Cardiohemodynamics at bi-directional A-V & V-A blockade

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At the bi-directional A-V and V-A blockade of fourth degree accompanied with various frequency of atrial and ventricular rhythm there were disclosed several sorts of transient cohesion of atrial and ventricular cycles that determined quantitative characteristics of hemodynamic loading on myocardium, its contractility and systolic output. The sizeable change (de- or increase) of ventricular rate in comparison with atrial rhythm promotes to reduction of the absolute number of hemodynamically effective atrial systoles and prodigal pattern of the heart functioning. At the equality of durations of atrial and ventricular cycles absolute quantity of hemodynamically effective atrial systoles were maximum and were defined by duration, polarity, amplitude of ante- and retrograde trans-atrio-ventricular pressure gradient, velocity and volume of re- and out-flow. The exploration of dynamics of loading and ejection of the atriums and ventricles at bi-directional dissociation reveals the mechanism of various patho-phono-phenomena upon atrioventricular valves detected at auscultation and phonocardiography.

Key words: cardiohemodynamics, bi-directional A-V - V-A blockade, dissociation, ventricular rhythm, effective atrial contraction, inefficient atrial contraction, cardiac pacing, epicardial phonocardiography, critical care medicine.

Actuality

At critical conditions there is no information about the action of the heart conduction system disturbances on circulation system. The cardiohemodynamics at the bi-directional A-V – V-A blockade of fourth degree is not known.

The purpose

The gole of the investigation was to study the cardiohemodynamics at the bi-directional A-V – V-A blockade of fourth degree accompanied with various frequencies of the atrial and ventricular rhythm.

Material and methods

Clinical investigation

“The Rules of the Rights of the Patients " were always defended . There were surveyed 11 critical patients with bi-directional A-V - V-A blockade of fourth degree accompanied by slow idioventricular rhythm before and after cardiac pacing. "USCI" multicontact electrode-catheter in the right part of the heart transveinsly was inserted and VVI pacing was performed. It was carried out 24 hours respiratory ventilation, ECG, hemodynamic (echocardiography), acid-base balance and other systems monitoring (USP).

Experimental investigation

There were strictly protected "The Rules of use of experimental animals " and " The international principles of the Helsinki declaration of the humane manipulation with animals" and examined 10 narcotized open chest mongrel dogs. After standard premedication and under local anesthesia by Novocainum it was isolated and cannulated peripheral vein. There was used introduction narcosis by Thiopental sodium in a doze of 10 mg/kg. After trachea intubation and i.v. injection of relaxant the lung ventilation (CMV mode) was realized. The tidal volume was equal to 350 ml. It was applied Ether Anaesthesicus at a doze about 2 v/%. After the left thoracotomy was opened pericard and the

ascending aorta was separated from the pulmonary artery. On these vessels volume gauges of the electromagnetic blood flowmeter were implanted. All main vessels and heart chambers were catheterized and supplied with local electrodes. Intracardiac pressure, blood flow, electrogrames and ECG curves were registered on "Mingograph-82" at high-speed movement of a tape. X-ray TV examination was performed on the device "TUR-700; speed of registration was equal 32 - 80 frame/sec (Germany). The sequence of realization of experiment was following: there were initially registered intracardiac electrogrames, primary parameters of hemodynamics and X-ray contrast cinematography of the ventricles, atriums and main vessels. Then the conduction system of the heart was scanned and by the transvein electrodestruction of the A-V junction the bi-directional A-V – V-A blockade of fourth degree was created. There was used the method of local hypothermia of the sinus node (1) and the atrial rhythm was fixed at 120 bpm. There was also used VVI pacing at a frequency 180,120 and 60 imp/min and three frequency range of the atrial and ventricular rhythm ratio were composed: 1/ A-A > V_1- V_1 , 2/ A-A = V_1- V_1, 3/ A-A < V_1- V_1 . Subsequently all hemodynamic parameters were registered repeatedly.

Statistics

Mean and standard deviation were reported. Statistical significance was defined as a *p* value of < 0,05. Student's ***t*** tests were used as appropriate to compare data at sinus rhythm and bi-directional A-V – V-A blockade.

