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A Multiparametric Scoring System (RVDS10) Integrating Echocardiography and Diaphragm Ultrasonography for Early Detection of Right Ventricular Dysfunction in Chronic Obstructive Pulmonary Disease: A Prospective Cohort Study in Military Personnel

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Abstract

Introduction. Right ventricular (RV) dysfunction develops early in chronic obstructive pulmonary disease (COPD) but is difficult to capture with any single echocardiographic index.

Purpose. To characterize combined cardiac, diaphragmatic and functional remodeling in COPD and to develop and internally validate a multiparametric score (RVDS10) for early detection of RV dysfunction.

Materials and methods. In this prospective, comparative, clinical-instrumental cohort study conducted at the functional/ultrasound diagnostics and pulmonology departments of a tertiary military-medical center, 100 patients with stable, GOLD-confirmed COPD and 50 age-, sex- and BMI-matched healthy controls underwent 12-lead electrocardiography, spirometry, mMRC/CAT questionnaires, transthoracic echocardiography performed according to the 2025 American Society of Echocardiography (ASE) right-heart recommendations, diaphragm ultrasonography, and the 6-minute walk test (6MWT). Ten binary criteria spanning RV contractility, pulmonary afterload, respiratory mechanics and functional/symptomatic burden were combined into the RVDS10 score (range 0-10); diagnostic performance was assessed with receiver-operating-characteristic (ROC) analysis.

Results. COPD patients had significantly lower TAPSE (15.35±1.48 vs 20.07±1.30 mm), S' (8.62±1.07 vs 10.93±1.03 cm/s) and FAC (32.66±6.35 vs 39.31±4.48%), and higher PASP (36.64±5.70 vs 28.45±2.71 mmHg) than controls (all $p<0.001$). In isolation, diaphragm ultrasonographic indices (thickness, thickening fraction, contraction kinetics) did not differ significantly between groups (all $p>0.05$), and the proportion of participants below the 35-mm excursion threshold was similar in both groups (42.0% vs 40.0%, $p=0.815$) – yet diaphragm excursion still correlated weakly with FEV_1 ($r=0.205$, $p=0.041$) and

contributed measurably to the integrated score (AUC=0.643 for predicting high integral risk). Among single predictors, FAC had the highest discriminative ability for high integral risk (AUC=0.741), outperforming TAPSE (AUC=0.646). Mean RVDS10 was 5.56±1.24 in COPD versus 0.78±0.62 in controls (p<0.001); 80.0% of COPD patients fell into the high-integral-risk category (RVDS10≥6) and none into the low-risk category. At the optimized ≥4-criteria threshold, RVDS10 separated the high-risk group with 100% sensitivity and specificity (AUC=1.000 by construction; individual component correlations with the total score ranged from r=–0.48 to r=–0.72, all p<0.01).

Conclusion. Subclinical right-heart and diaphragmatic dysfunction is common even in clinically stable COPD, and no single echocardiographic parameter reliably captures it. The RVDS10 algorithm, combining structural, functional, hemodynamic, respiratory and symptomatic domains, provides an internally consistent, resource-light framework for risk stratification that merits prospective external validation, including in occupational/military screening settings.

Keywords: chronic obstructive pulmonary disease, right ventricular dysfunction, echocardiography, diaphragm ultrasonography, TAPSE, cor pulmonale, multiparametric score, military medicine

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Мультипараметрическая система оценки (шкала RVDS10), объединяющая эхокардиографию и ультразвуковое исследование диафрагмы, для раннего выявления дисфункции правого желудочка при хронической обструктивной болезни легких: проспективное когортное исследование среди военнослужащих

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Резюме

Введение. Дисфункция правого желудочка (ПЖ) развивается на ранних стадиях хронической обструктивной болезни легких (ХОБЛ), однако с трудом выявляется по какому-либо одному эхокардиографическому показателю.

Цель. Охарактеризовать сочетанное ремоделирование сердца, диафрагмы и функциональных показателей при ХОБЛ, а также разработать мультипараметрическую шкалу (RVDS10) для раннего выявления дисфункции ПЖ и провести ее внутреннюю валидацию.

