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Features and causes of cardiac arrhythmias in patients with decompensated chronic cor pulmonale and coexistent arterial hypertension [Nazipa E. Aidargalieva, Assel Zh.Teleusheva]

Key words*:Chronic cor pulmonale, arterial hypertension, arrhythmia, premature complexes.*

Annotation: *To study the nature and characteristics of cardiac arrhythmias in patients with decompensated chronic cor pulmonale (dCCP) and coexistent arterial hypertension (AH) were examined 132 patients. It is revealed that the combination dCCP with AH increases the amount of high-grade extrasystoles both ventricular and supraventricular, resulted by deterioration of left ventricular systolic function, increase of LV myocardial mass, as a consequence of increasing the load incident on the target organs in hypertension. In addition, increase of blood pressure variability at night in these patients is also one of the factors that increase the number of ventricular arrhythmias.*

Actuality:

One of the reasons that may underlie in the development of sudden death in patients with chronic cor pulmonale is a disorder of heart rate. Recently, more and more data is accumulated confirming that cardiac arrhythmias are often accompanied by COPD, and the frequency of life-threatening arrhythmias in these patients approaches that of patients with acute myocardial infarction [1]. Under CCP it is observed various rhythm and conduction disturbances. Main pathogenetic factors leading to arrhythmias and blockades are hypoxemia, causing degenerative changes in the myocardium, a toxic effect on the myocardium, due to chronic inflammation of the bronchopulmonary tree and increase blood viscosity (polycythemia), leading to increased pressure on the heart and pulmonary hypertension [2]. Addition of CCP to hypertension undoubtedly worsens the prognosis of life in these patients.

In the literature there are a lot of data about the nature of rhythm disturbances in patients with bronchopulmonary diseases, particularly COPD and CCP [1-5]. However, the effect of hypertension on the nature of arrhythmias in patients with decompensated CCP has not been completely studied.

The aim of the work was to study the nature, characteristics of rhythm disturbances in patients with CCP combined with arterial hypertension 1 and 2 degrees.

Materials and Methods:

The study involved 132 patients with CCP and in combination with arterial hypertension. Study group comprised 72 patients with dCCP combined with AH, which, depending on the degree of arterial hypertension were divided into 2 groups: group 1 was consisted of 36 patients dCCP associated with AH 1 degree (dCCPAH1), whose average age was 65,1 ± 1.76 years, among them there were 20 men (55.5 %), women 16 persons (44.5%), group 2 was consisted of 36 patients dCCP associated with AH 2 degrees (dCCPAH), whose average age amounted to 66,1 ± 2,1 years, men were 23 persons (63.8 %) and 13 - women (36.2%). The control group was consisted of 60 patients, which depending on the degree of compensation CCP were divided into 2 groups: 30 patients with compensated CCP (cCCP) consisted group 3a, whose average age was 55,4 ± 2,1 years, there were 19 men (63.2 %) and 11 women (36.8%) in this group, 30 patients dCCP consisted group 3b, whose average age patients was 58,2 ± 1,8 years, among them there were 21 men (70 %) and 9 women (30%).

The diagnosis in all patients was confirmed by complex clinical- instrumental and laboratory examination. In addition, all patients were underwent with ECG monitoring method by Holter device "Poly-Spectrum- CM" of Neurosoft Company, Russia. The main determining parameters were following: heart rate, supraventricular and ventricular arrhythmias, conduction disorders . Determination of parameters of circadian blood pressure profile (CBPP) was conducted on the device for BP monitoring BPLab MnSDP -1, Russia. The study of intracardiac hemodynamics was conducted on ultrasound scanner of expert class with color mapping and spectral tissue Doppler Medison SA- 9900, South Korea. Respiratory function was performed on the computer spirograph FlowScreen, U.S. with registration of flow-volume loops.

The study excluded patients with clinical and anamnestic data of ishemic heart disease, symptomatic arterial hypertension, diabetes mellitus.

Results and discussion:

According to study the heart arrhythmias were detected in all groups of patients (Tab 1). Supraventricular arrhythmias were detected in 100 % of patients, and their number increased along with degree of decompensated CCP.