Results and discussion

1. Clinical part.

The bi-directional A-V – V-A blockade of fourth degree in most cases developed at a final stage of a critical conditions. The blockade separated united electrical field of the heart by two independent parts that produced steady electrical and mechanical dissociation of atriums and ventricles and were passed by slow idioventricular rhythm and sinus tachycardia about 37,45±2,2 bpm and 111,9±4,2 bpm accordingly. So, there was registered A-A < V- V pattern at that the frequency of atrial contractions outran ventricular on 199 % (P<0,001).

The cardiohemodynamics at bi-directional A-V – V-A blockade with idioventricular rhythm in critical patients

M±m. n=11. Table #1

Hemodynamic parameters	Data at sinus rhythm	Data at bi-directional block	P by Student
End diastolic volume index of the left ventricle (cm^3/m^2)	67,42±1,8	107,26±1,3	<0,001
Ejection fraction of the left ventricle (%)	59,1±2	33±1,42	<0,001
Frequency of ventricular contraction (bpm)	74,5±0,9	37,45±2,2	<0,001
Frequency of atrial contraction (bpm)	74,5±0,9	111,9±4,2	<0,001
Mean arterial pressure (mm.Hg.)	84,6±1,2	72,18±2,37	<0,001
Peripheral vascular resistance (dyne x sec x cm^{-5})	1194±37	2492±215	<0,001
Cardiac Index ($L/min/m^2$)	3,23±0,04	1,32±0,12	<0,001
Stroke volume index ($cm^3/Str/m^2$)	44,1±1,5	35,4±1,4	<0,002
Stroke work index (gr x meter/Str/m^2)	49,7±1,7	34,9±2,26	<0,001

It means that 1/ one ventricular cycle includes three atrial complexes, i.e. 1600 msec in length one ventricular cycle are served by 533,33 msec duration three atrial complexes, i.e. after every 533,33 msec it is formed hemodynamically effective or inefficient atrial systole. Hemodynamic state of this kind atrial and ventricular complexes varies from a cycle to a cycle and in left part of the heart differs from the right, i.e. they are not hemodynamically identical in the high and low pressure basin; 2/ atrial and ventricular cycles simultaneously or with a certain delay generated common cardiocomplexes. In most clinical cases sinus node produced impulses with an error within the limits of 50-120 msec and atriums showed well-defined hemodynamic "flexibility" : they changed the duration of an own cycle, inotropic activity, configuration of pressure trace, magnitude of intraatrial pressure and etc. After formation of the definite number of polymorphic cardiocycles they finished one circle and began new line of the hemodynamically effective and inefficient atrial cycles which in a variation number line were distributed by certain frequency and had circulating character; 3/ the asynchronity and difference in frequency of a rhythm diversely influenced on myocardial rhythmo-diastolic and rhythmo-inotropic dependences; 4/the dissociation of atriums and ventricles formed a large number of compensating mechanisms that have multiform hemodynamics.

The central hemodynamic changes at the A-A < V- V pattern basically were unidirectional (table 1.) : systemic pressure, cardiac index and left ventricle ejection fraction were located on critical low numbers. At an acceptable level of the left ventricle volume loading and heavy damage of the heart contractile system VVI pacing and following to it normalization of the ventricular rate did not promote to a gain of the heart performance - indexes of the left ventricle stroke volume and stroke work were much lower of standard.

2. Experimental part.

After modeling of the bi-directional dissociation there were determined three groups: I pattern - atrial rhythm was less than ventricular - A-A > V_1 - V_1 - (120/180 bpm); II pattern - atrial rhythm was equal to ventricular - A-A = V_1 - V_1 - (120/120 bpm); III pattern - atrial rhythm was greater than ventricular - A -A < V_1 - V_1 - (120/60 bpm).

The cardiohemodynamics at I, II and III patterns were different, especially in details (table 2). The greatest level of the cardiac output (at intact contractile system!) in comparison with an initial value was observed after significant increase of the ventricular rate - A-A > V_1 - V_1 . At the II pattern (A-A = V-V) there were registered the most favourable conditions of circulation and greatest number of hemodynamically effective atrial contractions, equaling to 67,4 % ($P < 0.001$).

As a structure unit the hemodynamically effective atrial contraction is absolutely uncertain concept. At the bi-directional blockade atrial contraction may take place during of a presystole of the ventricle, phase of its isometric contraction, sphygmic interval or in early diastole and again at presystole of the next ventricular cycle. Therefore, the hemodynamic conditions of atrial and ventricular working are defined on the basis of the durations and differences in length of their cycles.