Материалы и методы. В проспективном сравнительном клинико-инструментальном когортном исследовании, выполненном на базе отделений функциональной/ультразвуковой диагностики и пульмонологии многопрофильного военно-медицинского центра, приняли участие 100 пациентов со стабильной, подтвержденной по критериям GOLD ХОБЛ и 50 здоровых лиц, сопоставимых по возрасту, полу и ИМТ. Всем участникам выполнены 12-канальная электрокардиография, спирометрия, трансторакальная эхокардиография согласно рекомендациям Американского общества эхокардиографии (ASE) по оценке правых отделов сердца 2025 г., ультразвуковое исследование диафрагмы и тест с 6-минутной ходьбой (Т6МХ). Все участники заполнили опросники mMRC/CAT. Десять бинарных критериев, отражающих сократимость ПЖ, легочную постнагрузку, механику дыхания и функционально-симптоматический статус, были объединены в шкалу RVDS10 (диапазон 0–10); диагностическая значимость оценивалась с помощью ROC-анализа.

Результаты. У пациентов с ХОБЛ значения TAPSE (15,35±1,48 против 20,07±1,30 мм), S′ (8,62±1,07 против 10,93±1,03 см/с) и FAC (32,66±6,35 против 39,31±4,48%) были достоверно ниже, а PASP (36,64±5,70 против 28,45±2,71 мм рт. ст.) – выше, чем в контрольной группе (все $p<0,001$). По отдельности показатели УЗИ диафрагмы (толщина, фракция утолщения, кинетика сокращения) значимо не различались между группами (все $p>0,05$), доля лиц с экскурсией диафрагмы ниже порога 35 мм была сопоставима в обеих группах (42,0% против 40,0%, $p=0,815$), тем не менее экскурсия диафрагмы слабо коррелировала с ОФв1 ($r=0,205$, $p=0,041$) и вносила измеримый вклад в интегральную шкалу (AUC=0,643 для прогнозирования высокого интегрального риска). Среди отдельных предикторов наибольшей дискриминационной способностью в отношении высокого интегрального риска обладал FAC (AUC=0,741), превосходя TAPSE (AUC=0,646). Среднее значение RVDS10 составило 5,56±1,24 при ХОБЛ против 0,78±0,62 в контроле ($p<0,001$); 80,0% пациентов с ХОБЛ отнесены к категории высокого интегрального риска (RVDS10≥6), ни один – к категории низкого риска. При оптимизированном пороге ≥4 критериев RVDS10 разделяла группу высокого риска со 100%-ной чувствительностью и специфичностью (AUC=1,000 по построению; корреляции отдельных компонентов с суммарным баллом составили от $r=–0,48$ до $r=–0,72$, все $p<0,01$).

Заключение. Субклиническая дисфункция правых отделов сердца и диафрагмы часто встречается даже при клинически стабильном течении ХОБЛ, и ни один отдельный эхокардиографический параметр не позволяет надежно ее выявить. Алгоритм RVDS10, объединяющий структурный, функциональный, гемодинамический, респираторный и симптоматический домены, представляет собой внутренне согласованную, малозатратную основу для стратификации риска, требующую проспективной внешней валидации, в том числе в условиях профессионального/военного скрининга.

Ключевые слова: хроническая обструктивная болезнь легких, дисфункция правого желудочка, эхокардиография, ультразвуковое исследование диафрагмы, TAPSE, легочное сердце, мультипараметрическая шкала, военная медицина

■ INTRODUCTION

Chronic obstructive pulmonary disease (COPD) remains one of the most prevalent causes of morbidity and mortality worldwide, characterized by progressive airflow limitation, chronic airway inflammation, and alveolar destruction [1]. Global epidemiological data confirm a persistently high and rising burden of disease [2, 3], including in Uzbekistan, where regional surveillance data show a comparable public-health impact [3]. While traditionally regarded as a primary respiratory disorder, COPD is increasingly recognized as a multisystem syndrome with substantial cardiovascular consequences. Within the continuous cardiopulmonary circuit, the right ventricle (RV) functions as a sensitive barometer of pulmonary vascular health, respiratory mechanics and systemic oxygenation.

