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Rare diseases of pulmonary circulation

Right ventricular free wall motion abnormalities as a simple method of assessment in patients with pulmonary hypertension (RCD code: II-1A.O)

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Abstract

Background: Pulmonary hypertension (PH) is a cardiovascular pathology leading to right-sided heart failure. A qualitative assessment of right ventricular (RV) function in echocardiography provides valuable information on a patient's condition. The standard echocardiographic parameter, assessed in PH patients, is RV free wall motion. **Aim**: To verify the utility of RV free wall motion assessment via echocardiography in PH patients. **Methods**: Data from 30 PH patients, regardless of aetiology (except for left heart disease), was retrospectively analyzed. Based on the RV free wall motion visual echocardiographic assessment the population was divided into: group 1- normokinetic; group 2- hypokinetic RV. All patients underwent a medical interview, physical examination, basic laboratory work-up, echocardiography, and right heart catheterization (RHC). Twenty-one patients underwent a cardiopulmonary exercise test (CPET). **Results**: The analysis revealed, that patients with RV free wall hypokinesis were characterized by impaired gas exchange parameters (higher values of ventilatory equivalents for oxygen and carbon dioxide, higher end-tidal oxygen pressures, lower end-tidal carbon dioxide pressures and higher minute ventilation – carbon dioxide production relation slope) and cardiovascular response to exercise (lower increase in O2 pulse during exercise) obtained in the CPET. RHC showed that patients with hypokinetic RV had higher diastolic and mean pulmonary artery pressures (dPAP, mPAP), lower cardiac index, and higher pulmonary vascular resistance. **Conclusions**: RV free wall motion abnormalities, assessed using echocardiography in PH patients, are found in those with more advanced disease. They are characterized by impaired ventilation in the CPET and more advanced haemodynamic abnormalities in RHC. The association between this parameter and prognosis requires validation in a larger population of patients. JRCD 2017; 3 (5): 161–167

Key words: pulmonary hypertension, right heart catheterization, cardiopulmonary exercise test, right ventricular failure, rare disease

Background

Pulmonary hypertension (PH) is a haemodynamic condition that can be a complication of various cardiovascular and respiratory diseases and can be divided into pre-capillary PH (pulmonary arterial hypertension (PAH), PH due to lung diseases, chronic thromboembolic PH, PH with unclear and/or multifactorial mechanisms) or post-capillary PH (PH due to left heart disease, PH with unclear and/or multifactorial mechanisms).

The prevalence of PH (defined in echocardiography as a systolic pulmonary artery pressure (sPAP) > 40 mmHg) is 2.6% in the general population, and reaches 8.3% in patients over 85 years of age [1]. According to the 2015 European Society of Cardiology (ESC) Guidelines, normal mean pulmonary arterial pressure (mPAP) is < 20 mmHg, while a value of ≥ 25 mmHg at rest in right heart catheterization (RHC) indicates PH [2]. The most important non-invasive methods employed in the preliminary diagnosis of PH, estimation of right ventricular (RV) function, and assessment of

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Table 1. Demographic characteristics of study populations

	Group 1 (normokinetic RV free wall motion, n=8)	Group 2 (hypokinetic RV free wall motion, n=22)	P value	Whole population (n=30)
Age, years	60.5 IQR: 40.5 – 67.5	65.5 IQR: 50 – 74	0.59	61.5 IQR: 43 – 74
Female sex,% (n)	50 (4)	68.2 (15)	0.361	63.3 (19)
BMI (kg/m^2)	25.2 IQR: 22 – 31.7	24.4 IQR: 21 – 26.8	0.656	24.4 IQR: 21 – 27.6
Arterial hypertension,% (n)	50 (4)	45.5 (10)	0.825	46.7 (14)
Diabetes,% (n)	12.5 (1)	18.2 (4)	0.712	16.7 (5)
Ischemic heart disease,% (n)	0 (0)	4.5 (1)	0.54	3.3 (1)
Atrial fibrillation,% (n)	37.5 (3)	13.6 (3)	0.149	20 (6)

Data are presented as median and interquartile range. Statistically significant parameters are marked with italics.

functional capacity, include transthoracic echocardiography (TTE) and the cardiopulmonary exercise test (CPET).

