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Bendopnea Predicts Right Ventricular Energy Failure in Patients with Pulmonary Hypertension

Bendopne Pulmoner Hipertansiyon Hastalarında Sağ Ventriküler Enerji Yetersizliğini Öngördürür

ABSTRACT

Objective: The development of right ventricular failure has a significant adverse prognostic impact on the course of pulmonary hypertension. Right ventricular energy failure has been shown to double the mortality of pulmonary hypertension even after correction for many established risk predictors. We hypothesize that bendopnea may indicate right ventricular energy failure in patients with pulmonary hypertension.

Methods: We prospectively enrolled patients with pulmonary hypertension who were admitted to our pulmonary hypertension outpatient clinic between January 2021 and June 2021. Bendopnea was assessed by asking patients to bend forward and report any shortness of breath within 30 seconds. Routine physical examination, laboratory tests, echocardiography, and right heart catheterization parameters were collected.

Results: A total of 167 patients were enrolled into the study. Bendopnea and right ventricular energy failure was present in 79 (47.3%) and 43 (25.7%) patients, respectively. Bendopnea accurately predicted the presence of right ventricular energy failure (area under the curve, 0.667; 95% CI, 0.574-0.760; $P < 0.001$) and had a significantly superior diagnostic power compared with many other symptoms and signs.

Conclusions: Our study shows that bendopnea predicts right ventricular energy failure in patients with pulmonary hypertension and can be added to our physical examination armamentarium as an easy, rapid, and noninvasive prognostic tool.

Keywords: Bendopnea, dyspnea, heart failure, hemodynamics, pulmonary hypertension

ÖZET

Amaç: Sağ ventrikül yetersizliği gelişimi pulmoner hipertansiyon (PH) seyrinde önemli bir olumsuz etkiye sahiptir. Sağ ventriküler enerji yetersizliğinin (SVEY) pek çok yerleşik risk öngördürücüsüne karşı düzeltme yapıldıktan sonra dahi PH mortalitesini iki kat arttığı gösterilmiştir. Biz, PH hastalarında bendopnenin SVEY'i öngördüreceğini kurguladık.

Yöntem: Ocak 2021 ile Haziran 2021 tarihleri arasında PH polikliniğimize başvuran hastalar ileriye dönük olarak çalışmaya alındı. Bendopne hasta öne eğildiğinde 30 saniye içerisinde nefes darlığı oluşmasına göre değerlendirildi. Rutin fizik muayene, laboratuvar testleri, ekokardiyografi ve sağ kalp kateterizasyonu verileri toplandı.

Bulgular: Çalışmaya 167 hasta alındı. Bendopne ve SVEY hastaların sırasıyla 79 (%47,3) ve 43'ünde (%25,7) gözlendi. Bendopne SVEY'i tutarlı bir şekilde öngördü (eğri altı alan, 0,667; %95 güvenlik aralığı, 0,574 ila 0,760; $P < 0,001$) ve pek çok diğer belirti ve bulguya kıyasla daha üstün tanısal güce sahipti.

Sonuç: Çalışmamız bendopnenin PH hastalarında SVEY'i öngördürdüğünü ve fizik muayene cephanemize kolay, hızlı ve girişimsel olmayan bir ek prognostik gereç olarak eklenebileceğini göstermektedir.

Anahtar Kelimeler: Bendopne, dispne, kalp yetersizliği, hemodinami, pulmoner hipertansiyon

Pulmonary hypertension (PH) is a mortal disease with an approximate prevalence of 1% worldwide. Although multiple etiologies can cause PH, its progression follows a common path in which increased pulmonary vascular afterload ultimately results in right ventricular (RV) failure. Right ventricular failure further aggravates symptoms and has a significant adverse prognostic impact.[1] However, the occurrence of RV failure