Mean values of cardiac arrhythmias in patients in control groups. **Table 1.**

Rhythm disturbances/number of day	**Compensated CCP (gr.3a) n=30**	**Decompensated CCP (gr.3b) n=30**
Single PVC	211,45±44,0	386,5±92,6
Bigeminy	5,1±1,2	3,45±1,9
Trigeminy	3±1,0	1,8±0,9
Paired PVC	1,15±0,4	6,6±1,7*
Repetitive PVC	0	0,4±0,1*
Ventricular tachicardia	0	0,1±0,04*
Single PSC	268,25±86	1471,6±679
Paired PSC	5,9±1,53	107±57
SVT	1,15±0,6	5,8±3,2
Tachicardia	14,7±3,0	26,25±10,6
Bradicardia	64,4±17,7	34,05±10,6

Note: n- **the number of patients**:

*p <0,01 between 3a and 3b group

Thus, in patients with compensated cor pulmonale (cCCP) number of single supraventricular premature complexes (SSPC) was 268,2 ± 86,0, and in patients with dCCP 1541,1 ± 676 that 5.75 times greater than in patients with cCCP (Chart 1). Sinus tachycardia was also frequently detected in patients with dCCP, reaching 26,3 ± 10,6, whereas in patients cCCP the figure was 14,7 ± 3,0. In addition CCP decompensation accompanied with increase of adverse prognostic supraventricular rhythm. Under cCCP number of paired supraventricular premature complexes (PSPC) was 5,9 ± 1,5, while the group dCCP it was 107 ± 57,0 that is more than 19 times. Number of supraventricular tachycardia (SVT) in the group dCCP was 5,8 ± 3,2, whereas in patients with cCCP amount of SVT was 1,2 ± 0,6. Along with decompensation CCP it was observed a decrease episodes of sinus bradycardia at night that confirms the inappropriate normal rate variability and lack of adequate power reducing the incidence rate at night. Thus, this indicator in patients with dCCP was equal to 35,8 ± 10,6, while in patients with cCCP number of sinus bradycardia in night-time was 64,4 ± 17,7.

As for ventricular arrhythmias, it showed an increase in their total number along with degree of decompensation CCP. So the total number of single ventricular premature complexes (sVPC) in patients with dCCP compared with cCCP increased by 2.3 times, amounting to 386,5 ± 92,6 and 161,6 ± 24,0 respectively. In addition, there was a significant increase in high-grade VPC in patients with dCCP. Thus, the amount of paired VPC (pVPC) in patients with dCCP was 6,7 ± 1,7, 5.8 times greater than in patients with cCCP where this indicator was 1,2 ± 0,4 (p <0, 05). Number of pVPC (pVPC) in dCCP group was 0,4 ± 0,1, while in patients with cCCP they were not registered (p <0.05) .

Upon addition of arterial hypertension there have been a tendency to increase the number of single supraventricular complexes, with the greatest number of them registered with

increasing degree of hypertension in patients dCCPAH2, amounting to 2048,3 ± 645 (Tab.2).

Mean values of cardiac arrhythmias in patients with decompensated CCP combined with hypertension and without it.

Table 2.

Rhythm disturbances/number of day	Decompensated **CCP (gr.3б) n=30**	Decompensated **CCP AH1(gr.1) n=36**	Decompensated **CCP AH2 (gr.2) n=36**
Single PVC	386,5±92,6	176,2±31,5	514±87⌂
Bigeminy	3,5±1,9	22,6±7,5°	13,57±2,9**
Trigeminy	1,8±0,9	0,07±0,04	9,4±2,5**⌂
Paired PVC	6,6±1,7	7,7±2,1	10±2,1
Repetitive PVC	0,4±0,1	0,4±0,2	1,5±0,35*⌂⌂
Ventricular tachicardia	0,1±0,04	0	0
Single PSC	1471,6±679,0	1036±273	2048,3±645
Paired PSC	107,0±57,0	24,6±7,5	97,57±24,9⌂⌂
SVT	5,8±3,2	17,3±8,07	4,7±0,7
Tachicardia	26,3±10,6	0,6±0,16	10±2,8°⌂
Bradicardia	34,1±10,6	0♦	2,5±1,0▼

Note: n- the number of patients

° p <0,05 between 3b and 1, *p <0,01 **p <0,05 between 3b and 2 , ⌂p <0,01 ⌂⌂p <0,05 between 1 and 2

These figures were significantly higher in groups dCCPAH 1 and 2 degree than in groups with compensated CCP, but did not change significantly compared with the group dCCP that allows us to conclude that the increase in the degree of hypertension in decompensated patients had no impact on a substantial change of supraventricular rhythm disturbances, last matched decompensation CCP.