The RV is a thin-walled, crescent-shaped chamber with higher compliance than the left ventricle. This ventricle has high volumetric capacity, but because of its construction, is not adapted to increased pressure loads. Therefore, an increase in afterload results in dilatation and thickening of its walls, which can lead to RV dysfunction. Furthermore, the character of afterload seems to be different in PH than in a pulmonary valve stenosis. In PH patients, a more rapid progression of heart failure is observed. The underlying cause for this may be an increase in the stiffness and pressure of the pulmonary circulation as well as pulse wave reflection phenomenon. However, the mechanisms behind the above mentioned condition are complex, not fully determined, and require further investigations.

The complex assessment of RV function in PAH patients plays an important diagnostic and prognostic role. Of the parameters routinely assessed in echocardiography, RV free wall motion is important because of its accessibility and simplicity.

The aim of the study was to verify the utility of RV free wall motion assessment via echocardiography in PH patients.

Methods

A retrospective analysis was conducted using the medical documentation of 30 consecutive patients (19 females, 11 males) with PH confirmed in RHC (mPAP ≥ 25 mmHg), aged 29–81 years (median 61.5 IQR: 43 – 74 years), who had RV free wall motion assessed by TTE. All patients underwent a medical interview, physical examination, TTE, and RHC, while CPET was performed on 21 patients. The use of CPET was limited mainly by inability to perform the exercise or intolerance of the mask used during the test. The condition of patients remained stable between the conducted procedures.

Patients were divided into two groups based on visual assessment of RV free wall motion: group 1- normokinetic RV free wall (n = 8) and group 2- hypokinetic RV free wall motion (n = 22). Characteristics of study groups are shown in Table 1.

The aetiology of PH was heterogeneous and comprised both pre- and post-capillary PH. Table 2 shows the clinical classification of the PH population based on 2015 ESC Guidelines.

The distribution of all variables was verified using the Kolmogorov-Smirnov test. The data was presented as a median value with an interquartile range (IQR) for continuous variables or with a number and a percentage for categorical data. Statistical analysis was performed using the Mann–Whitney U test, chi square test and ROC curve analysis. $P < 0.05$ was considered statistically significant. A statistical software package, Statistica 13.1 (StatSoft Inc., USA), was used for analysis.

All aspects of the study are in compliance with the Declaration of Helsinki.

Right heart catheterization

The procedure was conducted under local anesthesia via internal jugular or femoral venous access. Both the thermodilution and indirect Fick method of measurements were performed [3,4]. The standard protocol includes the assessment of: systolic, diastolic and mean pulmonary artery pressures (sPAP, dPAP, mPAP, respectively), cardiac output (CO), cardiac index (CI), right atrial pressure (RAP), pulmonary vascular resistance (PVR), and systemic resistance.

Transthoracic echocardiography

TTE was used to exclude any significant heart abnormalities and to assess RV morphology and function. The two-dimensional and Doppler echocardiographic assessments included RV free wall motion, tricuspid annular plane systolic excursion (TAPSE), acceleration time (ACT), as well as sPAP, mPAP, and tricuspid regurgitation pressure gradient (TRPG). Systolic pulmonary artery

