

## **The influence of the ventricular tachycardia with retrograde dissociation of atriums on cardiohaemodynamics.**

**R.N.Shonia., G.S.Shonia.**

**(Critical Care Medicine Institute. Tbilisi. Georgia).**

In the critical patients and experimental condition upon narcotized mongrel dogs with opened chest the influence of the ventricular tachycardia with retrograde dissociation of atriums on cardiohaemodynamics 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 angiocardiology were performed. In this study it was proved that at ventricular tachycardia accompanied by retrograde dissociation the magnitudes of the basic parameters of intracardiac and central haemodynamics of the unphysiologically linked ventricular and atrial cycles are defined by the coincidence of phases of ventricular and atrial cycles. This phenomenon determine the hemodynamic significance of any version of the ventriculo-atrial complex that is modeling in inverse sequence and, in particular, defines conditions of functioning of atriums, process of the volume loading of ventricle and the value of its stroke volume.

**Key words:** ventricular tachycardia, retrograde dissociation, haemodynamics.

**Introduction.** The haemodynamics of emergency regimen working heart at disintegration its electrical system in the critical patients with terminal forms of arrhythmia is not investigated. The given wording equally concerns of ventricular tachycardia. Irrespectively of a functional state of retrograde conductivity, at activation of ectopic ventricular focus it is build up the electrical contour that in any case represents the variant of depolarization of ventricle and atrium in a pathological sequence. So, change of the succession of contraction of various chambers of the heart determine their functioning in the order, return physiological. The generation of the repetition versions of such ventricular and atrial complexes at ventricular tachycardia make for formation of severe infringement of intracardiac haemodynamics, especially - in a segment of system of haemocirculation with low pressure and resistance.

The functional state of the retrograde conduction in many respects determines the hemodynamical status of a ventriculo-atrial complex. At a present of the ventricular tachycardia accompanied by the retrograde ventriculo-atrial dissociation the basic characteristics of electrical and mechanical activity of “functionally linked” ventricular and preceding or following it atrial cycles in each given cardiocycle is “hemodynamically depending”. The “configuration” of such ventriculo-atrial complex is defined by the duration of a V-V and A-A intervals, so by the rhythm of the mentioned chambers. The given parameter creates the frequency of formation of the variant of a coincidence of phases of ventricular and atrial cycles and determines the hemodynamic significance of any version of the ventriculo-atrial complex that is modeling in pathologically sequence and, finally, defines conditions of functioning of atrium, process of a volume loading of ventricle and the magnitude of its stroke volume.

Arrhythmia at the critical patients is an unexplored area of the medicine. It is enough to note that in modern cardiology there is no information about the influence of an asynchronous contraction of ventricles and atriums on intracardiac and central haemodynamics.

The purpose of our investigation is the studying of importance of ventricular tachycardia accompanied by the retrograde ventriculo-atrial dissociation on formation of hemodynamic disturbances.

### **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 was generated persistent ventricular tachycardia with retrograde dissociation. It was carried out 24 hours cardiomonitoring. In the right part of the heart transveinsly was inserted multicontact electrode of "USCI". Haemodynamics was estimated by dopplerechocardiography ("Ultramark-ATL" - USA.).

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

After standard premedication and under local anaesthesia 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 (MVV mode) was realized by the device "RO-6"(RF). It was applied Ether Anaestheticus at a doze about 2 v/%. Respiratory volume was equal to 300 ml.

After the left thoracotomy was opened pericard. Pulmonary artery was separated from ascending aorta. On these vessels was 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 a 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 realization of experiment was following: initially it was registered intracardiac electrogrames, primary parameters of haemodynamics and X-ray contrast research. It was also scanned the conduction system of the heart. In the absence of intact retrograde conduction the ventricular tachycardia with retrograde ventriculo-atrial dissociation and accompanying to it hemodynamic disturbances by VVI pacing was modeling. Subsequently, all parameters repeatedly were registered.

Statistics. Mean and standard deviation are 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 ventricular tachycardia.

