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Original Article

Value of ST-T changes and heart rate variability in pulmonary heart disease during 24 h dynamic electrocardiography examination

Hairong Ye[1], Dandan Qiu[2], Feng Jia[3]

[1]*Department of Physical Examination, Jiangxi Chest Hospital, Nanchang 330006, Jiangxi Province, China;* [2]*Department of Surgery, Jiangxi Chest Hospital, Nanchang 330006, Jiangxi Province, China;* [3]*Department of Laboratory Medicine, Jiangxi Chest Hospital, Nanchang 330006, Jiangxi Province, China*

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Abstract: Purpose: This study was designed to analyze the value of 24 h dynamic electrocardiography (DCG) examination in the diagnosis of pulmonary heart disease (PHD). Methods: Ninety cases of patients with PHD were included as the observation group, and 50 cases of healthy patients were enrolled as the healthy group. Both groups received DCG examination. Results: The proportion of ST depression and elevation as well as the magnitude and duration of ST depression differed significantly between the observation and healthy group ($P < 0.05$), and the magnitude of ST elevation in patients of the observation group with decompensated heart failure was greater than that of patients with compensated cardiac function and the healthy group ($P < 0.05$). The incidence rates of sinus bradycardia (SB), ventricular premature beats (VPB), paroxysmal ventricular tachycardia (PVT), and ventricular fibrillation (VF) in patients in the observation group with decompensated heart failure were higher than those of patients with compensated cardiac function and the healthy group ($P < 0.05$). The differences in standard deviation of the NN (R-R) intervals (SDNN) and standard deviation of average NN intervals (SDANN) between the three groups were significant, and the root mean square of successive RR intervals (RMSSD) of patients in the observation group with decompensated heart failure were lower than those of the healthy group ($P < 0.05$). The differences in deceleration capacity (DC), left ventricular ejection fraction (LVEF), and heart rate variability (HRV) between the three groups were significant ($P < 0.05$). Conclusion: The results obtained by DCG examination can help clinical assessment of cardiac function in the decompensated or compensated stage, which can assist in judging the condition of PHD and guide clinical treatment.

Keywords: Pulmonary heart disease, 24 h ambulatory electrocardiography, examination, ST-T changes, heart rate variability

Introduction

Pulmonary heart disease (PHD) is specifically a heart disease occurring after chronic disease in the lungs or chest, or after chronic lesions in the blood vessels of the pulmonary circulation. It leads to pulmonary hypertension and causes increasing pressure in the right ventricle, which slowly induces right ventricular hypertrophy after loss of compensatory capacity, and ultimately leads to heart failure [1, 2].

Morphologic observations have shown that patients with PHD tend to have hypertrophy, dilatation, and widening of ventricular wall in the right ventricular outflow tract, while pathologic examination has shown a significant decrease in capillary density, an increase in interstitial components, and growth and thickening of myocardial fibers [3, 4]. These structural changes will occur before cardiac dysfunction, and the waveforms of the electrocardiogram (ECG) examination show significant abnormal changes before emergence of typical heart failure manifestations, including abnormal changes in P waves, QRS waves in the thoracic leads, and ST-T waveforms [5]. Conventional ECG has been used for the examination of PHD, but it has limitations in acquisition of information over a short detection time, and cannot

reflect the dynamic performance [6]. Dynamic electrocardiography (DCG) is a modified ECG, which is able to collect ECG waveforms 24/7 and obtain complete ECG information [7]. DCG exhibits a higher detection rate in a variety of cardiac diseases [8, 9].

In addition to the conventional ECG, pulmonary function tests, magnetic resonance imaging (MRI), and echocardiography are commonly used in the clinical examination of pulmonary heart disease; however, the effectiveness of DCG examination is not verified in large samples. Therefore, this study took this as the starting point and selected 90 patients with OHD in our hospital as subjects, and selected 50 healthy individuals as controls to analyze the value of DCG examination.

Materials and methods

Baseline data

Ninety patients with PHD from January 2018 to June 2020 were included as the observation group, and all of them met the diagnostic criteria of PHD [1]. Diagnostic criteria for compensatory stage of cardiac function: normal cardiac function and New York Heart Association (NYHA) grade 1-2. Diagnostic criteria for decompensatory stage of cardiac function: NYHA grade 2 or above. Fifty healthy controls were enrolled during the same period, all of whom were excluded from any disease by physical examination. Inclusion criteria: subjects had clear consciousness and normal cognitive function, and voluntarily signed the study informed consent form. The study obtained the ethical approval of the Jiangxi Chest Hospital. Exclusion criteria: subjects with connective tissue lesions, increased resistance to pulmonary circulation due to pulmonary thrombosis, idiopathic pulmonary hypertension, secondary pulmonary hypertension caused by congenital heart disease; structural and functional abnormalities of the heart due to cardiovascular system diseases.