Table 2. The etiology of pulmonary hypertension in each study group

				Group 1 (normokinetic RV free wall motion, n=8)	Group 2 (hypokinetic RV free wall motion, n=22)	P value	Whole population (n=30)
ETIOLOGY	Pre-capillary PH	1. PAH,% (n)	Idiopathic PAH	12.5 (1)	50 (11)	0.064	40 (12)
			Connective tissue diseases	12.5 (1)	22.7 (5)	0.536	20 (6)
			Congenital systemic-to- pulmonary shunts	50 (4)	18.2 (4)	0.082	26.7 (8)
		3. PH due to lung diseases,% (n)		0 (0)	9.1 (2)	0.377	6.7 (2)
		4. Chronic thromboembolic PH,% (n)		12.5 (1)	0 (0)	0.092	3.3 (1)
		5. PH with unclear and/or multifactorial mechanisms,% (n)		12.5 (1)	0 (0)	0.092	3.3 (1)

PH, pulmonary hypertension; PAH, pulmonary arterial hypertension

pressure was estimated from the tricuspid regurgitation peak velocity (TRV) and an estimated right atrial pressure (eRAP), where sPAP = 4(TRV)2 + eRAP. RAP was estimated according to the method introduced by Rudski et al., based on inferior vena cava (IVC) respiratory collapsibility and classified as 5, 10 or 15 mmHg [5]. Assessment of RV free wall motion was based on visual evaluation by an experienced echocardiographist.

Cardiopulmonary exercise testing

The CPET was conducted with a symptom-limited treadmill exercise test using the RAMP protocol. During the exercise, PH patients had continuous ECG monitoring with heart rate assessment. The maximal load (expressed in Watts) was limited by the patient's exercise tolerance. Of the numerous parameters, peak oxygen uptake (peak VO_2), peak carbon dioxide exertion (peak VCO_2), end-tidal partial pressure of oxygen ($PetO_2$), end-tidal partial pressure of carbon dioxide ($PetCO_2$), ventilatory equivalents for oxygen (VE/O_2), ventilatory equivalents for carbon dioxide (VE/CO_2), minute ventilation–carbon dioxide production relationship from the initiation to peak exercise (VE/VCO_2 slope), and O_2 pulse were used for analysis.

Results

Patients with RV free wall motion abnormalities did not differ significantly in demographic variables, comorbidities, and PH aetiology (Table 1 and 2).

Impaired motion of the RV free wall was associated only with an insignificant trend towards higher brain natriuretic peptide concentration (Table 3). Among basic laboratory tests there were no differences between study groups (Table 3).

The most significant differences between groups were observed in CPET parameters (Table 3). The group with hypokinetic RV free wall motion was characterized with worse parameters reflecting gas exchange in the lungs (higher values of VE/CO_2, VE/O_2, $PetO_2$ and lower values of $PetCO_2$) and cardiorespiratory response to exercise (lower increase in O_2 pulse during exercise and higher indicator of increased exercise ventilation VE/VCO_2 slope). There were no differences in the maximum load and metabolic gas exchange parameters (peak VO_2 and peak VCO_2) between groups (Table 3).

Furthermore, patients with RV free wall motion abnormalities were characterized by higher mPAP, dPAP, and lower CI. In addition, they had increased PVR and systemic resistance as assessed by the thermodilution method in RHC (Table 4).

Among TTE parameters, patients with hypokinetic RV free wall had combined impairment of parameters reflecting the increase in ventricular afterload (Table 5).

ROC curve analysis revealed a relationship between mPAP (AUC = 0.76, CI 0.56 - 0.97), PVR (AUC = 0.85, CI 0.72 - 0.99), CI (AUC = 0.76, CI 0.58 - 0.94) obtained by the thermodilution in the RHC and the impaired RV motion assessed in TTE (Figure 1A–C, respectively).