**Results and discussion.** In clinical conditions the occurrence of a ventricular tachycardia with ventriculo-atrial dissociation in comparison with sinus rhythm resulted to fragmentation of a single whole electrical field of the heart on two independent parts: ventricular and atrial. The separation of an electrical field was connected to presence of a IV degree blockade by retrograde pathway of conduction system of the heart. The frequency of atrial contraction was governed by pacemaking activity of a sinus node, was equaled to  $85 \pm 10,3$  bpm and was on 14 % more the same in comparison with sinus rhythm in control group. The frequency of the ventricular rhythm was formed by pacemaking activity of the ectopic ventricular nidus, was equaled to  $94,7 \pm 4,1$  bpm and exceeds a control parameter on 27 %. The frequency of the ventricular rhythm exceeds atrial on 11,4 %. (Table 1).

**Table 1.****The influence of the ventricular tachycardia with retrograde dissociation on cardiohaemodynamics in critical patients****M±m. n=7**

Parameters of haemodynamics	Data in sinus rhythm (control group)	Data in ventricular tachycardia (in critical patients)	P by Student
Left ventricular end diastolic volume (cm <sup>3</sup> )	118±2,9	191,1±4,5	<0,001
Ejection fraction of the left ventricle (%)	59,1±2	28,7±1,0	<0,001
Heart rate (ventricular) (bpm)	74,5±0,69	94,7±4,1	>0,05
Heart rate (atrial) (bpm)	74,5±0,9	85±10,3	<0,02
Mean arterial pressure (mm.Hg.)	84,6±1,2	67,8±1,7	<0,001
Peripheral vascular resistance (dyne x sec x cm <sup>-5</sup> )	1194±37	1065±54,2	<0,001
Cardiac index (litre/min/meter <sup>2</sup> )	3,23±0,04	2,76±0,1	=0,25
Stroke index (ml/stroke/meter <sup>2</sup> )	44,1±1,5	29,5±1,6	<0,001
Stroke work index (gramme x meter/stroke/meter <sup>2</sup> )	49,7±1,7	27,4±2,0	<0,001
Index of minute work of heart (kGm/min/meter <sup>2</sup> )	3,9±0,01	2,6±0,2	<0,05

The end diastolic volume of the left ventricle was equal to 191,1±4,5 cm<sup>3</sup>. It exceeded standard for normodynamic type of haemocirculation on 62 % (P < 0,001). The inotropic activity of the left ventricle had decreased : ejection fraction of the left ventricle was on 51,4 % (P < 0,001) below of standard. Indexes of stroke volume and stroke work of the left ventricle proportionally decreased about 33,1 % and 44,8% (P<0,001). Despite of moderate growth of the frequency of the ventricular beats, cardiac index and index of minute work of left ventricle were accordingly on 14,5 % and 33,4 % (P < 0,001) below the norm. The fall of arterial pressure under to critical meaning was observed that indicated to the evolution of a shock.

The treatment by antiarrhythmic drugs was utilized in all cases. Repeated attempt to invert ventricular rhythm in atrial by AAI pacing had a transient effect in 14,2% of cases. After cardioversion sinus rhythm temporarily was restored only at 28,5% of patients.

The experimental researches completely co-coordinated with the clinical data (Table 2.).

**Table 2.**

**The influence of ventricular tachycardia with retrograde dissociation on cardiohaemodynamics in narcotized dogs.**

**M±m. n=10.**

Parameters of haemodynamics	Data in sinus rhythm	Data in ventricular tachycardia	P by Student
Left ventricular peak systolic pressure (mm.Hg.)	152±5,1	114,5±1,6	<0,001
Left ventricular end diastolic pressure (mm.Hg.)	6,6±0,2	5,18±0,23	<0,01
dp/dt <sub>max</sub> of left ventricle (mm.Hg./sec)	2000±123	1542±90,8	<0,05
dp/dt <sub>min</sub> of left ventricle (mm.Hg./sec)	1900±108	1350±82	=0,01
Stroke volume of left ventricle (ml/stroke)	14,8±0,62	10,41±0,3	<0,001
heart rate (ventricular) (bpm)	166,6±2,1	200±3,18	<0,001
Cardiac output (litre/min)	2467±110	2083±86	<0,05
Mean aortal pressure (mm.Hg.)	112±3,4	89,7±2,8	<0,01
Peripheral vascular resistance (dyne x sec x cm <sup>-5</sup> )	3552±176	3629±164	>0,5
Stroke work of left ventricle (gramme x meter/stroke)	28,3±0,62	14,7±0,42	<0,001
Minute work of left ventricle (kGm/min)	3,82±0,31	2,57±0,24	<0,02
Pulmonary artery peak systolic pressure (mm.Hg.)	34,2±1,6	24,8±0,75	<0,01
Pulmonary artery end diastolic pressure (mm.Hg.)	18,3±0,54	19,4±0,71	>0,25
Mean pulmonary artery pressure (mm.Hg.)	23,6±0,89	21,1±0,76	>0,05
Pulmonary vascular resistance (dyne x sec x cm <sup>-5</sup> )	751±27	821±28	>0,1
Right ventricular peak systolic pressure (mm.Hg.)	36,4±1,4	26,5±1,2	<0,01
Right ventricular end diastolic pressure (mm.Hg.)	5,7±0,2	4,2±0,17	<0,002
dp/dt <sub>max</sub> of right ventricle (mm.Hg./sec)	520±22,8	360±17,4	<0,002
dp/dt <sub>min</sub> of right ventricle (mm.Hg./sec)	450±19,3	340±16,1	<0,01
Stroke work of right ventricle (gramme x meter/stroke)	6,1±0,37	3±0,24	<0,001
Minute work of right ventricle (kGm/min)	0,82±0,07	0,61±0,02	<0,05