Methods

Equipment: Dynamic ECG analysis system (Cardio Scan II Software Technology, DMS Ltd., USA), SCAIII stereocardiograph (Beijing Madix Medical Technology Co., Ltd.), M-type color Doppler ultrasound diagnostic instrument.

All patients received the DCG examination at the same time in the morning within 2 days of admission. The physicians strictly followed the operation procedures of ECG and instructed the patients to keep the precautions in mind during the test to minimize human errors. The ECG monitor was retrieved after 24 h and the data were collected through the DMS Cardio Scan II ambulatory ECG analysis system and all meaningless errors as well as artifacts were eliminated using the automated analysis operating system.

Outcome measurement

Baseline data: gender, age and condition were recorded.

ST-segment alteration: The proportion of ST-segment depression or elevation, the magnitude and duration of displacement in the two groups Criteria for ST segment abnormality [10]: horizontal or downward sloping depression of ST segment ≥ 0.10 mV in each lead, arched elevation > 0.30 mV in leads V1-V3, and > 0.10 mV in leads V4-V6 and limb leads; ST segment exhibits arched elevation.

Incidence of ventricular arrhythmias: the incidence of arrhythmia was distinguished as sinus bradycardia (SB), atrioventricular block (AVB), ventricular premature beats (VPB), accelerated idioventricular rhythm (AIR), paroxysmal ventricular tachycardia (PVT), and ventricular fibrillation (VF).

Heart rate turbulence (HRT): turbulence dynamicity (TD), turbulence onset (TO), and turbulence slope (TS) in the observation and healthy groups. TO neutral value = 0, TS neutral value = 2.5 ms/RRI; normal criteria: TO < 0 and TS > 2.5 ms/RRI; abnormal criteria: TO ≥ 0 with TS ≤ 2.5 ms/RR interval [11].

Heart rate variability (HRV) time-domain index: standard deviation of the NN (R-R) intervals (SDNN), standard deviation of average NN intervals (SDANN), and root mean square of successive RR intervals (RMSSD) in the observation and healthy groups. Normal range: SDNN: (141.7±29.2) ms, SDANN: (130.9±28.3) ms, RMSSD: (39.0±15.0) ms.

Deceleration capacity (DC) was recorded by the dynamic ECG analysis system. Abnormal crite-

Table 1. Comparison of baseline data between the two groups ($\bar{x}$±s)/[n (%)]

Data		Observation group (n = 90)	Healthy group (n = 50)	t/X^2	*P*
Gender	Male	49 (54.44)	28 (56.00)	0.031	0.859
	Female	41 (45.56)	22 (44.00)		
Cardiac function	Compensatory phase	43(47.78)	/	/	/
	Decompensatory phase	47(52.22)	/	/	/
Age (years)		52.43±6.92	50.94±7.12	1.208	0.229
MPAD (mm)		25.76±3.19	19.15±2.37	12.801	< 0.001
RVD (mm)		26.43±3.32	20.18±4.21	9.679	< 0.001
PASP (mmHg)		52.16±5.18	46.28±4.32	6.815	< 0.001

Table 2. Comparison of the proportion of ST segment depression or elevation in the two groups [n (%)]

Group		Number of cases	ST-segment depression	ST-segment elevation
Observation group	Cardiac function compensated group	43	20 (46.51)	5 (11.63)
	Cardiac function decompensated group	47	40 (85.11)	16 (34.04)
Healthy group		50	5 (10.00)	4 (8.00)
X^2			5.182	4.827
P			0.006	0.037

ria [12]: DC ≤ 4.5 ms, where low risk was 4.5-10 ms, medium risk was 2.5-4.4 ms, and high risk was < 2.5 ms.

Left ventricular ejection fraction (LVEF) and HRV were measured by echocardiography.