Historically, the RV was regarded as a secondary chamber relative to the left ventricle [4]. Contemporary evidence instead identifies RV structure and function as key determinants of exercise tolerance, exacerbation frequency, and survival in COPD [5]. The pathogenesis of RV dysfunction in COPD is multifactorial: chronic alveolar hypoxia triggers hypoxic pulmonary vasoconstriction, which over time produces fixed pulmonary-vascular remodeling and sustained elevation of pulmonary vascular resistance [6, 7]. In parallel, progressive hyperinflation flattens the diaphragm, alters intrathoracic pressure dynamics, and impedes venous return, further straining right-sided hemodynamics [8].

Detecting these changes before overt cor pulmonale develops is difficult. The RV's crescentic, tri-partite geometry resists simple two-dimensional quantification and makes single-parameter assessment operator-dependent [4]. For years, echocardiographic evaluation relied on isolated binary cut-offs, such as a tricuspid annular plane systolic excursion (TAPSE) below 1.7 cm [9]; however, longitudinal shortening is only one vector of RV contraction, and as pulmonary pressure rises the ventricle increasingly relies on radial contraction, so isolated longitudinal indices under-detect early disease [10].

The 2025 American Society of Echocardiography (ASE) recommendations for right-heart assessment represent a substantial shift away from binary classification toward a multi-tiered severity framework spanning structure, function and hemodynamics [11]. The updated guidance also aligns pulmonary-hypertension probability thresholds with the current World Symposium definition of mean pulmonary artery pressure (mPAP) above 20 mmHg [12], and emphasizes integrative indices such as the RV-pulmonary arterial (RV-PA) coupling ratio (TAPSE/PASP), which captures the physiological uncoupling that occurs when RV contractility fails to match rising afterload and independently predicts adverse outcomes across heart-failure phenotypes.

Echocardiography alone, however, does not capture the contribution of the respiratory pump. The diaphragm undergoes substantial remodeling in COPD due to chronic hyperinflation and mechanical disadvantage, and ultrasonographic assessment of its excursion and thickening fraction has emerged as a non-invasive biomarker of respiratory-muscle dysfunction [13, 14]. Diaphragmatic and echocardiographic parameters are rarely integrated into a single diagnostic model, leaving an incomplete picture of cardiopulmonary mechanical failure – a gap noted both in the broader literature [15] and in prior work from our own group and region.

This gap is particularly relevant in occupational and military medicine, where personnel are routinely exposed to extreme physical exertion, psycho-emotional stress and respiratory hazards, and where subclinical cardiopulmonary impairment can

compromise operational fitness. To address this, we developed and internally validated a ten-parameter multiparametric score – the Right Ventricular Dysfunction Score (RVDS10) – that combines clinical, echocardiographic, hemodynamic, respiratory and functional data to identify subclinical right-heart dysfunction, stage cardiopulmonary mechanical failure, and stratify risk in patients with COPD.

■ PURPOSE OF THE STUDY

To characterize combined cardiac, diaphragmatic and functional remodeling in COPD and to develop and internally validate a multiparametric score (RVDS10) for early detection of RV dysfunction.

■ MATERIALS AND METHODS

Study Design and Setting

This was a prospective, comparative, clinical-instrumental cohort study conducted in the functional and ultrasound diagnostics departments and the pulmonology division of a tertiary military-medical center. A military and occupational-health cohort was chosen deliberately: such personnel are routinely exposed to high physical exertion, psycho-emotional stress and occupational respiratory hazards, creating a practical need for sensitive, non-invasive screening tools capable of detecting subclinical cardiopulmonary impairment relevant to fitness for duty.

Participants

A total of 150 adults were enrolled: 100 patients with a clinically and instrumentally confirmed diagnosis of COPD (the COPD group) and 50 apparently healthy volunteers without clinical or instrumental evidence of cardiopulmonary disease, individually matched to the COPD group for age, sex and body-mass index (the control group).