In experimental setting by modeling of the ventricular tachycardia accompanied with ventriculo-atrial dissociation there were registered noticeable decreasing of inotropic activity, stroke and minute volume, stroke work and peak systolic pressure of the left ventricle and reduction of the average pressure in aorta. The directivity of the hemodynamical shifts of the basic parameters of circulatory dynamics in the right and left parts of the heart practically were identical.

The described hemodynamical alterations were explained by formation of complex of quantitative and qualitative changes in a various phases of the left ventricle, first of all in a diastole, resulted to displacement of the volume/pressure curve (V/P) of the left ventricular diastolic function to the left and downwards, i.e. to its volumetric underloading, from one's part caused by beginning of the retrograde dissociation, resulting to uncoordinated contractions of the ventricles and atriums and

significant shortening of a diastole phase of the left ventricle, determined by increasing of the ventricular rhythm.

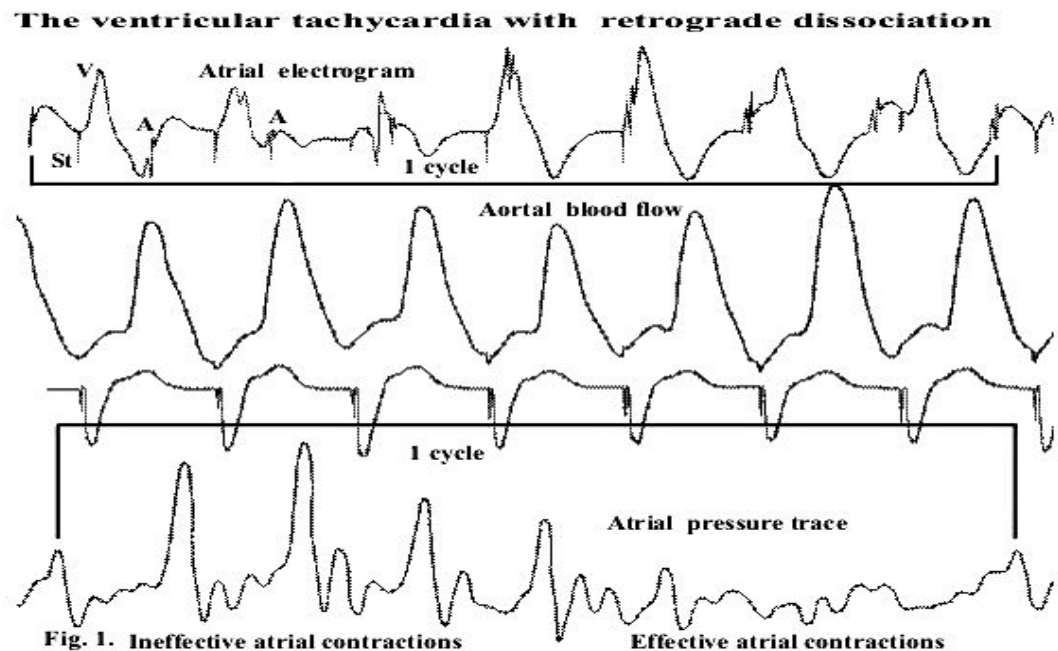
The ventricular rhythm with retrograde dissociation inverted a physiological sequence of depolarization and contraction of various chambers of heart. As a consequence of fragmentation of an electrical field of heart on two independent parts - atrial and ventricular, resulting to conventional complete asynchronous in their functioning, duration of ventricular cardiocycle by an average parameter was on 60 msec shorter than atrial ( $V_1 - V_1 < A - A$ ). In this connection quantity of hemodynamically effective atrial systoles were on 51,1 % ( $P < 0,001$ ) less in comparison with absolute number of ventricular contractions. Common cardiocycle was not always commenced by ventricular contraction and supplied by "personal" atrial systole. For this reasons the basic hemodynamical parameters, characterized levels of preloading on ventricles, varied from a cycle to a cycle, that radically influenced on inotropic state of ventricles and the magnitude of the stroke volume.