Statistical methods

Statistical analysis was performed with SPSS 23.0. Counted data were expressed as [n (%)] and compared by X^2 test. Measured data were expressed as ($\bar{x}$±s) and examined by t test. Multi-point comparisons were analyzed with ANVOA with post hoc F test. Graphs were produced with Graphpad Prism 8. $P < 0.05$ was considered significant.

Results

Baseline data

There was no difference in the proportion of males and females, and mean age between the two groups ($P > 0.05$). The proportion of patients with compensated cardiac function was 47.78%, and 52.22% in the observation group. The Main pulmonary artery diameter (MPAD), Right ventricular diameter (RVD), and Pulmonary artery systolic pressure (PASP) in the observation group were higher than those in the healthy group ($P < 0.05$) (**Table 1**).

ST segment changes

The proportions of ST-segment depression and elevation in patients with decompensated heart failure were higher than those in patients with compensated cardiac function and healthy group ($P < 0.05$), and the proportion of ST-segment depression in patients with compensated cardiac function was higher than that in healthy group ($P < 0.05$) (**Table 2**). The magnitude and duration of ST-segment depression in patients with decompensated heart failure were greater than those in patients with compensated cardiac function and the healthy group ($P < 0.05$), and the magnitude and duration of ST-segment depression in patients with compensated cardiac function were greater than those in the healthy group ($P < 0.05$). The magnitude of ST-segment elevation in patients with decompensated heart failure was greater than that in patients with compensated cardiac function and the healthy group ($P < 0.05$) (**Figures 1**, **2**).

Incidence of arrhythmia

The incidence rates of SB, VPB, PVT, and VF in patients with decompensated heart failure were higher than those in patients with compensated cardiac function and the healthy group ($P < 0.05$), and the difference between

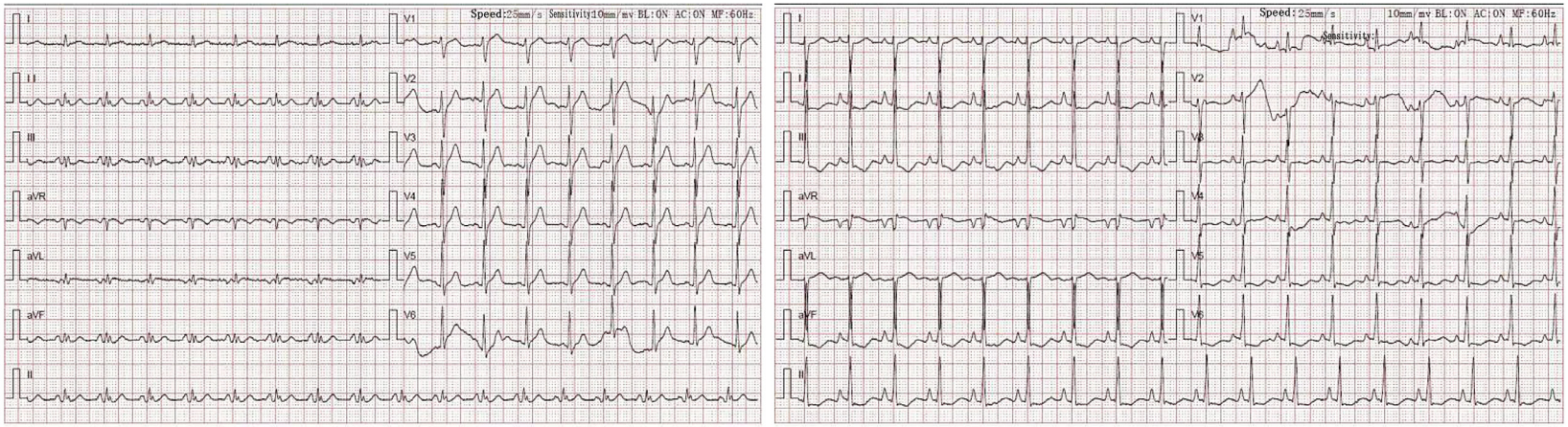

Figure 1. Compensatory dynamic electrocardiography of pulmonary heart disease. The left picture shows sinus tachycardia with low voltage and P pulmonale; the right picture shows sinus rhythm with P pulmonale and right ventricular hypertrophy with strain.