Just now there were detected hemodynamically (1) completely or (2) partially effective and (3) completely inefficient (4) right and (5) left atrial contractions, take place relatively to (6) previous or (7) subsequent ventricular (8)systole or (9) diastole phases (the order of priorities of the atrial contractions have a hemodynamic meaning!), in sum about 9 categories now, which have different timing, barographic and volume characteristics. At the intact valvular and septal heart systems the hemodynamic value of mentioned atrial complexes basically are defined by the (1) duration, (2) polarity, (3) amplitude of ante- or retrograde trans-atrio-ventricular pressure gradients and (4) volume of the displaced blood. The common cardiocycle principally is formed by the concrete version of coincidence of various phases of cycles of atriums and ventricles; and then, based on self-regulation, the quantitative characteristics of the given hemodynamic structure finally are formed on hand available volume/pressure and other hemodynamic dependences. At atrial septal defect with

mono- or bi-directional shunts, mitral valve insufficiency and other atelocardia their hemodynamic characteristics are quite different. So, the existing data (2) should be reconsidered.

At dissociation we reduce the uncoordinated atrial functioning to a common denominator. In this asymmetric endless loop the symmetry was found out. At any heart rate the different hemodynamical versions of the atrio-ventricular cycles ratio were registered. In the course of this variation number line the sequence of coincidences of phases of atrial and ventricular cycles were varied in accordance with a law of an arithmetical series. The distance of atrial contraction from a ventricular systole was equaled to difference of the cycles A-A - V_1- V_1, so to a step of the arithmetical progression and was calculated as **an = a1 + d (n-1).** The duration of the line of the arithmetical progression, so the numbers of hemodynamically effective or inefficient atrial contractions in the unit of time, so the frequency of

The cardiohemodynamics at bi-directional A-V - V-A blockade in narcotized dogs M±m. N=10 **Table #2**

Parameters of hemodynamics	Data at A-A>V-V Version I	Data at A-A = V-V Version II	P_1 (2-3)	Data at A-A<V-V Version III	P_2 (2-5)	P_3 (3-5)
1	2	3	4	5	6	7
Left ventricular peak systolic pressure (mm.Hg.)	149,5±3,1	130,2 ±2,9	<0,01	116,4±2,6	<0,001	<0,02
Left ventricular end diastolic pressure (mm.Hg.)	3,8±0,12	4,5±0,2	<0,05	5,5±0,17	<0,001	<0,02
dp/dt_{max} of left ventricle (mm.Hg./sec)	2352±96	2593±108	>0,25	1812±81	<0,01	<0,002
Stroke volume of left ventricle (ml/stroke)	15,29±0,31	18,54±0,4	<0,001	21,31±0,54	<0,001	<0,01
Stroke work of left ventricle (gramme x meter/stroke)	30,1±0,72	31,8±0,96	>0,25	32,1±1	>0,25	>0,5
Left ventricular end diastolic volume (cm^3)	32,3±0,9	36,7±1,1	<0,02	41,8±1,5	<0,02	<0,05
Ejection fraction of left ventricle	0,49±0,02	0,52±0,024	>0,5	0,52±0,023	>0,5	>0,5
Frequency of contractions of ventricles (beat/min)	180±0,08	120±0,1	<0,001	60±0,09	<0,001	<0,001
Hemodynamically effective atrial contractions (%)	47,9±0,9	67,4±1,2	<0,001	57±1	<0,001	<0,001
Cardiac output (litre/min)	2752±112	2224±96	<0,02	1278±74	<0,001	<0,001
Mean aortal pressure (mm.Hg.)	83,6±3,2	82,2±3,6	>0,5	67,8±2,8	<0,02	<0,02
Peripheral vascular resistance (dyne x sec x cm^{-5})	2432±103	2954±127	<0,02	4251±174	<0,001	<0,001

repeatability of coincidence of phases of atrial and ventricular cycles were controlled by the general least common multiple number of the durations of atrial and ventricular cycles (Fig.1.).

