Vectorcardiographic assessment of acute hypoxia effects in pulmonary hypertension due to chronic bronchitis

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ABSTRACT

Objective: Vectorcardiography (VCG) QRS loop area is thought to reflect hemodynamic abnormalities in cardiac lesions. The aim of the present study was to evaluate the response of VCG QRS loop area and intrapulmonary flow (PF) to acute hypoxia in patients with chronic bronchitis (CB) and pulmonary arterial hypertension (PAH).

Methods: One-hundred and eleven patients (mean age 45.0±1.0 years) with chronic bronchitis (CB) and 43 comparable healthy subjects were included into the study. Diagnosis of CB was based on clinical, radiological, spirography and oximetry investigations. Cardiac catheterization, 2-dimensional and Doppler echocardiography, VCG by Mcfee-Parungao (VCG) and electrocardiography were used to establish PAH and right ventricular hypertrophy (RVH). Pulmonary flow was studied using impedance plethysmography (IP). Vectorcardiography, IP and Doppler echocardiography were also performed to assess the changes in QRS loop area, PF and mean pulmonary artery pressure (PAPm) in response to acute hypoxia (inhalation of hypoxic mixture 16% O_2 for 5-10 minutes) in 21 patients with CB and 7 healthy subjects.

Results: At baseline, patients with CB were characterized by pulmonary ventilation disturbances of mixed and obstructive types. Among patients who underwent acute hypoxia test PAPm was increased in 10 patients (29.2±2.1 mmHg) (Group 1), while in the latter 11 patients the PAPm was within normal values (16.5+1.8 mmHg) (Group 2). The IP showed reduction of PF and increase in pulmonary vascular resistance. Patients with CB react to the acute hypoxic test by increase in PAPm by 32% (absolute increase up to 22.5±0.7 mm Hg) in group 2 and by 38.9% (absolute increase up to 39.7±2.1 mmHg) in patients of group 1. There was a displacement of PF on IP to the upper and middle zones of lungs during acute hypoxia: PF in the upper and middle zones of the right lung increased by 35.6% and 32.5% in patients of group 2; while in patients of group 1 the PF increased by 33.5% and 4.8% at the same zones. On VCG QRS loop area was enhanced significantly in horizontal plane by 68% ($p<0.05$), but more significant changes were recorded in the anterior direction (by 75%, $p<0.02$) during acute hypoxia.

Conclusion: Thus, in patients with early stages of PAH, changes in PF and QRS loop area during acute hypoxia may reflect the compensatory redistribution of pulmonary flow in response to hypoxia. *(Anadolu Kardiyol Derg 2007; 7 Suppl 1; 198-200)*

Key words: vectorcardiography, pulmonary hypertension, pulmonary flow, pulmonary circulation

Introduction

Pulmonary arterial hypertension (PAH) due to alveolar hypoxia developed in connection with obstructive and restrictive processes in lungs (chronic obstructive pulmonary disease and high altitude hypoxia) is a serious hemodynamic risk factor for right ventricular (RV) overload and hypertrophy with further its exhaustion and development of chronic cor pulmonale and congestive RV heart failure (1-5).

That is why, the questions of early diagnosis of PAH in chronic obstructive pulmonary diseases before the involvement in pathological process of the RV as the target organ and pursuit of noninvasive new electrocardiographic (ECG) and vectorcardiographic (VCG) criteria of electrodynamic forces characteristic for RV overload and hypertrophy in patients with chronic obstructive pulmonary diseases have special importance (1, 6).

The aim of the present study was to identify the VCG parallels in diagnosis of early changes of pulmonary flow in PAH far before the involvement of RV in pathological process in patients with chronic bronchitis (CB) and to evaluate the response of VCG QRS loop area and intrapulmonary flow to acute hypoxia (O_2=16%, 10 minutes) in patients with chronic bronchitis (CB) and pulmonary artery hypertension .

Methods

One-hundred and eleven patients (mean age 45.0±1.0 years) with chronic bronchitis (CB) and 43 comparable healthy subjects were included into the study.

Diagnosis of CB was established on clinical, radiological, spirography and oximetry investigations. Cardiac catheterization, 2-dimensional and Doppler echocardiography, VCG by Mcfee-Parungao method (7) and ECG were used to establish PAH and right ventricular hypertrophy. Pulmonary flow was studied using impedance plethysmography (IP).

Among 111 patients with CB and 43 healthy subjects assessment of pulmonary circulation reactivity to acute hypoxia was

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performed in 28 participants (mean age 43.4±1.7 years), whom constituted 3 groups: Group 1 (control) – 7 apparently healthy persons; Group 2 – 11 patients with CB and normal PAPm and Group 3 – 10 patients with CB and high PAPm.

It is known, that diagnosis of early and silent manifestations of diseases may be accomplished through exposure to additional load, exercise or stimuli which might cause the noticeable reaction of the pulmonary circulation. With this purpose the functional test with inspiration of hypoxic gas mixture (O_2=16%) during 10 minutes was performed in 28 subjects.

For the assessment of the reaction of pulmonary circulation to acute hypoxia test IP, Doppler echocardiography and VCG were performed before and during the test.

The magnitude of the intrapulmonary flow was estimated using indirect zonal IP, which allows to register changes in electrical resistance in different pulmonary zones (8).