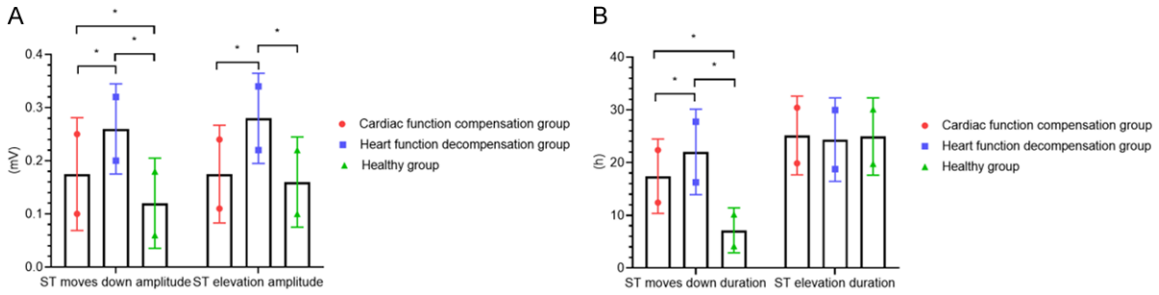

Figure 2. ST-segment alterations. The magnitude (A), and duration (B) of ST-segment. *$P < 0.05$.

Table 3. Comparison of the incidence of arrhythmias [n (%)]

Group		Number of cases	SB	AVB	VPB	AIR	PVT	VF
Observation group	Cardiac function compensated group	43	7 (16.28)	2 (4.65)	10 (23.26)	2 (4.65)	1 (2.33)	1 (2.33)
	Cardiac function decompensated group	47	17 (36.17)	3 (6.38)	34 (72.34)	3 (6.38)	8 (17.02)	10 (21.28)
Healthy group		50	6 (12.00)	2 (4.00)	5 (10.000)	0 (0.00)	0 (0.00)	2 (4.00)
X^2			4.856	0.628	5.027	0.521	4.968	4.383
P			0.013	0.313	0.008	0.415	0.017	0.026

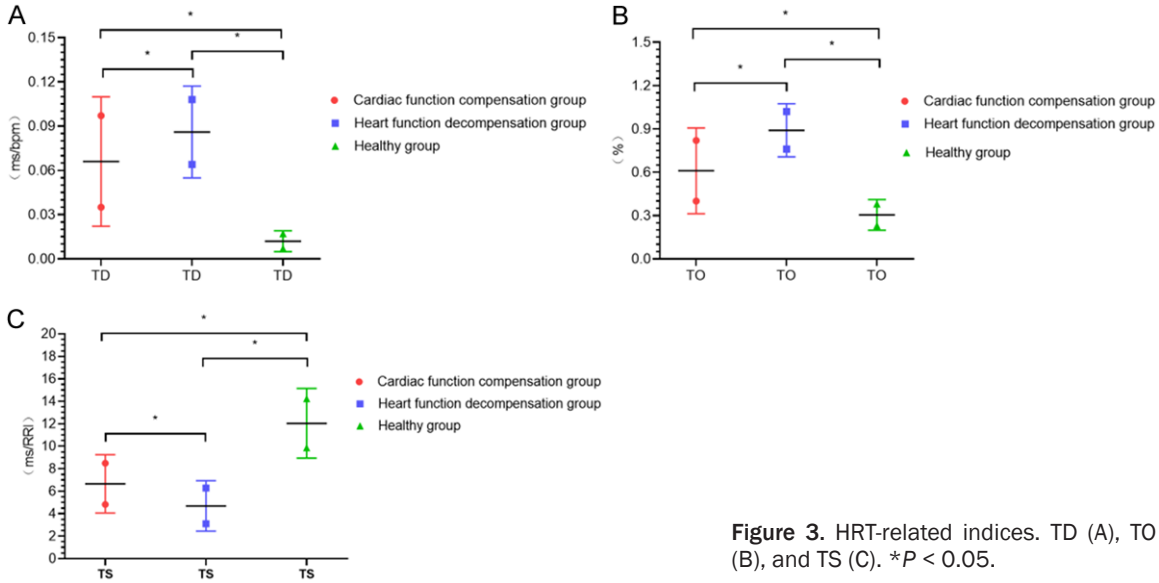

Figure 3. HRT-related indices. TD (A), TO (B), and TS (C). *$P < 0.05$.

the patients with compensated cardiac function and the healthy group was not significant ($P > 0.05$). There was no significant difference in the incidence rates of AVB and AIR between the three groups ($P > 0.05$) (**Table 3**).