Discussion

Our study describes the differences in functional and haemodynamic parameters between PH patients with impaired or preserved RV function. Evaluation of the RV was based on TTE-derived assessment of RV free wall motion, which is non-invasive and widely available. Impairment of RV free wall motion was associated with worse clinical status reflected by impaired parameters of gas exchange in the lungs, indicators of increased exercise ventilation, worse CPET results, and abnormal haemodynamic parameters measured in the RHC. Moreover, impaired RV contractility indicated an abnormal cardiorespiratory response to

Table 3. Differences in values of laboratory and cardiopulmonary exercise test parameters

	Group 1 (normokinetic RV free wall motion, n=8)	Group 2 (hypokinetic RV free wall motion, n=22)	P value
Laboratory tests			
BNP (pg/mL)	137.7 IQR: 118.5 – 517.4	525 IQR: 152 – 1088	0.287
Troponin (IU/L)	0.02 IQR: 0.01 – 0.03	0.02 IQR: 0 – 0.05	0.893
ALT (IU/L)	26 IQR: 20 – 28	19 IQR: 12- 28	0.33
AST (IU/L)	21 IQR: 20 – 35	21 IQR: 18 – 23	0.513
Total bilirubin (mg/dL)	1.3 IQR: 0.8 – 1.7	1 IQR: 0.7 – 1.8	0.71
Total cholesterol (mg/dL)	222 IQR: 174 – 258	171 IQR: 148.5 – 199	0.112
C-reactive protein (IU/L)	2 IQR: 0.7 – 5	4.5 IQR: 3 – 7.8	0.228
Fibrinogen (mg/dL)	306.5 IQR: 297 – 316	373 IQR: 297 – 404	0.43
Creatinine (mg/dL)	0.81 IQR: 0.71 – 1	0.9 IQR: 0.8 – 1	0.492
Urea (mg/dL)	43 IQR: 31 – 58	39 IQR: 29.5 – 63	0.789
Uric acid (mg/L)	6.3 IQR: 6 – 10.3	6.6 IQR: 5.2 – 7.8	0.659
Hemoglobin (g/dL)	14 IQR: 12.1 – 15.3	14.2 IQR: 13 – 15.7	0.69
White blood cell (10^9/L)	6.5 IQR: 5.8 – 9.7	7.6 IQR: 5.3 – 8.9	0.915
Platelets (10^9/L)	171 IQR: 152 – 273	190 IQR: 147 – 242	0.977
Cardiopulmonary exercise test parameters			
Maximum load (W)	95 IQR: 48 – 142	56 IQR: 41 – 79	0.068
Metabolic gas exchange			
Peak VO_2 (mL/min/kg)	16.3 IQR: 13.6 – 22.2	12.6 IQR: 9.2 – 15.2	0.062
Peak VCO_2 (l/min)	1.2 IQR: 0.7 – 1.9	0.9 IQR: 0.6 – 1.1	0.235
Gas exchange in the lungs			
$PetO_2$ AT (mmHg)	104.6 IQR: 95.4 – 108.3	123.8 IQR: 121.1 – 128.8	*0.002*
$PetO_2$ max (mmHg)	109 IQR: 103.6 – 114.9	130.1 IQR: 123.3 – 132.4	*0.003*
$PetCO_2$ AT (mmHg)	31.2 IQR: 30.4 – 44.3	18 IQR: 16.6 – 22.8	*0.009*
$PetCO_2$ max (mmHg)	31.7 IQR: 30.3 – 52.5	17.2 IQR: 13.6 – 21.8	*0.007*
VE/O_2 AT	26 IQR: 23 – 32	48 IQR: 42 – 62	*0.003*
VE/O_2 max	32 IQR: 29 – 33	59 IQR: 50 – 70	*0.002*
VE/CO_2 AT	31 IQR: 22 – 37	53 IQR: 41 – 62	*0.006*
VE/CO_2 max	32 IQR: 23 – 36	58 IQR: 44 – 68	*0.006*
Indicator of increased exercise ventilation			
VE/VCO_2 slope	33.6 IQR: 26.3 – 38.8	59.7 IQR: 50.2 – 72.5	*0.014*
Cardiovascular response			
O_2 pulse rest (mL)	3.6 IQR: 3.1 – 4.1	3.8 IQR: 3.2 – 4.4	0.794
O_2 pulse peak (mL)	8.5 IQR: 6.2 – 17.8	6.5 IQR: 5 – 8.1	0.135
Δ O_2 pulse (mL; absolute value)	4.4 IQR: 3.4 – 10.3	2.5 IQR: 1.4 – 4	*0.04*