The functional state of retrograde conduction represented as a dominant determined hemodynamical status of a ventriculo-atrial complex. At the presence of the retrograde dissociation the position of atrial cycle in common cardiocycle does not appear as "ventriculo-depending". The configuration of the functionally linked ventricular and attended on it atrial cycles were defined by duration of their cardiocycles, i.e. - frequency of their rhythm.

The given formula entirely modeling the hemodynamical version of every ventriculo-atrial complexes and all subsequent unphysiological mechanics of myocardium - all spectrum of mechanisms of compensations directed to supplying ventricles by volume.

1. It was revealed, that atrium show well-defined "flexibility" and depending on of definite hemodynamical conditions changes the duration of an own cycle, inotropic activity, configuration of pressure trace, magnitude of intraatrial pressure and etc.

2. In spite of the fact that in the most cases sinus node generated impulses with an error within the limits of 30-60 msec., during the long record of intraatrial pressure traces the pieces with identical duration of their cycles were registered (practically it was one variation number line), in the course of which the order of coincidence of the phases of the atrial and ventricular cycles varied in the certain sequence, in particular, according to the law of an arithmetical series (Fig. 1).



It was determined, that certain distance of atrial contraction from a beginning of a ventricular systole was equaled to a difference of cycles  $A-A$  and  $V_1-V_1$ . In elementary mathematics such difference is denoted as a step of arithmetical progression. Any member of an arithmetic progression, i.e. distance of a beginning of atrial contraction from ventricular, is possible to calculate by the well-known formula:  $a_n = a_1 + d(n-1)$ . With the purpose of simplification of process of the analysis, for a zero point of readout we conditionally accepted those atrial and ventricular complexes, contraction of which were formed simultaneously. It was established, that at ventricular tachycardia accompanied by ventriculo-atrial dissociation was formed not asynchronism of atrial and ventricular contractions, but certain sequence of coincidence of phases of atrial and ventricular cycles controllable by a difference of cycles  $[(A - A) - (V_1 - V_1)]$ , i.e. by a step of arithmetical progression. The frequency of repeatability of coincidence of phases of atrial and ventricular cycles in the unit of time, i.e. the duration of a line of an arithmetical progression was supervised by the rhythm – by the general least common multiple number of the duration of atrial and ventricular cycles (Fig. 2).