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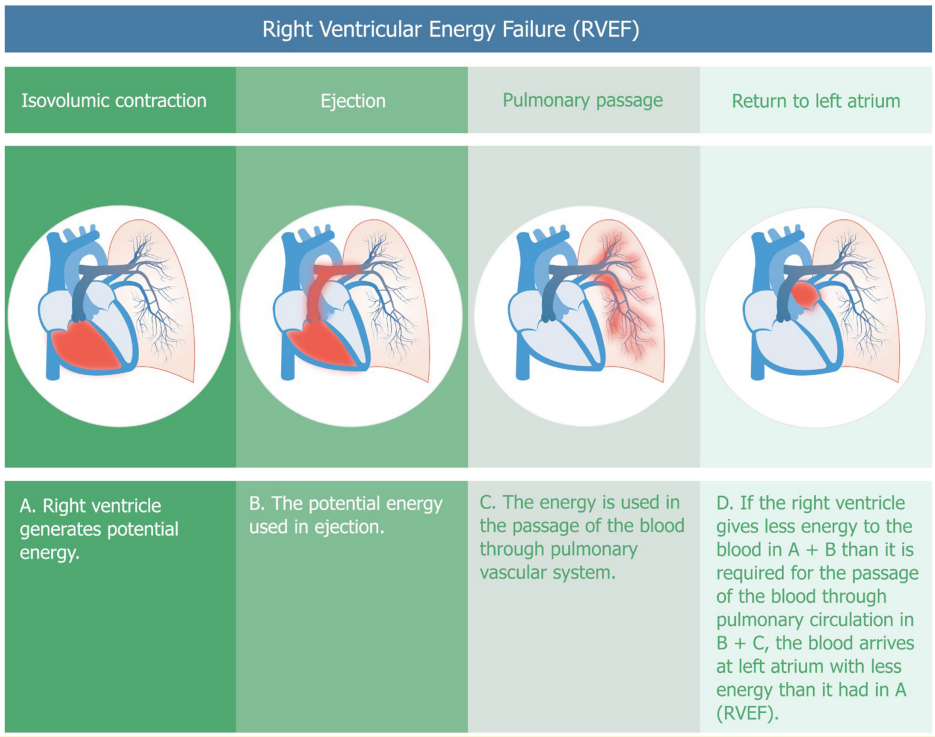

Figure 1. The concept of RV energy failure. The RV transfers its mechanical energy during contraction to the RV stroke volume, which is dissipated during the transpulmonary travel of the blood through pulmonary vascular resistance. As RV energy output is normally greater than the energy lost in pulmonary circulation, the blood arriving to the left atrium has a higher hydraulic energy than the blood in the RV at the beginning of RV systole. As the transferred volume is constant (stroke volume), hydraulic energy (pressure per volume) can be approximated as pressure. Therefore, when left atrial pressure is equal or lower compared with RV end-diastolic pressure (which is practically equal to the right atrial pressure), it can be deduced that RV energy output fails to exceed the energy lost in the pulmonary circulation. This is defined as right ventricular energy failure (reproduced from Reference 5 with permission from Elsevier © 2023). RV, right ventricular.

depends not solely on pulmonary vascular or RV contractile status in isolation but rather on their interaction, namely RV–pulmonary arterial coupling.[2-4] To this end, we have recently introduced the term RV energy failure (RVEF), which defines the inability of RV to generate enough energy output relative to the corresponding pulmonary vascular load (Figure 1), and shown it has a strong prognostic power even after correction for many established risk predictors.[5] Despite its prognostic utility, however, its identification needs invasive evaluation.

Bendopnea is another recently introduced term defined as shortness of breath while bending forward. Although it is first defined in patients with heart failure (HF),[6] it has also been reported in patients with PH and associated right-sided HF.[1,7] Although the mechanism underlying bendopnea and whether its mechanism in the right- and left-sided HF is the same are not clear, an increase in ventricular filling pressures due to an increase in intrathoracic or intraabdominal pressure was proposed as the primary mechanism. We hypothesize that these mechanisms may uncover already diseased ventriculoarterial coupling in the pulmonary circulation and therefore may be a noninvasive indication of RVEF in patients with PH.

In this study, we aimed to evaluate whether bendopnea can be used as a sign of RVEF.

ABBREVIATIONS

AUC	Area Under Curve
CRP	C-Reactive Protein
HF	Heart Failure
NT-Probnp	N-Terminal Pro-Brain Natriuretic Peptide
PCWP	Pulmonary Capillary Wedge Pressure
PH	Pulmonary Hypertension
RV	Right Ventricular
RVEF	RV Energy Failure

Materials and Methods

The study was undertaken at Marmara University Pendik Training and Research Hospital, a tertiary center for PH. Ethical committee approval was obtained from Ethics Commitee of Marmara University (Approval Number: 09.2021.1111, Date: 08.10.2021), and the study was undertaken in accordance with the declaration of Helsinki. We prospectively enrolled patients with PH, who were admitted to our PH outpatient clinic between January 2021 and June 2021, were under stable treatment, and had a plan for or already had right heart catheterization within the last 3 months. The patients who could not perform the bendopnea test for orthopedic or other reasons, who had clinical deterioration that required a change in their treatment within the last 3 months, and whose echocardiographic or hemodynamic data could not be obtained were excluded from the study.