The changes of PAPm in response to acute hypoxia were assessed by Doppler echocardiography by the method of Kitabatake et al. (9).

The assessment of the RV reaction to acute hypoxia and related changes according with reaction of pulmonary circulation and pulmonary hemodynamics were performed using VCG (7). The VCG is an indirect method, which has advantages over other methods like ECG in diagnosis of early hemodynamics changes in pulmonary circulation. The special importance has the utilization of corrected VCG leads. We used the system by McFee and Parungao (7), which provides qualitative characteristics of the early changes in the pulmonary circulation and RV. According to the method of Chow et al. (10), the following parameters of RV were quantified: the planimetry area of the QRS loop in the II, III and IV quadrants of the transverse plane and its right and anterior/frontal orientations.

Results

Patients with CB had cough syndrome, moderate expiratory dyspnea, and rarely (mostly at night) wheezing in the chest. There were no significant signs of PAH, except splitting of the S2 at pulmonary trunk area on auscultation. The impairments of lung ventilation were of moderate degree with mixed type with prevalence of obstruction. There were no obvious signs of RV involvement as the target organ damage according to ECG, VCG and echocardiography data. There was no significant reduction in the oxygen saturation (SaO_2).

Regional IP data (Table 1) showed a unique reaction of intrapulmonary flow to brief acute hypoxia in healthy subjects and patients with CB. In healthy subjects, there was a prevalence of pulmonary flow in the lower lung zones. In CB patients with normal PAPm and high PAPm at rest pulmonary flow has tendency to redistribution to upper lung zones. In healthy subjects at the peak of hypoxia, the pulmonary flow in the upper lung zones increased by 24% and in lower lung zones, in contrary, reduced approximately to the same value.

Similarly, redistributive reactions in response to aggravation of alveolar hypoxia took place in patients with normal PAPm at rest with increase of pulmonary flow in the upper and middle lung zones by 35.6% and 32.5%, respectively. In patients with CB and stable high PAPm the extent of increase of pulmonary flow in upper lung fields was equal to 33.5%.

These changes in redistribution of pulmonary circulation in response to acute hypoxia in healthy subjects as well as CB patients were accompanied by increase in PAPm (Table 2).

The latter hemodynamics changes during alveolar hypoxia, though being compensatory reaction to improve the blood

Table 1. IP data of intrapulmonary flow (in parts of lungs) before and during acute hypoxia (O_2=16%) in CB patients with high and normal PAPm and healthy subjects

Patients groups	Intrapulmonary flow, Ohm/sec		
	Upper zone	Middle zone	Lower zone
Healthy subjects (n=7)			
Baseline	1.30±0.15	1.37±0.09	1.94±0.13
Hypoxia	1.59±0.16	1.49±0.13	1.42±0.13
% of changes	+24.2	+8.4	-22.4
p			0.02
CB patients with normal PAPm (n=11)			
Baseline	0.73±0.09	0.61±0.08	0.67±0.09
Hypoxia	0.99±0.09	0.73±0.08	0.48±0.04
% of changes	+35.6	+32.5	- 21.0
p	0.05		
CB patients with high PAPm (n=10)			
Baseline	1.00±0.12	0.74±0.13	1.05±0.11
Hypoxia	1.27±0.11	0.77±0.11	0.84±0.12
% of changes	+ 33.5	+ 4.8	- 21.8
p			0.05

CB- chronic bronchitis, PAP- pulmonary artery pressure, IP- impedance plethysmography

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oxygenation, caused overload of RV in patients with CB and healthy subjects as documented by VCG. Vectorcardiography during acute hypoxia documented RV overload signs as increase in QRS loop in the II, III, and IV quadrants of transverse plane in healthy subjects, orientation of QRS loop to right with increase in QRS loop area by 68% in CB patients with high PAPm and frontal orientation of QRS loop with increase in QRS loop area by 75% in patients CB and normal PAPm.

Discussion

Our study shows that reaction of pulmonary circulation to acute hypoxia in form of increase in pulmonary flow and its redistribution in the upper lung zones in early stages of PAH due to CB being the compensatory reaction, causes RV overload and should be considered as a serious hemodynamic factor of overload, hypertrophy and RV exhaustion. Early and transient changes in pulmonary circulation in patients with CB manifested at VCG by increase in QRS loop in transverse plane and its orientation to right and front.

Thus, in patients with early stages of PAH changes in pulmonary flow and QRS loop area during acute hypoxia may reflect the compensatory redistribution of pulmonary flow in response to hypoxia.

Table 2. Pulmonary arterial pressure data of CB patients with high and normal PAPm and healthy subjects before and during acute hypoxia (O_2=16%)

Patients groups	PAPm, mmHg
	Healthy subjects (n=7)
Baseline	12.4±0.7
Hypoxia	15.4±1.9
% of changes	+24.2
p	
	CB patients with normal PAPm (n=11)
Baseline	16.5±1.8
Hypoxia	22.5±0.7
% of changes	+32.0
p	0.05
	CB patients with high PAPm (n=10)
Baseline	29.2±2.1
Hypoxia	39.7±2.1
% of changes	+38.9
p	0.01

CB- chronic bronchitis, PAP- pulmonary artery pressure

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