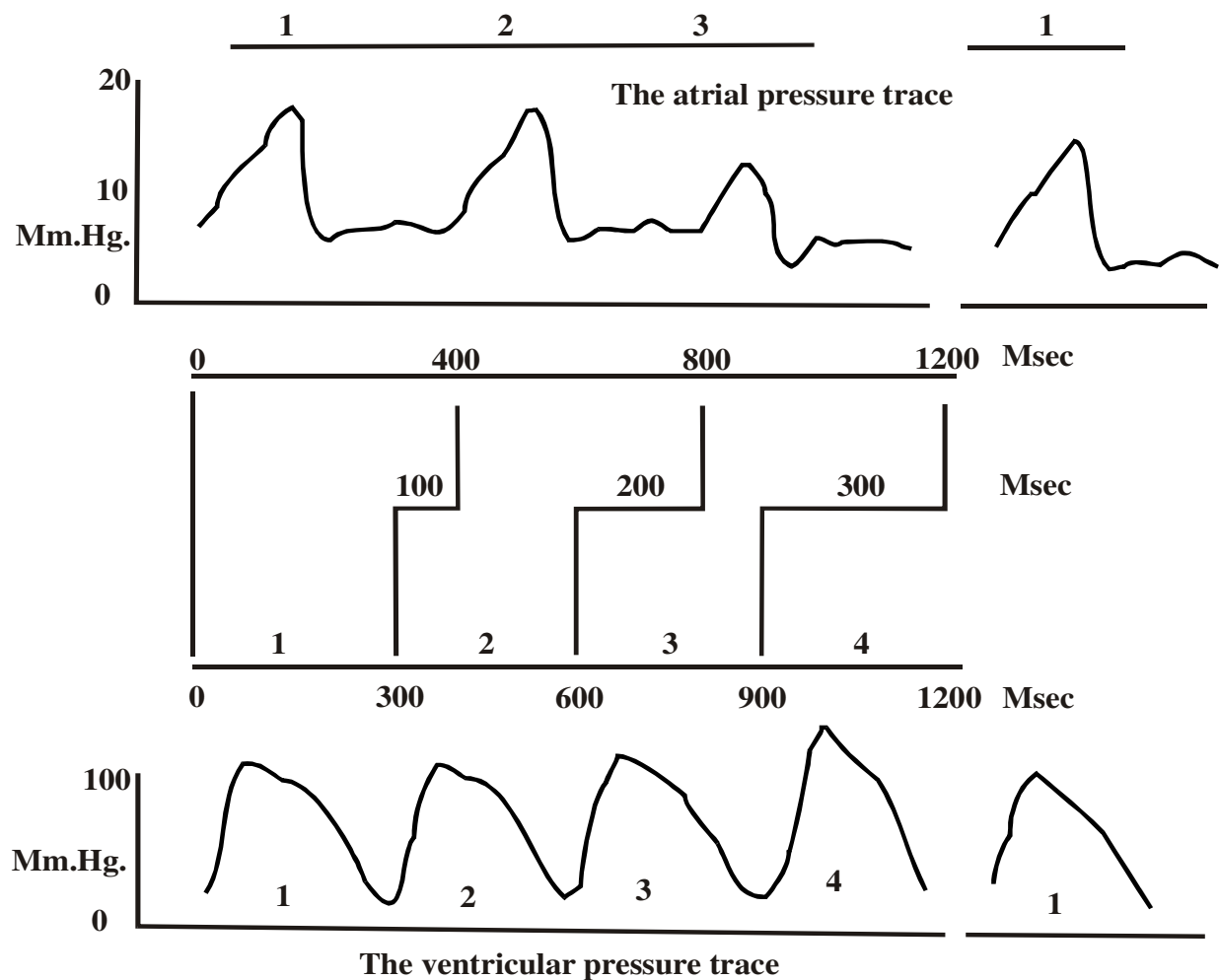


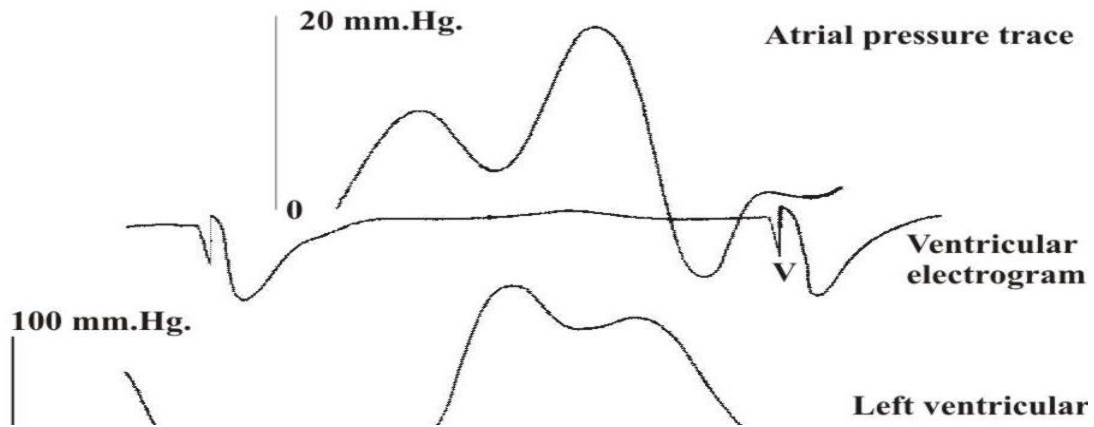
Fig.2.