All patients were evaluated by a multidisciplinary team consisting of cardiologists, pulmonologists, a thoracic surgeon, a rheumatologist, and a radiologist with pulmonary vascular imaging expertise. Baseline demographic data, symptoms (dyspnea, orthopnea, hemoptysis, weight gain, abdominal distention, syncope, and angina), and signs (the presence or absence of an accentuated S_2, S_3, any murmur, hepatomegaly, ascites, and jugular venous distension) related to PH were recorded. After baseline physical examination, the patients were placed on a comfortable chair for the assessment of bendopnea and asked to bend forward and report any shortness of breath within 30 seconds. The patients were also questioned about the severity (mild, moderate, or severe) of bendopnea to assess the impact of the symptom on their daily lives. Bendopnea was classified as mild if the patient develops shortness of breath while putting on or tying shoes, moderate if the patient needs someone's help, and severe limitation if could not do this action at all.[8]

Blood samples were taken at the time of the first visit, and routine laboratory tests included creatinine, N-terminal pro-brain natriuretic peptide (NT-proBNP), cardiac troponin T, hemogram, and C-reactive protein (CRP). A 6-minute walk test was performed in each patient. A comprehensive echocardiographic examination was performed in accordance with the American Society of Echocardiography recommendations[9,10] using an Epiq 7 machine (Philips Medical Systems, Andover, Mass, USA) equipped with a 3.5 MHz transducer.

A Swan-Ganz catheter (Edwards Lifesciences, Irvine, Calif, USA) was used to perform right heart catheterization via the right jugular vein. The calibration of the system was controlled before recordings with a square-wave test. All pressure tracings were evaluated by visual examination for physiologic accuracy, and the end-expiratory pressure values were taken. Cardiac output was measured using the indirect Fick method. Right ventricular energy failure was defined as the pulmonary capillary wedge pressure (PCWP) equal to or lower than the mean right atrial pressure.

Statistical Analysis

Statistical Package for the Social Sciences (SPSS) (version 26.0; SPSS Inc., Chicago, Ill, USA) statistical software was used for all analyses. Continuous variables were expressed as mean ± SD or median (interquartile range, IQR). Categorical variables were expressed as numbers (percentages). The normality of continuous variables was assessed with the Kolmogorov–Smirnov test. The comparison of independent variables according to the presence or absence of bendopnea was performed with chi-square, Fischer's exact test, Student's *t*-test, Mann–Whitney *U*, Kruskal–Wallis test, and 1-way analysis of variance, as appropriate. The predictive value of bendopnea for RVEF was calculated and compared with other symptoms using receiver operating characteristics curve analysis. For all statistics, a *P* value below 0.05 was considered significant.

Results

During the study period, a total of 167 patients were enrolled into the study. There were 102 (61.1%) patients with group I PH, 38 (22.8%) with group II PH and 27 (16.2%) with group IV PH. Among group I patients, there were 36 (35.3%) patients with idiopathic pulmonary arterial hypertension, 27 (26.5%) patients with connective tissue disease, 36 (35.3%) patients with congenital heart disease, and 3 (2.9%) patients with venoocclusive disease. Among group IV patients, 15 (57.7%) had been operated on for chronic thromboembolic PH, 6 (22.2%) had been deemed as inoperable, and 6 (23.1%) had undergone pulmonary balloon angioplasty. In the whole population, bendopnea was present in 79 out of 167 patients (47.3%).

Baseline characteristics according to the presence or absence of bendopnea are shown in Table 1. Baseline demographics were similar between patients with and without bendopnea. In general, symptoms and signs were more frequent in patients with bendopnea compared to those without bendopnea. The most common symptom was exertional dyspnea, which was present in 127 patients (79%), which is followed by orthopnea in 74 (44.3%) and palpitation in 70 (41.9%). The most common finding was an accentuated S_2, which was present in 119 patients (71.3%), followed by tricuspid regurgitation murmur in 114 (68.3%) and jugular venous distention in 100 (59.9%). Bendopnea was present in 79 (47.3%) patients, 36 (21.5%), 31 (18.6%), and 12 (7.2%) of whom reported mild, moderate, and severe limitation, respectively.

