrial inflow and outflow varied from a cycle to a cycle. As at VVP pacing the frequency of the atrial contractions considerably prevailed above the ventricular the certain periodicity of the coincidence of atrial and ventricular cycles was observed: at that pattern of rarefying pacing the frequency of the hemodynamical coupling of the left atrial and ventricular cycles was on the average equaled to 2 to 3 (10 to 15, 20 to 30 & etc.). It meant, that after implementation 2 left ventricular and 3 serving them atrial cycles the periodicity of the hemodynamical splicing of the cycles repeated, however in two variants. At the first variant diastole of 2 left ventricular cycles were served by 2 hemodynamically effective and 1 ineffectual atrial contractions that occurred at closed mistral valve. At the second variant the inverse process was observed: 1 hemodinamically effective and 2 inefficient atrial complexes served diastole of 2 left ventricular cycles. The difference was in the following: left atrial systole, accomplished in accordance with "physiological sequence", acquired hemodynamical significance at the point of zero-initial transmitral pressure gradient. If the left atrial systole coincided with closed mistral valve it was hemodynamically inefficient and formed regurgitation to pulmonary veins. Left ventricular volume loading was realized by left atrioventricular diastolic pressure gradient, without participation of the left atrial systole. The structure of charging of the left ventricle in such coincided cardiocycles was multivendor. The mentioned process was more versatile in the right ventricle. The diversity of the hemodynamical mechanisms supervising process of the ventricular loading in many respects depended on a degree of rarefying of the ventricular rate, i.e. from a ratio of the duration of the left atrial and ventricular cycles –left atrial/ventricular cycles time index.

At the given pattern of the VVP pacing the number of the inefficient left atrial systoles, accomplished at closed mitral valve, was equal to 46,3 % of the total quantity of left atrial complexes and at numerical value was on 13,7 % (P<0.05) less in comparison with hemodynamically effective left atrial cycles.

At VVP pacing hemodynamics of the inefficient left atrial complexes considerably differed from those at sinus rhythm: significantly increased the level of the volume loading on the left atrium – left atrial diastolic pressure at VVP in comparison with sinus rhythm was elevated by 29,3 % (P<0,001). The gain of the left atrial diastolic loading was caused by contraction of the left atrium at closed mitral valve that in the given phase confirmed by presence of high and reverse left ventriculo-atrial pressure gradient about 67,2± 2,6 mm.Hg. The described shifts activated atrial contractility and increased left atrial systolic pressure in comparison with sinus rhythm by 71,8 % (P<0,001). For this period antegrade systolic transmitral blood flow was absent and high systolic left atrial pressure created regurgitation from left atrium to pulmonary veins due to shaping inverse left atrial - wedge pressure gradient about 6,7±0,2 mm.Hg. After completion of the left atrial systole the polarity of the pressure gradient inversed again and formation of the antegrade transmitral pressure gradient and opening of the mitral valve was observed and then the left ventricular filling period followed. Thus, left atrial systole did not participate in the process of the diastolic loading of the left ventricle.

The magnitude of the diastolic pressure, contractile activity, systolic and middle pressure of the hemodynamically effective left atrial complexes at VVP pacing were accordingly by 66,6%, 24,6%,47,9 % and 17,4 % greater than at sinus rhythm. Earlydiastolic and presystolic

transmitral pressure gradients were heightened, indicating on increase of the transmitral blood flow that promoted to a rise of the end diastolic volume of the left ventricle and its contractility. At rarefying of the ventricular rate significantly increased its filling period. Moreover: in comparison with sinus rhythm the VVP pacing resulted to appreciable increasing of the duration of the transmitral pressure gradient for 1 minute (normalized parameter) even in conditions of statistically significant decrease of the ventricular rate. In this connection stroke volume of the left ventricle at VVP pacing was on 37 % ($P < 0.001$) more that at a sinus rhythm.

Conclusions.

1.Rarefying ventricular pacing (VVP) to carry into the functional blocking of the intact retrograde conductivity.
2.At VVP pacing atrial rate always prevail on ventricular.

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To be continued.

პარკუჭების რიტმის იმპულსთა წყვილით გამაიშვიათებელი ელექტროსტიმულაციის (VVP) გავლენა კარდიოჰემოდინამიკაზე კრიტიკულ ავადმყოფებში.

რ.შონია.

კრიტიკული მედიცინის ინსტიტუტი. თბილისი.

კრიტიკულ ავადმყოფებში და ექსპერიმენტში შესწავლილია სუპრავენტრიკულური ტაქიკარდიის თანხლებით მიმდინარე მწვავე კარდიორესპირატორული უკმარისობისას რესპირატორული და რუტინული ანტიშოკური მედიკამენტური თერაპიის ფონზე განხორციელებული პარკუჭების რიტმის იმპულსთა წყვილით

გამაიშვიათებელი ელექტროსტიმულაციის (St_1 + St_2 დაყოვნებული იმპულსი; ალფავიტური კოდი-VVP) გავლენა კარდიოჰემოდინამიკაზე.

კვლევის პროცესში გამოიყენებოდა კარდიო- და მჟავა-ტუტოვანი ბალანსის მონიტორინგი, ხანგრძლივი რესპირატორული და პროგრამირებული ინფუზიური თერაპია, დოპლერექოკარდიოგრაფია, სხვადასხვა სახის ელექტროკარდიოსტიმულაციის, გულისა და მაგისტრალურ სისხლძარღვთა კათეტერიზაციის, ელექტრომაგნიტური ფლოუმეტრიისა და ანგიოკარდიოგრაფიის მეთოდები.

დადგინდა, რომ VVP ელექტროკარდიოსტიმულაცია ინტაქტური რეტროგრადული გამტარობის არსებობისას იწვევს ამ გზის და პარკუჭების მიოკარდის რეფრაქტერობის ზრდას და V_1-V_1>A-A ვერსიის ფუნქციური რეტროგრადული პარკუჭოვან-წინაგულოვანი დისოციაციის განვითარებას, პარკუჭების რიტმის მომენტალურ გაიშვიათებას, პარკუჭებზე მოცულობითი დატვირთვის, მათი დარტყმითი მოცულობისა და მუშაობის, აორტაში მოცულობითი ნაკადისა და წნევის საგრძნობ მატებას და აყალიბებს ჰემოდინამიკურად ეფექტურ და არაეფექტურ წინაგულოვან კომპლექსებს, რომლებიც ხასიათდებიან მეტად რთული ჰემოდინამიკური და ამავდროულად ადაპტაციური მექანიზმებით.

კრიტიკულ ავადმყოფებში გულ-სისხლძარღვთა მწვავე უკმარისობით VVP ელექტროკარდიოსტიმულაციის ჩატარებისას შესაძლებელია დადებითი ქრონო- და ინოტროპული ეფექტების მქონე ადრენორეაქტიული ჯგუფის პრეპარატების თერაპიული დოზების მნიშვნელოვანი გაზრდა, რაც მიოკარდის შეკუმშვადი ფუნქციის საგრძნობი აქტივაციის პარალელურად, პარკუჭების ფიქსირებული რითმის პირობებში არ იწვევს მათი შეკუმშვების სიხშირის მატებას.