Along with increasing the degree of hypertension it was found a significant increase in the number of sVPC. So for a group dCCP combined with hypertension grade 2 this figure was 514,0 ± 87,0 which is 2.9 times higher than in the group dCCPAH1 for which this indicator amounted to 176,2 ± 31,5 ($p < 0,05$). However, the number of single sVPC in group of dCCP was greater than in the group dCCPAH1 totaling 386,5 ± 92,6. Systolic pulmonary artery pressure (SPAP) was the lowest in the group dCCPAH1 that allows to think about a possible increase sVPC largely associated with decompensation of CCP and only in the group with AH2 degrees is associated with increased blood pressure.

Indicators of bi- and trigeminy in groups with dCCP did not show any changes resulted by increase of hypertension. It was only noted a significant increase in their dCCPAH2 group compared with the group cCCP ($p < 0.01$), which is probably due to decompensation of CCP .

Performance of ventricular cardiac arrhythmias high grade indicators in decompensated patients showed a clear tendency to increasing of pair and repetitive VPC along with increase of the degree of hypertension (Tab.4). So in the group dCCP number of pVPC was 6,7 ± 1,7, in the group dCCPAH1 this figure was equal to 7,7 ± 2,1, whereas in the group dCCPAH 2 -

10,0 ± 2,1. For repetitive VPC (rVPC) in decompensated patients figure was 0,4 ± 0,1. Upon accession, AH 1 number of rVPC did not changed at 0,4 ± 0,2, and in patients with AH grade 2 there was a significant increase compared them with 1 and 3b groups ($p<0.05$) to 1,5 ± 0,35.

In patients with AH it was revealed an even greater compared with the group dCCP reduction of episodes of sinus bradycardia at night , which means a lack of an adequate degree of reducing the incidence rate at night. So if in the group dCCP the number of night episodes of bradycardia was still 34,1 ± 10,6, then in the group with AH grade 2 the number of episodes of bradycardia at night was 2,5 ± 1,0.

When conducting correlation between indicators of spirography and supraventricular arrhythmias it was detected the feedback of moderate severity in all groups of patients examined (Table 3). Under cCCP this correlation was significant only between the index Tiffno and pSPC ($r = -0,603$, $p <0.05$). it was noted increasing of negative feedback along with change in degree of decompensation CCP. So patients dCCPAH1 had a strong significant negative correlation between vital capacity (VC) and pSPC ($r = -0,750$, $p <0.01$) and moderate between forced expiratory volume in 1 second (FEV1) and pSPC ($r = -0,627$ $p < 0.02$). In group of dCCPAH2 - between FEV1 and sSPC ($r = -0,532$, $p <0.05$), index Tiffno and sSPC ($r = -0,689$, $p <0.01$). Under dCCP the correlation was significant only between the VC, FEV1 and tachycardia.

The correlation of supraventricular rhythm disturbances with maximum expiratory flow was significantly in decompensated CCP group combined and without hypertension. So in the group dCCP average force feedback was found with single, paired SPC, SVT and tachycardia, in the group dCCPAH1 with tachycardia and in the group dCCPAH 2 with singles and paired SPC. Thus, in groups supraventricular arrhythmias increases with decreasing speed performance of respiratory function.

Correlations between some indicators of spirography and supraventricular arrhythmias. **Table 3.**

Index	cCCP **(gr.3a) n=30**		dCCP **(gr.3b) n=30**		dCCPAH1 **(gr.1) n=36**		dCCPAH2 **(gr.2) n=36**	
	sPSC	**sPVC**	**sPSC**	**sPVC**	**sPSC**	**sPVC**	**sPSC**	**sPVC**
FVC,%		-0,60***	-0,39	-0,40		-0,75*		
FEV1,%			-0,40	-0,43		-0,62**	-0,53***	
FEV1/FVC,%							-0,68*	
PEF ,%			-0,56*	-0,63*				
FEF 25%								
FEF 50%				-0,44***			-0,59**	
FEF 75%							-0,75*	-0,80*
FEF 25-75%				-0,45***			-0,61*	

Note: *- $p<0,01$, **- $p<0,02$, ***- $p<0,01$

As for ventricular arrhythmias, in the group dCCP the reverse significant correlation of average power was found between volume and speed characteristics of respiratory function and single VPC, bi-and trigemini ($p <0.05$) (Table 4). In group dCCPAH2 power of

correlation was increasing between the volume and speed characteristics of spirography and bi-and trigemini, pVPC and rVPC ($p < 0.01$). In this group there were the lowest measured lung function and high SPAP .