Echocardiographic and hemodynamic data are shown in Table 2. Ventricular filling pressures were significantly higher in patients with bendopnea compared to those without bendopnea. In mild, moderate, and severe bendopnea subgroups, PCWP and right atrial pressures were 9 (4), 12 (11), and 18.5 (11) mmHg ($P < 0.001$ for intergroup comparison) and 10 (7), 14 (7) and 14 (11) mmHg ($P < 0.001$ for intergroup comparison), respectively. Right ventricular energy failure was present in 43 patients (25.7%) in the whole cohort. It was present in 31 (39.2%) and 12 (13.6%) patients with and without bendopnea, respectively ($P < 0.001$). Right ventricular energy failure was present in 33.3% (12/36), 48.4% (15/31), and 33.3% (4/12) of the patients in mild, moderate, and severe bendopnea subgroups ($P = 0.413$ for intergroup comparison).

Bendopnea accurately predicted the presence of RVEF (area under curve [AUC], 0.667; 95% CI, 0.574-0.760; $P < 0.001$) and had a significantly superior diagnostic power compared with paroxysmal nocturnal dyspnea (AUC difference, 0.155; $P = 0.002$), hemoptysis (AUC difference, 0.121; $P = 0.006$), weight gain (AUC difference, 0.121; $P = 0.010$), abdominal distention (AUC difference, 0.109; $P = 0.013$), syncope (AUC difference, 0.120; $P = 0.005$) and angina (AUC difference, 0.159; $P < 0.001$). It also had a significantly superior diagnostic power compared with physical examination findings, including accentuated S_2 (AUC difference, 0.083; $P = 0.044$), the presence of S_3 (AUC difference, 0.149; $P = 0.001$), clubbing (AUC difference, 0.102; $P = 0.025$), rales (AUC difference, 0.220; $P < 0.001$), rhonchi (AUC difference, 0.167; $P < 0.001$), abdominal distention (AUC difference, 0.110; $P = 0.014$). Bendopnea only failed to exceed the diagnostic power of orthopnea in symptoms (AUC difference, 0.270; $P = 0.586$), and tricuspid regurgitation murmur (AUC difference, 0.047; $P = 0.326$) and jugular venous distention in signs (AUC difference, –0.072; $P = 0.086$) in RVEF prediction, but the size of the study was not enough to prove the indifference.