BNP, brain natriuretic peptide; ALT, alanine aminotransferase; AST, aspartate aminotransferase; AT, anaerobic threshold; peak VO_2, peak oxygen uptake; peak VCO_2, peak carbon dioxide exertion; $PetO_2$, end-tidal partial pressure of oxygen; $PetCO_2$, end-tidal partial pressure of carbon dioxide; VE/O_2, ventilatory equivalents for oxygen; VE/CO_2, ventilatory equivalents for carbon dioxide; VE/VCO_2 slope, minute ventilation–carbon dioxide production relationship from the initiation to peak exercise; Δ O_2 pulse (mL; absolute value), O_2 pulse peak – O_2 pulse rest
Data are presented as median and interquartile range. Statistically significant parameters are marked with italics.

Table 4. Differences in values of right heart catheterization parameters

	Group 1 (normokinetic RV free wall motion, n=8)	Group 2 (hypokinetic RV free wall motion, n=22)	P value
mPAP (mm Hg)	35 IQR: 29 – 48	54.5 IQR: 43–63	*0.033*
dPAP (mm Hg)	24 IQR: 17 – 32	36.5 IQR: 29–44	*0.011*
sPAP (mm Hg)	61.5 IQR: 49 – 82.5	87 IQR: 73–97	0.064
RAP (mm Hg)	9 IQR: 7 – 16	9 IQR: 7–14	0.961
CO thermodilution (l/min)	3.8 IQR: 3.5 – 6.1	3.5 IQR: 3.1–4	0.127
CI thermodilution (l/min/m^2)	2.2 IQR: 2.1 – 3.1	2 IQR: 1.7–2.4	*0.033*
PVR thermodilution (Wood u.)	4.1 IQR: 3.6 – 6.6	11.5 IQR: 8.2–16.1	*0.006*
PVR thermodilution (dyn*s/cm^5)	528 IQR: 294.7 – 696.4	975.5 IQR: 634.2–1617.8	*0.017*
Systemic resistance thermodilution (Wood u.)	19.1 IQR: 17.5 – 20.1	24.4 IQR: 20.4–28.3	*0.044*
Systemic resistance thermodilution (dyn*s/cm^5)	1529.7 IQR: 1398.9 -1607.1	1953.9 IQR: 1634–2264.6	*0.044*

mPAP – mean pulmonary arterial pressure; dPAP, diastolic pulmonary arterial pressure; sPAP – systolic pulmonary arterial pressure; RAP – right atrial pressure; PVR – pulmonary vascular resistance; CO – cardiac output; CI – cardiac index
Data are presented as median and interquartile range. Statistically significant parameters are marked with italics.

Table 5. Differences in values of parameters obtained in transthoracic echocardiography

	Group 1 (normokinetic RV free wall motion, n=8)	Group 2 (hypokinetic RV free wall motion, n=22)	P value
TAPSE (mm)	20 IQR: 19 – 27	16 IQR: 13–18	*0.002*
ACT (msec)	106 IQR: 85.5 – 121.5	77 IQR: 60–85	*0.011*
sPAP (mmHg)	57 IQR: 44 – 82.5	84 IQR: 68–96	0.12
mPAP (mmHg)	36.8 IQR: 28.8 – 52.3	53.2 IQR: 43.5–60.6	0.12
TRPG (mmHg)	47 IQR: 31.5 – 75	74 IQR: 55 – 81	0.12

TAPSE, tricuspid annular plane systolic excursion; ACT, acceleration time in pulmonary artery; sPAP, systolic pulmonary arterial pressure; mPAP, mean pulmonary arterial pressure; TRPG, tricuspid regurgitation pressure gradient
Data are presented as median and interquartile range. Statistically significant parameters are marked with italics.

exercise, suggesting that in PH patients it is RV dysfunction that limits exercise capacity.