The duration of the atrial cycle is equal to 400 msec, and ventricular - 300 msec; the step of an arithmetical progression is equal to 100 msec.; 1200 msec is a least common multiple number of the duration of an arithmetical progression. It consists of 3 atrial and 4 ventricular cycles; from fifth ventricular complex is formed the following on sequence cycle - atrial and ventricular contractions are formed simultaneously and simulate a configuration of first on sequence atrio-ventricular complex.

At ventricular tachycardia with retrograde dissociation due to "asynchronous" in contractions of ventricles and atriums, variants of a coincidence of the phases of their cycles were allocated as follows: atrial contraction took place during of a phase of isometric contraction, sphygmic interval, in early diastole or before the next systole of ventricle, i.e. in a physiological sequence. So, the notion of hemodynamically effective or noneffective atrial contractions at ventricular tachycardia accompanied by retrograde dissociation take on wider significance, as it is defined by the concrete version of coincidence of phases of atrial and ventricular cycles and indicates at the presence of close hemodynamical connection between atrial and ventricular cycles. This sort of "haemodynamically linked " ventriculo-atrial complex at ventricular tachycardia with retrograde dissociation is highly multifarious: the right and left parts of heart behave ambiguously as belong to the zone of high and low pressure.

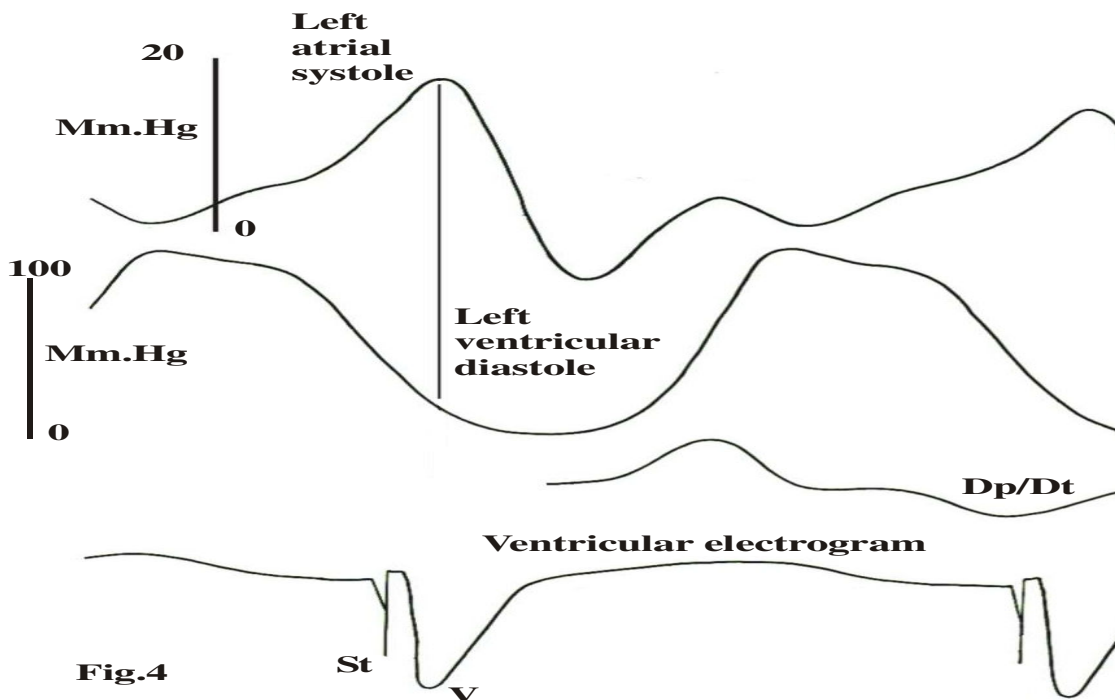
Despite of diversity, in absence of heart valvular disease and shock, using method of cycle-by-cycle analysis of superpositioned intracardiac pressure and blood flow traces at ventricular tachycardia with retrograde dissociation it was reconstructed main variants of ratio of atrial and ventricular cycles creating mechanisms of hemodynamical compensation at functioning of various chambers of heart in " an asynchronous mode " .