Table 1. Baseline Characteristics*

	All (n=167)	Bendopnea (n=79)	No Bendopnea (n=88)	*P*
Age, years	52 (25)	50 (26)	54 (25)	0.714
Female, %	110 (65.8)	55 (69.6)	55 (62.5)	0.333
BMI, kg/m^2	27 (7.84)	27 (7.70)	27.2 (7.79)	0.383
Heart rate, beats/min	80 (20)	78 (18)	80 (22)	0.875
SBP, mmHg	125 ± 27	126 ± 21	129 ± 23	0.685
Comorbidities				
Hypertension, n (%)	69 (41.3)	36 (45.6)	33 (37.5)	0.290
Diabetes, n (%)	23 (13.8)	14 (17.7)	9 (10.2)	0.161
Dyslipidemia, n (%)	25 (15)	14 (17.7)	11 (12.5)	0.345
CAD, n (%)	26 (15.6)	9 (11.4)	17 (19.3)	0.158
CKD, n (%)	13 (7.8)	8 (10.1)	5 (5.7)	0.284
Thyroid disorders, n (%)	39 (23.4)	16 (20.3)	23 (26.1)	0.370
Symptoms and the signs of PH				
Orthopnea, n (%)	74 (44.3)	50 (63.3)	24 (27.3)	<0.001
PND, n (%)	28 (16.8)	18 (22.8)	10 (11.4)	0.049
Exertional dyspnea, n (%)	127 (79)	62 (78.5)	65 (79.5)	0.866
Hemoptysis, n (%)	8 (4.7)	6 (7.5)	2 (2.2)	0.151
Weight gain, n (%)	11 (6.6)	7 (8.9)	4 (4.5)	0.353
Abdominal distention, n (%)	17 (10.2)	14 (17.7)	3 (3.4)	0.002
Syncope, n (%)	4 (2.4)	4 (5.1)	0	<0.001
Accentuated S_2, n (%)	119 (71.3)	66 (83.5)	53 (60.2)	<0.001
S_3, n (%)	15 (9)	13 (15.2)	3 (3.4)	0.008
TR murmur, n (%)	114 (68.3)	64 (81)	50 (56.8)	<0.001
Rales, n (%)	17 (10.2)	11 (13.9)	6 (6.8)	0.129
Hepatomegaly, n (%)	50 (29.9)	41 (51.9)	9 (10.2)	<0.001
JVD, n (%)	100 (59.9)	63 (79.7)	37 (42)	<0.001
eJVP, mmHg	8 (8)	10 (5)	4 (4)	<0.001
Functional class				
WHO class I, n (%)	62 (37.1)	25 (31.6)	37 (42)	0.224
WHO class II, n (%)	78 (46.7)	41 (51.9)	37 (42)	
WHO class III, n (%)	25 (15)	11 (13.9)	14 (15.9)	
WHO class IV, n (%)	2 (1.2)	2 (2.5)	0	
6MWT, m	360 ± 109	345 ± 105	374 ± 112	0.081
Laboratory parameters				
GFR, mL/min/m^2	106 ± 43	108 ± 52	105 ± 51	0.679
Hemoglobin, g/dL	13.3 ± 1.8	13.5 ± 2.1	13.1 ± 1.5	0.107
CRP, mg/L	1.3 ± 1.7	1.3 ± 2.1	1.2 ± 1.3	0.899
hs-cTnT, ng/L	10.3 ± 10.2	11.8 ± 10.4	8.9 ± 9.9	0.094
NT-proBNP, ng/L	1437 ± 2079	1734 ± 2639	1165 ± 1962	0.063
AST, mg/dL	24.4 ± 8.9	25.6 ± 9.9	23.4 ± 7.7	0.113
Baseline treatment				
Supportive therapies				
Anticoagulants, n (%)	65 (39)	32 (40.5)	33 (37.5)	0.734

(*Continued*)

Table 1. Baseline Characteristics* (*Continued*)				
Loop diuretics, n (%)	113 (67.7)	61 (77.2)	52 (59.1)	0.012
Spironolactone, n (%)	75 (44.9)	51 (57.2)	30 (34.1)	0.003
ACEi, n (%)	57 (34.1)	32 (38)	31 (35.2)	0.713
ß-blocker, n (%)	61 (36.5)	32 (38)	31 (35.2)	0.732
Statins, n (%)	27 (16.2)	16 (19)	12 (14.8)	0.605
PH specific therapy				
PDE5i	55 (33.6)	35 (44.3)	20 (23.9)	0.078
ERA	81 (48.6)	43 (54.4)	38 (43.1)	0.092
Riociguat	29 (17.4)	12 (16.5)	16 (18.2)	0.769
Follow-up				
Days, median (IQR)	509 (732)	545 (709)	557 (766)	0.345
Hospitalization, n (%)	30 (17.9)	24 (30.3)	6 (6.8)	<0.001

*Values are mean ± SD, median (IQR), or number (percentage) unless specified otherwise.
6MWT, 6-minute walk test; ACEi, angiotensin-converting enzyme inhibitors; AST, aspartate aminotransferase; BMI, body mass index; CAD, coronary artery disease; CKD, chronic kidney disease; CRP, C-reactive protein; ERA, endothelin receptor antagonists; eJVP, estimated jugular venous pressure; GFR, glomerular filtration rate; hs-cTnT, high-sensitivity cardiac troponin T; IQR, interquartile range; JVD, jugular venosus distention; NT-proBNP, N-terminal pro-brain natriuretic peptide; PCA, prostacyclin analogues; PDE5i, phosphodiesterase-5 inhibitors; PH, pulmonary hypertension; PND, paroxysmal nocturnal dyspnea; S_2, second heart sound; S_3, third heart sound; SBP, systolic blood pressure; TR, tricuspid regurgitation; WHO, World Health Organization.