Correlations between indicators of spirography and ventricular arrhythmias .

Table 4.

Index	**dCCP (gr.3b) n=30**			**dCCPAH2 (gr.2) n=36**			
	SPVC	**Bigem.**	**Trigem.**	**Bigem.**	**Trigem.**	**PPVC**	**GPVC**
FVC,%							
FEV1,%				-0,819*	-0,757*	-0,653*	-0,848*
FEV1/FVC,%				-0,886*	-0,838*	-0,778*	-0,945*
PEF ,%	-0,465***	-0,594*	-0,603*				
FEF 25%				-0,734*	-0,646*	-0,786*	-0,878*
FEF 50%	-0,447***	-0,460***	-0,467***	-0,866*	-0,799*	-0,789*	-0,943*
FEF 75%	-0,535**	-0,458***	-0,454***	-0,646*	-0,788*	-0,768*	-0,764*
FEF 25-75%				-0,872*	-0,806*	-0,844*	-0,972*

Note: *- p<0,01, **- p<0,02, ***- p<0,05

During conducting correlation it was found a direct relationship between cardiac arrhythmias and right atrial size (RA), as well as the level of SPAP . (**Tab.** 5) .

Correlation between the size of the right atrium, SPAP and cardiac arrhythmias.

Table 5

Index	**cCCP (gr.3a) n=30**		**dCCP(gr.3b) n=30**		**dCCPAH1(gr.1) n=36**		**dCCPAH2(gr.2) n=36**	
	RA, cm	**SPAP** , Mm.Hg.	RA, cm	**SPAP** , Mm.Hg.	RA, cm	**SPAP** , Mm.Hg.	RA, cm	**SPAP** , Mm.Hg.
Single PVC					0,555***	0,66*	0,560***	0,642*
Bigeminy					0,865*	0,56***	0,689*	0,779*
Trigeminy								0,556***
Paired PVC				0,81*	0,823*	0,59***		
Repetitive PVC				0,51***	0,843*	0,58***		
Single PSC	0,482***						0,636*	
Paired PSC		0,447***						
SVT		0,502***			0,573*			

Note: *- p<0,01, **- p<0,02, ***- p<0,05

Thus in patients in the cCCP it was revealed a significant correlation of medium strength between sSPC and RA size (r=0,48, p<0.05). Under dCCP this correlation is poorly expressed and unreliable.

In patients dCCPAH1 it was revealed significant direct correlation between the size of the RA and SVT, as well as singles, pared, group VPC and ventricular bigeminy (r=0,7-0,8, p <0.01).

In group dCCPAH2 it was revealed significant direct correlation of the average strength between the size of the RA and sSPC, sVPC and ventricular bigeminy (r=0,56, p<0.05 and r=0,63, p<0.01).

Also it was found the significant correlation of medium degree between SPAP and cardiac arrhythmias . In the group cCCP with pSPC and SVT (r=0,44-0,53, p<0.05), in the group dCCP with pVPC (r=0,81, p<0.01), rVPC (r=0.5, p<0.05), in the group with dCCPAH1 with sVPC (r=0,66, p<0.01), bigemini (r=0,56, p<0.05), pVPC (r=0,59, p<0.05) and rVPC (r=0,58, p<0.05), in the group with dCCPAH2 with sVPC, bi- and trigemini (r=0,55-0,78, p<0,02-0,01).

Also it was found significant inverse correlation of average strength between the ejection fraction and LV contractility with ventricular disorders in groups (Tab. 6) in dCCP between EF %, ΔS% and pVPC and gVPC, ventricular tachicardia (r= -0,44-0,52, p<0.05) and in the group with dCCPAH1 between EF % and sVPC and pVPC (r = -0,74, p < 0.01 and r = -0,56, p < 0.05) SVT (r = -0,57, p <0,05), ΔS% with sPVC (r = -0,57, p < 0.05), in group dCCPAH2 between EF % and pVPC (r=-0.53, p<0,05), ΔS% with sVPC (r=-0,495, p<0.05). Reduction of myocardial contractility increases the number of ventricular cardiac arrhythmias.

Correlations between indicators of myocardial contractile function and ventricular arrhythmias.

Table 6.