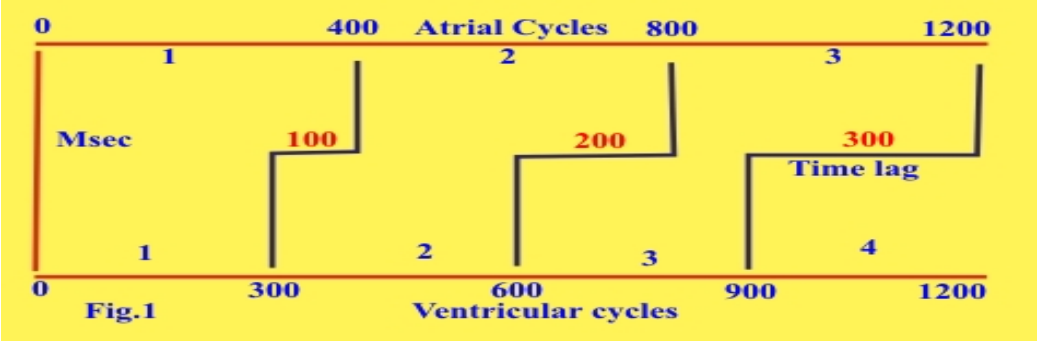
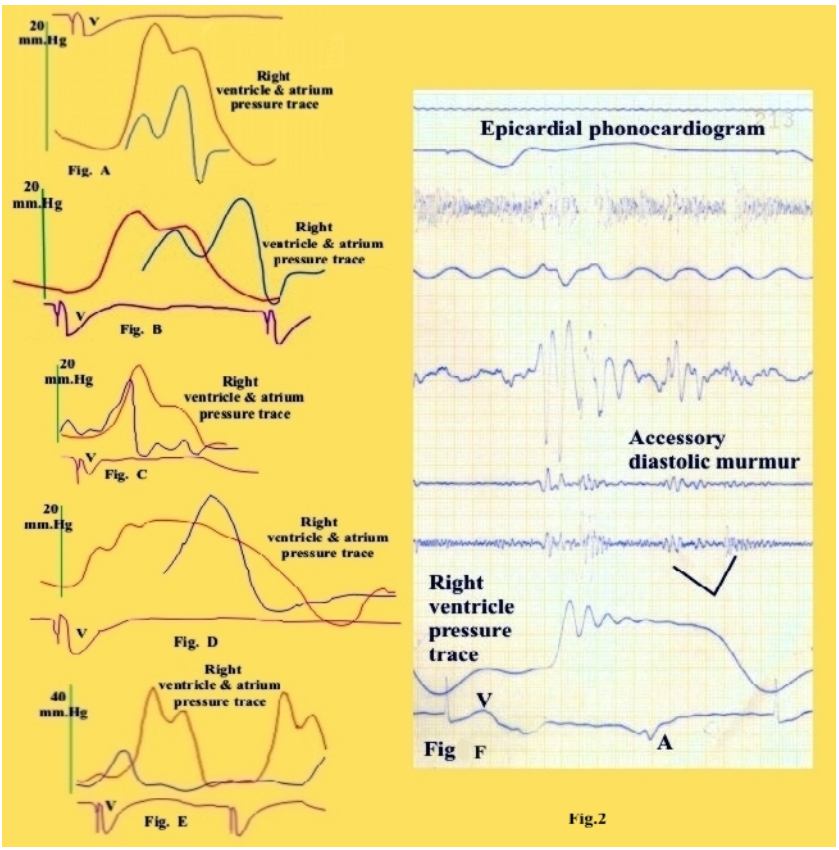


Fig.1

At the given stage of a study we defined five hemodynamic versions of the atrio-ventricular cycles ratio (Fig.2) those are in accordance with established procedure constructed and disappeared bio-tools: “has taken, used and has put back”. At the 1 version of the atrio-ventricular cycles ratio the left and right atrial systoles are formed from a beginning of a phase of an isometric contraction practically up to a protodiastole. The atrium cotracts at closed atrio-ventricular valve and can't open it, as the amplitude of intraatrial systolic pressure is less than intraventricular. Such atrial contraction is hemodynamically inefficient independently of a durations of atrial and ventricular cycles. At the given period “active” volume loading of the ventricle is absent. After the myocardial relaxation the antegrade diastolic blood flow charges ventricle, but it is underloaded , as the duration and magnitude of the perfusion pressure is significally less in comparison with control level (Fig. 2 A).