HRT-related indicators

Patients with decompensated heart failure had higher TD and TO and lower TS than patients with compensated cardiac function and the healthy group ($P < 0.05$), and patients with compensated cardiac function had higher TD and TO and lower TS than the healthy group ($P < 0.05$) (**Figure 3**).

HRV-related indicators

The SDNN and SDANN in patients with decompensated heart failure were higher than those in patients with compensated cardiac function and the healthy group ($P < 0.05$), and the SDNN and SDANN in patients with compensated cardiac function were lower than those in the

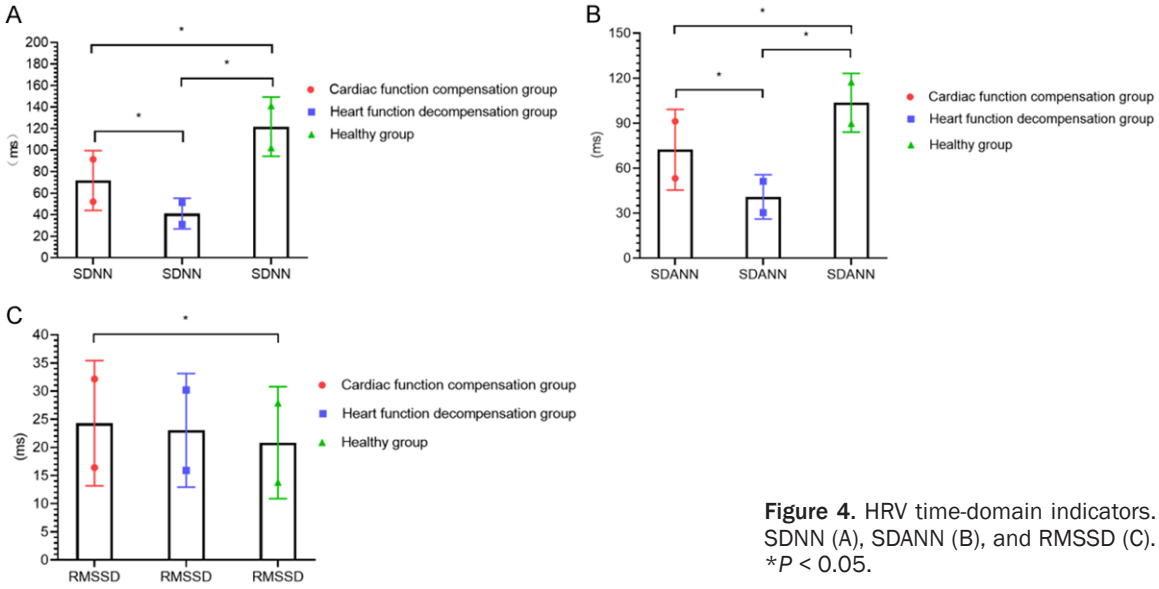

Figure 4. HRV time-domain indicators. SDNN (A), SDANN (B), and RMSSD (C). *$P < 0.05$.