The first variant of a coincidence of atrial and ventricular cycles: atriums contract at closed atrioventricular valves and don't open them, as the amplitude of intraatrial systolic pressure is less than intraventricular. Such atrial contraction is hemodynamically ineffectual and independently of a duration of atrial and ventricular cycles basically accomplished from a beginning of the phase of isometric contraction before the phase of ventricular relaxation. Transmitral blood flow at the given period was absent as the contraction of the left atrium basically coincided with a phase of ejection of the left ventricle and had been made at a closed mitral valve. This fact was confirmed by presence of an inverse and lofty left ventriculo-atrial pressure gradient with the beginning of the left atrial systole. In addition to there was no "wash off symptom " at left ventriculograms in the area of the mitral valve, corroborated its opening synchronously with pathological left atrial contraction. At that had been formed a contrary and transitory left-atrial-pulmonary-vein pressure gradient that generated the reverse blood flow from left atrium to adjacent large-scale pulmonary veins. After the relaxation of the left ventricle the mentioned pathological pressure gradient became extinct and antegrade blood flow was formed repeatedly. Diastolic phases of the left ventricle and left atrium coincided. The transatrioventricular diastolic blood flow was formed at the expense of diastolic pressure gradient. The left ventricle basically loaded just at that precise period. It was underloaded, as presystolic pressure gradient was significantly less in comparison with control level (Fig. 3).



**Fig.3**

The second variant of a coincidence of atrial and ventricular cycles: the initial part of atrial systole is formed in a phase of isometric relaxation of ventricles - at closed atrioventricular valves. For this reason inotropic activity and the amplitude of systolic pressure in atriums sharply grow and promote early opening of atrioventricular valves at the expense of formation of high atrio-ventricular diastolic pressure gradient at a final stage of atrial contraction. There was legible “wash down symptom” at left ventriculograms in the area of the mitral valve, confirmed its opening synchronously with “semi-pathological and semi-effective” left atrial contraction. (Fig. 4).

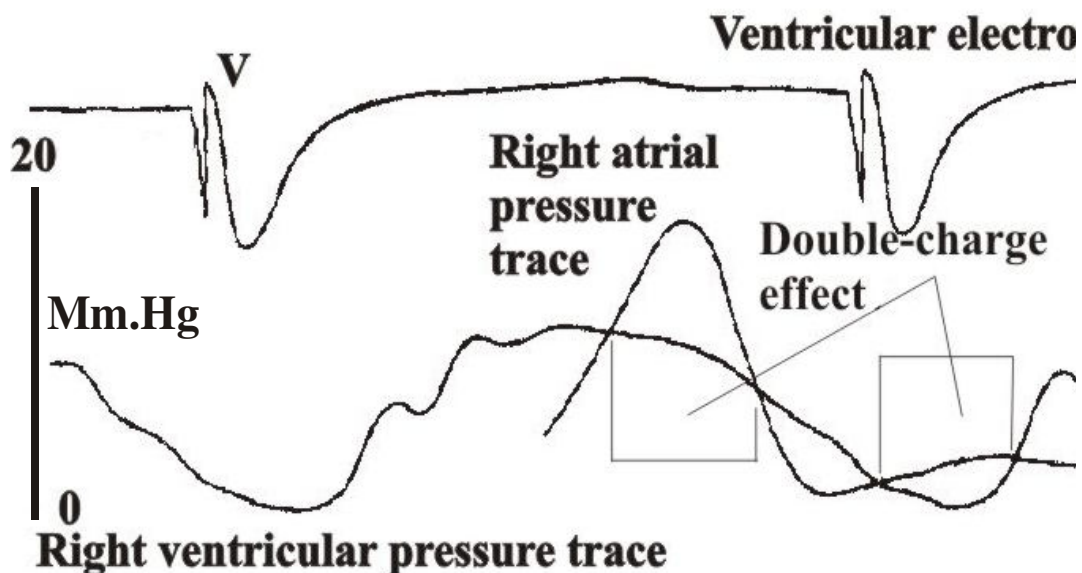


**Fig.4**



The third variant of a coincidence of atrial and ventricular cycles: the atrial systole is formed in late diastole, after opening of atrioventricular valves, loading the diastole of subsequent ventricular cycle and hemodynamically is identical of physiological atrial contraction.