At the 2 version of the atrio-ventricular cycles ratio atrial contraction is formed after protodiastole and promotes to early formation of the antegrade atrio-ventricular pressure gradient. High atrial systolic pressure breaks into the ventricular diastole and at once considerably changes the left ventricular diastolic X-ray geometry. Such right atrial systole is hemodynamically completely effective, but the left atrial contraction is partially effective. Their final classification need in additional processing of a material (Fig. 2 B).

At the 3 version of the atrio-ventricular cycles ratio the atrial systole is formed in late diastole, after opening of the atrio-ventricular valves, more or less sufficiently charges subsequent ventricular cycle and maybe hemodynamically completely or

The hemodynamics of the various atrio-ventricular cycles ratio in narcotized dogs
M±m . n =250. Table #3.

Parameters of hemo-dynamics Version of ratio of atrial and ventricular cycles	Atrial pressure at a1 point (mm.Hg)	Atrial pressure at a2 point (mm.Hg)	$d_p/d_{t\ max}$ of the left atrium (mm.Hg./ sec.)	Transmitral pressure gradient at early diastole (mm.Hg.).	Pressure gradient at presystole (mm.Hg.).	Left ventricular end diastolic pressure (mm.Hg)	$d_p/d_{t\ max}$ of the left ventricle (mm.Hg./ sec)	Stroke volume of the left ventricle (ml/stroke)
1	2	3	4	5	6	7	8	9
Ratio #1	**2,4±0,2**	**13,3±0,3**	**182±3,9**	**1,8±0,12**	**0,5±0,02**	**3±0,22**	**1050±74**	**7,4±0,23**
Ratio #2	**6,2±0,2**	**20,4±0,4**	**264±4,1**	**19,6±0,33**	**1±0,04**	**5,9±0,3**	**1875±86**	**13,2±0,3**
P_1 (1-2)	<0,001	<0,001	<0,001	<0,001	<0,001	<0,001	<0,001	<0,001
Ratio #3	**1,9±0,11**	**10,5±0,2**	**76±2,5**	**3,8±0,2**	**3,6±0,27**	**6,7±0,36**	**1954±91**	**14±0,36**
P_2 (1-3)	<0,05	<0,001	<0,001	<0,001	<0,001	<0,001	<0,001	<0,001
P_3 (2-3)	<0,001	<0,001	<0,001	<0,001	<0,001	>0,1	>0,5	>0,1
Ratio #4	**7,3±0,3**	**26,3±0,5**	**282,5±5**	**1,6±0,1**	**10,7±0,4**	**7,1±0,34**	**2123±108**	**14,5±0,32**
P_4 (1-4)	<0,001	<0,001	<0,001	>0,25	<0,001	<0,001	<0,001	<0,001
P_5 (2-4)	<0,02	<0,001	<0,02	<0,001	<0,001	<0,02	>0,1	<0,01
P_6 (3-4)	<0,001	<0,001	<0,001	<0,001	<0,001	>0,5	>0,25	>0,25
Ratio #5	**Hasn't**	**Hasn't**	**Hasn't**	**0,3±0,02**	**0,1±0,01**	**0,2±0,04**	**745±66**	**6,1±0,15**
P_7 (1-5)	-	-	-	<0,001	<0,001	<0,001	<0,01	<0,001
P_8 (2-5)	-	-	-	<0,001	<0,001	<0,001	<0,001	<0,001
P_9 (3-5)	-	-	-	<0,001	<0,001	<0,001	<0,001	<0,001
P_{10} (4-5)	-	-	-	<0,001	<0,001	<0,001	<0,001	<0,001

partially effective. In a case of displacement such physiological atrial systole to the right (to the next ventricular cycle) for 80 – 120 msec it easily will turn into the hemodynamically inefficient atrial contraction (Fig. 2 C.). At the given version of the atrio-ventricular cycles ratio "multistage atrial contraction phenomenon" is observed: on an ascending branch of the atrial systolic pressure trace the saw-teeth sweeps and about 20-30 msec durations plateaux are formed that indicate on unreadiness of the atrial contractile system, so finally the atrium takes clear-cut decision while running enlarges own ejection period. It is one of the compensating hemodynamic mechanism, it's a lot of them and at various cardiopathology they should gradually be opened. At asynchronous atrial hemodynamic indexes always are changeable. So, (1) at the presence of series of variable hemodynamic deformations the atrium always reproduces variable answer as the adaptive-tuning system and (2) in running order optimizes electrical and mechanical reply. The reproducibility of the myocardial tissue response is an intricate problem and it must not be ruled out that not only at asynchronous the atrium is able to "record a history of experience".