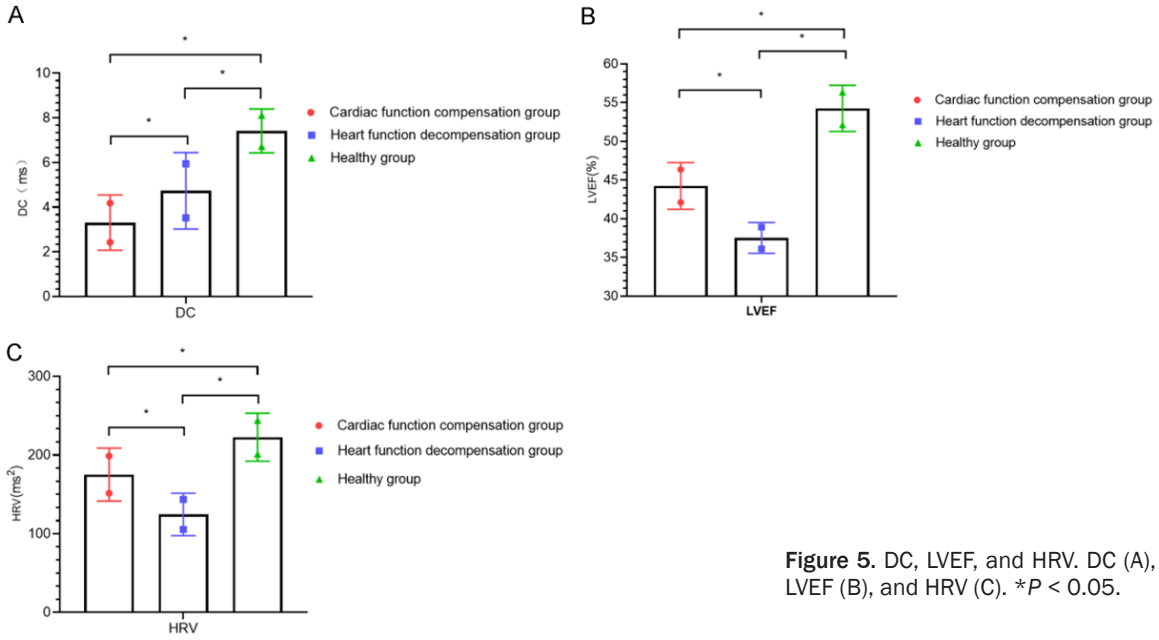

Figure 5. DC, LVEF, and HRV. DC (A), LVEF (B), and HRV (C). *$P < 0.05$.

healthy group ($P < 0.05$). The RMSSD of patients with decompensated heart failure in the observation group were lower than those of the healthy group ($P < 0.05$). The difference with patients with compensated cardiac function was not significant ($P > 0.05$), and the RMSSD did not differ between patients with compensated cardiac function and healthy group ($P > 0.05$) (**Figure 4**).

DC, LVEF, HRV

The DC, LVEF, and HRV in patients with decompensated heart failure were lower than those in patients with compensated cardiac function and the healthy group, and the DC, LVEF, and HRV in patients with compensated cardiac function were lower than those in the healthy group ($P < 0.05$) (**Figure 5**).

Discussion

The occurrence of PHD is closely related to economic level, living environment, quality of medical services, and education level [13]. Statistics show that more than 35% of hospitalized cardiac patients developed chronic PHD [14]. PHD in the decompensated phase leads to a variety

of complications such as electrolyte imbalance, acid-base imbalance, pulmonary encephalopathy, cardiac arrhythmias, and gastrointestinal bleeding, which significantly increases the mortality rate in patients [15, 16]. Therefore, timely diagnosis of the compensated phase in PHD and prevention of its transformation to the decompensated phase are crucial to improve the prognosis of patients, which requires a good and accurate clinical diagnosis of PHD at an early stage.

The DCG in this study could obtain the continuous 24-h real-time ECG data of patients, and record the cardiac electrical changes when patients ate and lived, and when cardiac symptoms occurred, providing a full picture of 24-hour cardiac changes of patients [17]. Previous studies have confirmed the value of DCG for the qualitative and quantitative diagnosis of arrhythmias, myocardial ischemia, and functional status of pacemakers [18, 19]. The abnormal ECG waveforms of myocardial ischemia are charactered by ST-T segment alteration, and there is a correlation between the abnormal manifestations and the site of injury [20]. Common ST-T segment changes also occur accompanied by heart rate changes, demonstrating arrhythmia [21]. ST-T changes are characterized by segmental changes, corresponding lead changes, and dynamic changes [22]. The present study showed that patients with PHD with cardiac function in the decompensated phase had more changes in ST-segment depression and elevation and greater magnitude and duration of depression compared to patients in the compensated phase, suggesting that ST-segment changes can be considered as an indicator to identify patients with PHD who are in compensated or decompensated cardiac function. It has been found that heart diseases are associated with varying degrees of arrhythmia that reflects the severity of PHD to some extent [23]. The occurrence of arrhythmias can also be used to determine the compensated or decompensated stages. The results of this study showed that patients in the decompensated phase were more likely to have atrial fibrillation, ventricular tachycardia, and premature ventricular contractions compared with patients in the compensated phase, suggesting that these three types of arrhythmias can be used as indicators of compensated or decompensated cardiac function in PHD.