Current guidelines recommend the use of BNP or its N-terminal fragment (NT-pro-BNP) for baseline evaluation and longitudinal follow-up of PH patients [2]. It was previously shown that in PH patients, BNP reflects pressure and volume overload of the RV and increases in proportion to RV dysfunction [6]. Moreover, increased levels of NT-pro-BNP are associated with the risk of clinical deterioration or death [7,8]. In the study, we did not revealed the statistically significant association between the impaired RV motion and elevated BNP concentration, however the trend for higher values in the RV dysfunction group was observed. These results might be related to the small size of the PH group and thus, studies on a larger PH group are required.

Among echocardiographic parameters, TAPSE differed between the study groups. It was lower in the group 2, indicating more pronounced RV dysfunction. Previous studies showed that TAPSE has prognostic value [9] and is associated with survival in PH patients [10].

A significant finding in our study was the confirmation of differences in CPET parameters. The CPET provides useful information on both cardiovascular and respiratory systems. In the study, the group with impaired RV free wall motion was characterized by abnormal gas exchange parameters (higher values of VE/CO_2, VE/O_2, $PetO_2$ and lower of $PetCO_2$), indicators of increased exercise ventilation (higher VE/VCO_2 slope), as well as parameters reflecting decreased cardiovascular response (lower increase in O_2 pulse during exercise).

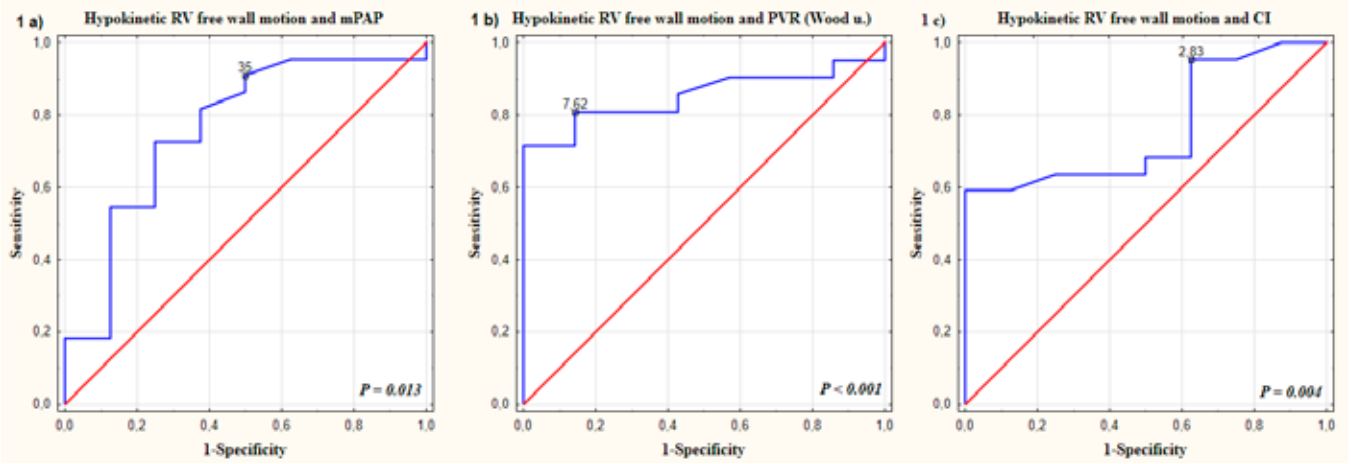

Figure 1. The relationships between mPAP (A), PVR (thermodilution method; B), CI (thermodilution method; C) obtained in RHC and the hypokinetic RV free wall motion (ROC curves analysis)