At the 4 version of the atrio-ventricular cycles ratio atrial and ventricular systoles are formed practically simultaneously . Such atrial contraction due to high systolic pressure additionally charges the ventricle during ejection phase and at once substantially changes ventricular systolic X-ray geometry (the heart geometry research at the asynchronous in many respects will change existing opinion of the ventricle's remodeling.). It considerably violates the process of ventricular contraction, but hemodynamically is effective (Fig.2 D.) Such serial complexes are easily modeling by V-A/VO plus inverse V-A delay overdrive pacing. At this pattern of operations the myocard quickly overworks itself: we've data of histochemistry and electron microscopy intravital experimental examinations,

which demonstrate the fact of an acute damage of the intact contractil system. Such anomalous atrial contractions also induce variable diastolic or systolic murmurs over atrio-ventricular valves that require further analysis (Fig.2 F). At asynchronous there were registered multiform transient cardiac murmurs over the mitral or tricuspid valves. They were formed at any point of the common cardiocycle - during systole or diastole,etc and changed dislocation, magnitude, duration and intensivity of the temporary murmur from cycle to cycle-it changed while running. As a rule, audiologic characteristics of such provisional cardiac murmurs at bi-directional A-V & V-A blockade fully were depended on concrete version of a ratio of atrial and ventricular cycles and were defined by duration, polarity, amplitude of ante- and retrograde trans-atrio-ventricular pressure gradients and velocity and volume of flow.

At the 5 version of the atrio-ventricular cycles ratio one atrial cycle serves two ventricular complexes. If the atrial cycle duration more than twice surpasses in ventricular, its systolic phase can take place in a physiological order and adequately loads 1-st on sequence ventricular diastole. As the phases of the given atrial and subsequent ventricular cycles completely coincide, the end diastolic and stroke volume of the second ventricular cycle are smaller in comparison with first ventricular cycle. (Fig.2.E). And vice versa: if two atrial cycles serve one ventricular diastolic phase the first on sequence atrial systole also has the greater hemodynamic meaning.

The comparative analysis has revealed, that at the 2, 3 and 4 versions of the atrio-ventricular cycles ratio the hemodynamic indexes of the volume loading, inotropic activity and systolic output are significally greater in comparison with 1 and 5 versions, i.e., the hemodynamically inefficient atrial systole up to 30 % reduces stroke volume (table 3.).

Conclusion

At the bi-directional A-V and V-A blockade accompanied with any frequency of the atrial and ventricular rhythm there are revealed different hemodynamic versions of the atrio-ventricular cycles ratio, which define the levels of the pre- and afterload on myocardium, its inotropy and the magnitude of the stroke volume.

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კარდიოჰემოდინამიკა ავე-რეტროგრადული დისოციაციისას.

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სხვადასხვა სიხშირით მიმდინარე ავე-რეტროგრადული დისოციაციისას ფორმდება წინაგულების და პარკუჭების ციკლთა თანხვედრის მრავალი ვერსია, რაც განსაზღვრავს მიოკარდზე ჰემოდინამიური დატვირთვის დონეს, ინოტროპულობასა და მწარმოებულობას. ამ პათოლოგიისას ჰემოდინამიკურად ეფექტური წინაგულოვანი შეკუმშვების მაქსიმალური რაოდენობა ყალიბდება მხოლოდ იმ შემთხვევაში, თუ წინაგულებისა და პარკუჭების ციკლთა ხანგრძლივობა თანაბარია. შესწავლილია მრავალი კომპენსაციური მექანიზმი მიოკარდის პათოლოგიურ რეჟიმში მუშაობისას. ისინი ნათელს ჰფენენ პათოლოგიური კლინიკური სიმპტომების აღმოცენებას, რომლებიც ვითარდება ხსენებული ელექტრული მოშლილობის მქონე ავადმყოფებში.