The fourth variant of a coincidence of atrial and ventricular cycles: it was revealed solely with respect to right ventricle and concludes in double volume loading of ventricle during one cardiocycle. The hemodynamical mechanism was forming in the following way: right atrium was contracted in ejection phase of the right ventricle. Systolic pressure in the right atrium considerably exceeded above the peak systolic pressure in the right ventricle and forming pathological antegrade transtricuspidal blood flow. The given transitory pressure gradient was functioning during  $40 \pm 0,26$  msec. It was the first episode of volume loading of the right ventricle, however during its systolic phase - right ventricle increases the volume of the chamber in the course of the ejection phase. At that had been formed a contrary and transitory right-atrial-cava pressure gradient which generated the reverse blood flow from right atrium to adjacent large-scale cava veins. After the relaxation of the right ventricle the mentioned pathological pressure gradient became extinct. From the beginning of the relaxation of the right atrium the systolic atrial pressure decreased, transtricuspidal pressure gradient changed polarity and tricuspid valve closed at the early stage of right ventricle diastole. On account of early loading of right ventricle by volume the speed of its relaxation was much lower than right atrial. So, the polarity of transtricuspidal pressure gradient changed very slowly, the opening of tricuspid valve had been registered later than usual and abridged regular right ventricular diastole (at the average  $35,4 \pm 0,3$  msec) formed with the beginning of the next cycle (Fig. 5).



**Fig.5**

Thus, at ventricular tachycardia with retrograde ventriculo-atrial dissociation the state of intracardiac and central haemodynamics are defined by the duration of ventricular and atrial cycles and concrete version of coincidence of phases of their cycles. The basic reasons of development of heavy disturbances in the system of blood circulation are mismatching of depolarization and contraction of atriums and ventricles, resulting to reduction of absolute number of hemodynamically effective atrial contractions, inotropic activity, volume underloading and stroke output of ventricles. The specified factors designated as basic, checking the level of pre- and afterloading on myocardium and its performance efficiency.

**წინაგულების რეტროგრადული დისოციაციით მიმდინარე პარკუჭოვანიტაქიკარდიის გავლენა კარდიოჰემოდინამიკაზე რ.შონია, გ.შონია.  
(კრიტიკული მედიცინის ინსტიტუტი.თბილისი.საქართველო.)**

**რეზიუმე:**

ნაშრომში განხილულია გულის ინტაქტური სარქველოვანი აპარატის პირობებში რეტროგრადული დისოციაციით მიმდინარე პარკუჭოვანიტაქიკარდიის გავლენა კარდიოჰემოდინამიკაზე კრიტიკულ ავადმყოფებსა და ექსპერიმენტში. კვლევის პროცესში გამოიყენებოდა ელექტროკარდიოსტიმულაციის, გულისა და მაგისტრალური სისხლძარღვების კათეტერიზაციის, კონტრასტული რენტგენოკინემატოგრაფიისა და ელექტრომაგნიტური ფლოუმეტრიის მეთოდები. კვლევის შედეგებმა სრულად შესცვალეს დღემდე არსებული წარმოდგენები რეტროგრადული დისოციაციით მიმდინარე პარკუჭოვანიტაქიკარდიისას პარკუჭებისა და წინაგულების ე.წ. “ასინქრონული ფუნქციონირების” თაობაზე. დადგინდა, რომ რეტროგრადული დისოციაციისას წინაგულის ციკლის დისლოკაცია პარკუჭის ციკლთან მიმართებაში ანტიფიზიოლოგიურია: პარკუჭებისა და წინაგულების ციკლები აყალიბებენ ფუნქციური თვალსაზრისით არაერთგვაროვანი კონფიგურაციის საერთო კარდიოციკლს, რომლის ჰემოდინამიკური

სტრუქტურაც კონტროლირდება პარკუჭებისა და წინაგულების პეისმეიკერული აქტივობით და ხასიათდება არითმეტიკული პროგრესიის კანონით. რეტროგრადული დისოციაციისას ეს ფენომენი აყალიბებს ჰემოდინამიკური კომპენსაციის დღემდე უცნობ მექანიზმთა მთელ კასკადს, რომელიც განსაზღვრავს წინაგულთა ფუნქციონირების პირობებს, პარკუჭების დიასტოლურ ფუნქციას, მათ ინოტროპულ აქტივობას, დარტყმითი მოცულობის ამპლიტუდას და სისხლის მიმოქცევის პულმონალურ და კავალურ სეგმენტებში ჰიპერტენზიის ხარისხს.

### **References:**

1. B.N.Goldreyer, J.T Bigger. Ventriculo-atrial conduction in man// Circulation. 1970.-V.41.-n.6.-P.935-946.