As expected, the group with hypokinetic RV free wall, which represents PH patients with more advanced right heart dysfunction, was characterized by higher values of VE/VCO_2 slope. This reflects the abnormal relationship between ventilation and perfusion in the pulmonary circulation. This well-known CPET parameter has prognostic significance and has been previously described in patients with chronic heart failure (HF) [11-14] and in PH patients [15]. A previous study demonstrated increased ventilatory responses to exercise in both PH and HF patients, however, a higher value of VE/VCO_2 slope was seen in the PH group [16]. The study of Schwaiblmair M et al. of 116 PH patients demonstrated, that $VE/VCO_2 \geq 55$ has a 7.8-fold, while VE/VCO_2 slope ≥60 a 5.8-fold increased risk of the mortality in the 24 months follow-up [15]. Furthermore, with an increase in the VE/VCO_2 slope, we observed higher values of $PetO_2$, VE/O_2, VE/CO_2 and lower values of $PetCO_2$ in PH patients with impaired RV function. These findings are consistent with those of Arena R. et al., who showed that $PetCO_2$ progressively decreased, while the VE/VCO_2 ratio and slope increased as PH disease severity worsened from mild to severe [17]. Moreover, differences in pulmonary function in chronic left HF and chronic right HF secondary to PH were also investigated. The right HF group was characterized by lower peak $PetCO_2$, and higher peak $PetO_2$, peak VE/VCO_2, and VE/VCO_2 slope during exercise [18].

In our study, the group with impaired RV function presented with higher PVR and lower $PetCO_2$. $PetCO_2$ was identified in a previous study as an independent predictor of PVR elevation in all PH patients [19].

Ventilatory inefficiency in PH patients can be measured as an increase in VE/O_2 and VE/CO_2. These parameters reflect the ventilatory requirement for eliminating the gas exchange parameters produced by aerobic metabolism. A higher VE/CO_2 describes the greater ventilatory requirement for eliminating CO_2 produced by aerobic metabolism. Our study showed higher values of these parameters in the group with impaired RV function. Previous studies have shown that in the anaerobic threshold, VE/CO_2 exhibited higher values in the PH group compared to HF patients [20] and healthy controls [21]. Moreover, the increase in VE/CO_2 during maximal incremental walking is associated with a significantly lower value of $PetCO_2$ [22]. By comparing initial CPET parameters of PH survivors (n = 87) with non-survivors (n = 29), Schwaiblmair et al. demonstrated significant differences in VE/O_2 (42.1 ±2.1 versus 56.9 ±2.6) and VE/CO_2 (47.5 ±2.2 versus 64.4 ±2.3) between these groups [15].

The reduction in O_2 pulse, which reflects cardiovascular response to exercise, is considered to be a typical CPET-response in PH patients [23]. We found similar values at rest among PH patients with a statistically significant increase of this parameter during exercise. Taking into consideration that this is an indicator of myocardial metabolic efficiency, and reflects stroke volume, it suggests more advanced heart deterioration in the group with impaired RV free wall motion. Moreover, it was previously shown that peak O_2 pulse with a combination of RV fractional area change identifies idiopathic PH patients at a particularly high risk of clinical deterioration [24].

RHC remains the gold standard in the process of PH diagnosis and evaluation of treatment effectiveness [2]. Nevertheless, the relationships between mPAP, PVR, CI and hypokinetic RV free wall in ROC curve analysis emphasizes the role of this non-invasive parameter in the monitoring of PH patients. We consider an increase in afterload, expressed in PVR, as the most important factor influencing right ventricular motion. Other factors such as RAP may be of importance, but because of the small number of subjects in the study population we were unable to demonstrate this phenomenon.

Conclusions

RV free wall motion abnormalities, assessed using echocardiography in PH patients, are found in those with more advanced disease. They are characterized by impaired ventilation in the CPET and more advanced haemodynamic abnormalities in RHC. The association between this parameter and prognosis requires validation in a larger population of patients.

Study limitations

Our study was based on retrospective analysis of a relatively small number of patients with various PH aetiologies. The assessment of RV free wall motion via TTE was based on visual evaluation.

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