DC can be used to quantitatively assess vagal tone in patients, thus aiding in clinical screening of high-risk groups in patients with PHD. Studies using DC metrics to detect vagal tone levels have shown high sensitivity and specificity [24]. The results of this study showed positive correlation between DC and LVEF and HRV values, and the DC, LVEF, and HRV values of patients in decompensated stage were lower than those of compensated patients and healthy individuals, and were also lower in compensated patients than in the healthy group. The vagus nerve is the decelerating nerve of the heart and can dominate autonomic changes in cardiac function; heart rate deceleration increases/decreases when vagus nerve function is enhanced or decreased [25]. LVEF is considered to be an independent factor for the occurrence of sudden cardiac death, and it is generally believed that lower LVEF levels are associated with a higher risk of sudden cardiac death [26]. However, has been found that LVEF has limitations as a predictor of sudden cardiac death and accurate predictions are not obtained in all patients. HRV is a quantitative indicator of autonomic function in the sinus node [27]. In this study, SDNN and SDANN in patients in decompensated phase were lower than those in compensated phase and healthy controls, and were lower in patients in compensated phase than in the healthy group ($P < 0.05$). Moreover, compared with compensated patients, the RMSSD of patients in decompensated phase did not differ significantly, but was lower than that of healthy group, indicating that patients with cardiac function in the decompensated phase are more likely to have adverse cardiac events, suggesting that the determination of HRV time-domain indicators can somewhat assist in determining whether cardiac function is in the compensated phase. SDNN reflects the overall changes in autonomic function within 24 h, RMSSD reflects the changes in vagal tone within 24 h [28]. It was also found that the SDNN and SDANN in patients with PHD in the decompensated phase of cardiac function were significantly lower than those in patients in the compensated phase [29].

In conclusion, the results obtained by DCG examination can help clinical identification of

cardiac function in the decompensated or compensated stage, which can assist in judging the condition of PHD and guide symptomatic treatment. However, this study was superficial, and only a few simple indicators were analyzed, while a retrospective analysis and prospective, larger sample size and more in-depth analytical studies should be conducted in the future to explore the application value of DCG in PHD.

Disclosure of conflict of interest

None.

Address correspondence to: Feng Jia, Department of Laboratory Medicine, Jiangxi Chest Hospital, No. 346, Dieshan Road, Donghu District, Nanchang 330006, Jiangxi Province, China. Tel: +86-13607045778; E-mail: jiafeng78@21cn.com

References

[1] Aubry A, Paternot A and Vieillard-Baron A. Cœur pulmonaire [Cor pulmonale]. Rev Mal Respir 2020; 37: 257-266.

[2] Rubin LJ. Cor pulmonale revisited. From ferrer and harvey to the present. Ann Am Thorac Soc 2018; 15 Suppl 1: S42-S44.

[3] Mandoli GE, Sciaccaluga C, Bandera F, Cameli P, Esposito R, D'Andrea A, Evola V, Sorrentino R, Malagoli A, Sisti N, Nistor D, Santoro C, Bargagli E, Mondillo S, Galderisi M and Cameli M. Cor pulmonale: the role of traditional and advanced echocardiography in the acute and chronic settings. Heart Fail Rev 2021; 26: 263-275.

[4] Zhang F, Cao Q and Zuo X. Acute cor pulmonale in acute respiratory distress syndrome. Zhonghua Wei Zhong Bing Ji Jiu Yi Xue 2017; 29: 272-275.

[5] Chin CWL, Everett RJ, Kwiecinski J, Vesey AT, Yeung E, Esson G, Jenkins W, Koo M, Mirsadraee S, White AC, Japp AG, Prasad SK, Semple S, Newby DE and Dweck MR. Myocardial fibrosis and cardiac decompensation in aortic stenosis. JACC Cardiovasc Imaging 2017; 10: 1320-1333.

[6] Kania M, Maniewski R, Zaczek R, Kobylecka M, Zavala-Fernandez H, Królicki L and Opolski G. Optimal ECG lead system for exercise assessment of ischemic heart disease. J Cardiovasc Transl Res 2020; 13: 758-768.

[7] Oehler A and Chiovaro J. The electrocardiogram following dynamic cardiomyoplasty. J Gen Intern Med 2017; 32: 223-224.

[8] Brockmeier K, Nazal R and Sreeram N. The electrocardiogram of the neonate and infant. J Electrocardiol 2016; 49: 814-816.

[9] Maillot N, Guenancia C, Yameogo NV, Gudjoncik A, Garnier F, Lorgis L, Chagué F and Cottin Y. Impact of the dynamic and static component of the sport practised for electrocardiogram analysis in screening athletes. Scand J Med Sci Sports 2018; 28: 575-584.

[10] Hanna EB, LeLorier PA and Glancy DL. Dynamic ST-segment abnormality. Am J Cardiol 2018; 121: 390-391.