Table 2. Echocardiographic and Hemodynamic Parameters*				
	All (n=167)	**Bendopnea (n=79)**	**No Bendopnea (n=88)**	***P***
Echocardiographic parameters				
LVEF, %	58.9 ± 5.8	57.6 ± 6.7	60.1 ± 4.6	0.063
RA area, cm^2	21.5 ± 5.8	23.2 ± 6.1	20.1 ± 5.1	<0.001
Mitral E/e'	15.3 ± 5.9	19.7 ± 5.8	12.1 ± 6.1	0.032
TR V_{max}, m sec^{-1}	3.86 ± 0.8	4.16 ± 0.73	3.58 ± 0.77	<0.001
TAPSE, mm	18.3 ± 4.7	16.9 ± 4.7	19.6 ± 4.3	<0.001
TAPSE/sPAP, mm/$mmHg^{-1}$	0.35 ± 0.24	0.27 ± 0.21	0.42 ± 0.24	<0.001
Pericardial effusion, n (%)	43 (25.7)	31 (39.2)	12 (13.6)	<0.001
Invasive hemodynamic parameters				
PA systolic pressure, mmHg	68 ± 30	79 ± 30	57 ± 26	0.001
PA mean pressure, mmHg	39 ± 19	44 ± 19	35 ± 17	<0.001
Ao systolic pressure, mmHg	129 ± 22	129 ± 24	129 ± 19	0.937
RA mean pressure, mmHg	9.8 ± 4.7	11.3 ± 4.7	8.3 ± 4.4	<0.001
PCWP, mmHg	11.6 ± 5.4	12.6 ± 6.4	10.5 ± 4.1	0.035
PVR, Woods	6.7 ± 5.3	8.4 ± 5.7	5.2 ± 4.2	<0.001
SVR, Woods	19.3 ± 11.2	21.3 ± 14.8	17.5 ± 5.8	0.058
SaO_2, %	94.5 ± 4.5	93.8 ± 4.6	95.1 ± 4.3	0.09
MvO_2, %	67.9 ± 8.8	66.4 ± 8.6	69.3 ± 8.7	0.05
CO, L/min	5.03 ± 1.68	4.4 ± 1.7	4.89 ± 1.58	0.087
CI, L/min/m^2	2.82 ± 0.94	2.68 ± 0.93	2.95 ± 0.94	0.126
SV, mL	62.9 ± 22.3	60.1 ± 22.4	60.7 ± 22.1	0.178
SI, mL/min	35.7 ± 12.4	33.9 ± 11.5	37.5 ± 10.2	0.145
RVEF, n (%)	43 (25.7)	31 (39.2)	12 (13.6)	<0.001

*Values are mean ± SD or number (%).
Ao, aortic; CI, cardiac index; CO, cardiac output; LVEF, left ventricular ejection fraction; MvO2, mixed venous oxygen saturation; PA, pulmonary artery; PCWP, pulmonary capillary wedge pressure; PVR, pulmonary vascular resistance; RA, right atrium; RV, right ventricular; RVEF, right ventricular energy failure; SaO2, systemic oxygen saturation; SI, stroke index; sPAP, estimated systolic pulmonary artery pressure; SV, stroke volume; SVR, systemic vascular resistance; TAPSE, tricuspid annular plane systolic excursion; TR, tricuspid regurgitation; Vmax, maximum velocity.

Discussion

Right ventricular failure has a grave impact on PH prognosis. Surprisingly, most of the hemodynamic variables, especially when they are taken in isolation, have failed to predict the development of RV failure, including pulmonary artery pressure that entitles the disease itself.[1] One explanation for this fact is pulmonary hemodynamics in PH show a substantial degree of variation. For example, pulmonary vascular resistance may increase up to 10 times its upper limit of normal; the RV, on the other hand, can boost its contractility up to 5 times.[3,4] Therefore, RV contractile capability and pulmonary vascular load should always be assessed in combination. Right ventricular energy failure concept is a new approach to this interaction and implies a dire situation, where the RV is incapable of generating enough energy to propel the blood through the pulmonary circulation. It has been shown that the development of RVEF nearly doubles the mortality of PH even after correction for prognostic parameters recommended by contemporary guidelines.[5]

Bendopnea, another recently defined entity, has been initially defined as a symptom of HF but was later shown to occur in idiopathic pulmonary arterial hypertension,[7] advanced aortic stenosis,[11] obstructive sleep apnea syndrome,[12] and approximately 6.7% of the general population.[13] To the best of our knowledge, this is the first study that aims to predict the presence of RVEF from noninvasive clinical evaluation. Our results indicate that bendopnea accurately predicts the presence of RVEF in patients with PH and has a significantly superior diagnostic power compared with many symptoms and physical signs. While the European Society of Cardiology and European Respiratory Society guidelines on PH propose bendopnea as an early symptom,[1] bendopnea was more frequent in patients with a worse echocardiographic and hemodynamic profile in our study, indicating more advanced right-sided HF and PH, which is not an unexpected finding as RVEF already selects a more deranged ventriculoarterial coupling situation.[14]