Index		**dCCP(gr.3b) n=30** EF%	ΔS%	**dCCPAH1(gr.1) n=36** EF%	ΔS%	**dCCPAH2(gr.2) n=36** EF%	ΔS%
Single PVC	r_{xy}			-0,728*	-0,676*		-0,495***
Trigeminy	r_{xy}				-0,550***		
Paired PVC	r_{xy}	-0,537***	-0,493***	-0,553***		-0,528***	
Repetitive PVC	r_{xy}	-0,637**	-0,580***	-0,547***			
Ventricular tachicardia	r_{xy}	-0,674***	-0,595***				

Note:*- p<0,01, **- p<0,02, ***- p<0,05

During conducting correlation between left ventricular myocardial mass (LVM), size of the interventricular septum (IVS) and cardiac arrhythmias in the group dCCPAH1 it was found a direct significant correlation with moderate severity with trigemini (LVM -r = 0,55, IVS -r = 0,50), and in the group with dCCPAH2 with sVPC (LVM -r = 0,38, IVS -r = 0,67) (Tab.7).

Correlation between the size of the interventricular septum, left ventricular mass and cardiac arrhythmias
Table 7.

Index		**dCCPAH1(gr.1) n=36** IVC,cm	**LVM ,g**	**dCCPAH2(gr.2) n=36** **IVC,cm**	**LVM ,g**
Single PVC	r_{xy}			0,67*	0,38
Trigeminy .	r_{xy}	0,50***	0,55***		

Note: *- p<0,01, **- p<0,02, ***- p<0,05
During conducting of correlation between arrhythmias and indicators of ABPM it was revealed the effect of time indexes and blood pressure variability on arrhythmias CCP group in combination with AH. It was found the direct dependence of moderate severity, although unreliable under dCCPAH1 between day time indexes of systolic blood pressure (TISBPd) , day time indexes of diastolic blood pressure (TIDBPd) and night time indexes of diastolic blood pressure (TIDBPn) and sSPC , in the group dCCPAH2 between TISBP, TIDBP both day and night time and pVPC and rVPC, and between TIDBP n and pVPC this correlation was significant (r=0,54, p<0.05)
Effect of blood pressure variability on arrhythmias is following: in the group dCCP it was found a direct significant correlation of average strength between the variability of daytime of diastolic blood pressure (VDBPd) and gVPC (r=0,49, p<0.05), ventricular tachicardia (r=0,59, p<0,01) , in the group dCCPAH2 between VDBPd and variability of nighttime of systolic blood pressure (VSBPn) and pSPC , SVT (r=0,61-0,71, p<0.01), variability of nighttime of diastolic blood pressure (VDBPn) and bi-and trigemini, gVPC (r=0,5-0.63, p<0.05-0.01). Under dCCPAH1 it was revealed the correlation between VSBPn and VDBPn and ventricular trigemini, pSPC, which was of medium strength and unreliable.
Thus, as a result of the study it was found that in patients with decompensated chronic cor pulmonale regardless of the presence of arterial hypertension the causes of cardiac arrhythmias are worsening pulmonary ventilation and increase in pulmonary hypertension. Reducing the speed and volume indicators of spirography, as well as increasing the size of RA and SPAP in these patients leads to appear supraventricular arrhythmias, particularly supraventricular singles and paired extrasystoles, and single and high grade ventricular extrasystoles.
Furthermore, we can assume that the appearance of high-grade VPC in patients with dCCP could be caused by changes in left ventricular myocardium by hypoxia, given the correlation between left ventricular ejection fraction and VPC.
With the addition of arterial hypertension it is observed the increase of VPC number in patients with dCCP. The reason for this is the reduction of myocardial contractile function in groups with hypertension, which leads to an increase in the amount of single and paired VPC, as well as an increase in left ventricular mass and hypertrophy of the interventricular septum, leading to a rise in the number and single VPC and trigeminy.
Increased variability in SBP and DBP at night, which is a feature of arterial hypertension in patients with CCP, increases bigemia, trigeminy, repetitive VPC in groups with arterial

hypertension. Under dCCPAH2 where the load incident on the target organs during the day was the largest the number of VPC increases.

As a result of increased activation of sympathetic nervous system in patients dCCP it was noted the disruption of normal variability night rhythm in the form of reducing the number of nocturnal physiological bradycardia. With joining AH these changes are compounded and physiological bradycardia in night hours recorded in individual cases.

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