[11] Blesius V, Schölzel C, Ernst G and Dominik A. HRT assessment reviewed: a systematic review of heart rate turbulence methodology. Physiol Meas 2020; 41: 08tr01.

[12] Hamm W, von Stülpnagel L, Klemm M, Baylacher M, Rizas KD, Bauer A and Brunner S. Deceleration capacity of heart rate after acute altitude exposure. High Alt Med Biol 2018; 19: 299-302.

[13] Sakao S. Chronic obstructive pulmonary disease and the early stage of cor pulmonale: a perspective in treatment with pulmonary arterial hypertension-approved drugs. Respir Investig 2019; 57: 325-329.

[14] Mekontso Dessap A, Boissier F, Charron C, Bégot E, Repessé X, Legras A, Brun-Buisson C, Vignon P and Vieillard-Baron A. Acute cor pulmonale during protective ventilation for acute respiratory distress syndrome: prevalence, predictors, and clinical impact. Intensive Care Med 2016; 42: 862-870.

[15] Creel-Bulos C, Hockstein M, Amin N, Melhem S, Truong A and Sharifpour M. Acute Cor pulmonale in critically ill patients with covid-19. N Engl J Med 2020; 382: e70.

[16] Alajaji W, Baydoun A, Al-Kindi SG, Henry L, Hanna MA and Oliveira GH. Digoxin therapy for cor pulmonale: a systematic review. Int J Cardiol 2016; 223: 320-324.

[17] Zhang Q, Fu L and Gu L. A cascaded convolutional neural network for assessing signal quality of dynamic ECG. Comput Math Methods Med 2019; 2019: 7095137.

[18] Yaghmaie N, Maddah-Ali MA, Jelinek HF and Mazrbanrad F. Dynamic signal quality index for electrocardiograms. Physiol Meas 2018; 39: 105008.

[19] Abdul-Kadir NA, Mat Safri N and Othman MA. Dynamic ECG features for atrial fibrillation recognition. Comput Methods Programs Biomed 2016; 136: 143-150.

[20] Khan MS, Arif AW and Doukky R. The prognostic implications of ST-segment and T-wave abnormalities in patients undergoing regadenoson stress SPECT myocardial perfusion imaging. J Nucl Cardiol 2020; [Epub ahead of print].

[21] Xiao L, Bai T, Zeng J, Yang R and Yang L. Nonalcoholic fatty liver disease, a potential risk factor of non-specific ST-T segment changes: data

from a cross-sectional study. PeerJ 2020; 8: e9090.
[22] Okada JI, Fujiu K, Yoneda K, Iwamura T, Washio T, Komuro I, Hisada T and Sugiura S. Ionic mechanisms of ST segment elevation in electrocardiogram during acute myocardial infarction. J Physiol Sci 2020; 70: 36.
[23] Bayoumi M, Kahn A, Snyder DL, Menias CO and Vela MF. Severe achalasia causing cor pulmonale. Am J Gastroenterol 2020; 115: 1159.
[24] Kong ZB, Wang XD, Shen SR, Liu H, Zhou L, Chen B, Chen Z and Yin SJ. Risk prediction for arrhythmias by heart rate deceleration runs in patients with chronic obstructive pulmonary disease. Int J Chron Obstruct Pulmon Dis 2020; 15: 585-593.
[25] Wang X, Jiang Z, Chen B, Zhou L, Kong Z, Zuo S, Liu H and Yin S. Cardiac autonomic function in patients with acute exacerbation of chronic obstructive pulmonary disease with and without ventricular tachycardia. BMC Pulm Med 2016; 16: 124.
[26] Zaman S, Goldberger JJ and Kovoor P. Sudden death risk-stratification in 2018-2019: the old and the new. Heart Lung Circ 2019; 28: 57-64.
[27] Jiménez Morgan S and Molina Mora JA. Effect of heart rate variability biofeedback on sport performance, a systematic review. Appl Psychophysiol Biofeedback 2017; 42: 235-245.
[28] Siecinski S, Kostka PS and Tkacz EJ. Time domain and frequency domain heart rate variability analysis on gyrocardiograms. Annu Int Conf IEEE Eng Med Biol Soc 2020; 2020: 2630-2633.
[29] Qian Z, Fan A, Dawa, Dawaciren and Pan B. Retrospective cohort analysis of heart rate variability in patients with high altitude pulmonary hypertension in Tibet. Clin Cardiol 2020; 43: 298-304.