Although the underlying mechanism of bendopnea is not clear, the increase in ventricular filling pressures related to the increased intra-thoracic pressure or intraabdominal pressure during bending was blamed as the primary mechanism. In the original study,[6] the authors found that a profile of high PCWP with a low cardiac output was significantly more common in those with bendopnea. Accordingly, they reasoned that bendopnea in left-sided HF indicates diminished left ventricular contractile reserve that cannot increase its output with increased filling pressures. However, it is not clear whether the mechanism of bendopnea in HF is the same as the mechanism in PH. The only study that compared bendopnea in HF and PH showed that bendopnea was significantly more common in patients with HF.[15] The authors interpreted these results as bendopnea being associated with an increase in PCWP independent of right-sided pressures and therefore claimed that this symptom was specific to left-sided HF with increased LV filling pressures. However, the majority of the patients in group I PH in this study seem not to have RVEF, which can partly explain why bendopnea appeared to be a specific symptom of increased left-sided filling pressures. Our results indicate that bendopnea is not specific to increased left-sided filling pressures and suggest the mechanism and the prognostic implication of bendopnea in HF and PH may be different. We speculate that bendopnea in PH probably is a right-sided analog of the abovementioned mechanisms and manifests already deranged RV-pulmonary arterial coupling by increasing venous return and/or pulmonary vascular resistance (Figure 2). However, further studies are needed to clarify its mechanism in PH.

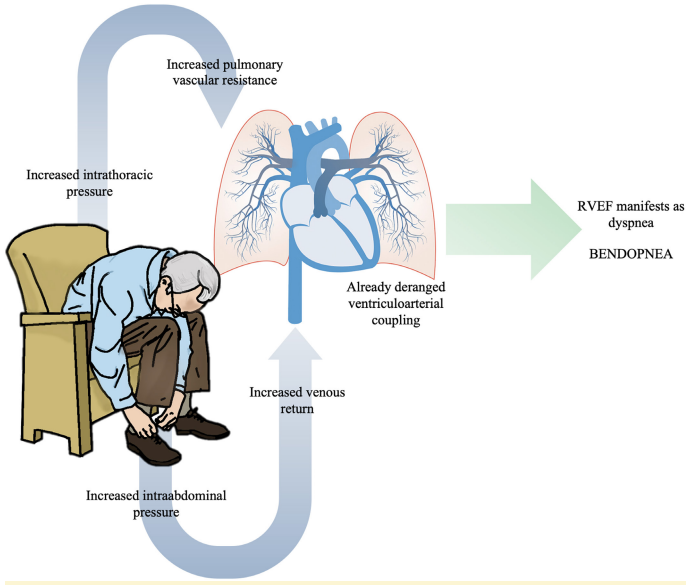

Figure 2. Possible pathophysiologic mechanism of bendopnea in pulmonary hypertension. RVEF, right ventricular energy failure.

Our study has several limitations. The bendopnea test has a subjective nature which may reduce its reliability. The physical examination was performed by a single physician; therefore, a comparative evaluation was not possible. The right heart catheterization could not be performed during the bendopnea test, which might also have helped to elucidate the underlying mechanism. Although prognostic utility of RVEF has been shown previously, the direct prognostic impact of bendopnea in PH also needs to be elucidated in further studies.

In conclusion, our study shows that bendopnea predicts RVEF in patients with PH and can be added to our physical examination armamentarium as an easy, rapid, and noninvasive prognostic tool. Our results also confirm and expand that bendopnea is not specific to HF with reduced ejection fraction, and its mechanistic and prognostic meaning in PH may differ from that in HF.

Ethics Committee Approval: This study was approved by Ethics Committee of Marmara University (Approval Number: 09.2021.1111, Date: 08.10.2021).

Informed Consent: Written informed consent was obtained from the patients who agreed to take part in the study.

Peer-review: Externally peer-reviewed.

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Conflict of Interests: The authors have no conflict of interest to declare.

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