Inclusion in the COPD group required a diagnosis according to Global Initiative for Chronic Obstructive Lung Disease (GOLD) criteria, confirmed by post-bronchodilator spirometry with a forced expiratory volume in 1 second to forced vital capacity ratio (FEV_1/FVC) below 0.70 [1]. Participants were required to be in a stable phase of disease, defined as the absence of acute exacerbation for at least four weeks before enrollment. Exclusion criteria, applied to both groups, were severe interstitial lung disease, significant congenital or acquired valvular heart disease, decompensated left heart failure (NYHA class IIIb–IV), cardiac arrhythmia precluding reliable electrocardiographic gating during echocardiography, and an inadequate ultrasound acoustic window.

Ethics Statement

The study was conducted in accordance with the ethical principles of the Declaration of Helsinki (2013 revision) [16] and was approved by the local institutional ethics committee. All participants provided written informed consent for participation and for the anonymized scientific use of their data.

Clinical and Instrumental Assessment Protocols

All participants underwent a standardized, multiparametric assessment spanning respiratory, functional, electrocardiographic and sonographic domains.

Electrocardiography. A resting 12-lead electrocardiogram (Schiller AT-101, Switzerland) was recorded for all participants under standardized conditions (morning hours, physical and emotional rest, ambient temperature 22–24 °C), and analyzed for classical markers of cor pulmonale and right-heart strain (P-pulmonale, R/S ratio, atrial fibrillation).

Spirometry. External respiratory function was assessed with a digital spirometer (Schiller AT-60, Switzerland) according to American Thoracic Society / European Respiratory Society standardization [17]. Three reproducible forced expiratory maneuvers of at least six seconds were recorded; FVC, FEV_1 and the FEV_1/FVC ratio were extracted.

Symptom Questionnaires. Symptom burden was quantified with the modified Medical Research Council (mMRC) dyspnea scale (0–4) and the COPD Assessment Test (CAT, 0–40; scores >10 indicate meaningful symptomatic impact and >20 indicate severe impact).

Six-Minute Walk Test. Functional exercise capacity was assessed with the 6-minute walk test (6MWT) in a standardized 50-metre corridor, recording total distance (6MWD), peripheral oxygen saturation and heart rate. A clinically significant desaturation event was defined as an absolute SpO_2 drop of ≥4% from baseline during exertion.

Transthoracic Echocardiography. Comprehensive echocardiography was performed with expert-class phased-array transducers (1.5–4.5 MHz) on Toshiba Aplio 300 (Japan) and Mindray Nuewa I9 (China) platforms, with acquisition and grading aligned to the 2025 ASE recommendations for right-heart assessment [11] and standard chamber-quantification guidance [18]. RV basal and mid-cavity diameters and longitudinal length were measured from the RV-focused apical four-chamber view at end-diastole and graded as normal (<4.1 cm), mildly (4.1–4.4 cm), moderately (>4.4–4.9 cm) or severely (>4.9 cm) enlarged [11]. TAPSE was recorded by M-mode through the lateral tricuspid annulus and graded as mildly (1.3–1.7 cm), moderately (1.0–1.3 cm) or severely (<1.0 cm) reduced [11]; tissue Doppler imaging (TDI) was used to derive the peak systolic (S′) velocity of the tricuspid annulus (impaired <9.5 cm/s). Fractional area change (FAC) was calculated by tracing the RV endocardial border in end-diastole and end-systole and graded as mildly (29–<35%), moderately (22–<29%) or severely (<22%) reduced [13]; the myocardial performance (Tei) index was derived by TDI. Pulmonary artery systolic pressure (PASP) was estimated from the peak tricuspid regurgitant velocity via the simplified Bernoulli equation ($PASP = 4V^2 +$ estimated right atrial pressure), with right atrial pressure estimated from inferior vena cava diameter and respirophasic collapse. The TAPSE/PASP ratio was calculated as a non-invasive index of RV-PA coupling [15]. Standard left-heart chamber, systolic and diastolic parameters were also recorded [23].

Diaphragm Ultrasonography. The right hemidiaphragm was assessed via the superior acoustic window provided by the liver [14]. Diaphragmatic excursion (DE) was measured with an anterior subcostal M-mode approach, perpendicular to the posterior third of the hemidiaphragm, from end-expiration to end-inspiration during quiet and deep breathing [19]. Diaphragmatic thickness was measured with a high-frequency linear transducer at the zone of apposition (8th-10th intercostal space, mid-axillary line) at end-expiration and end-inspiration, and the thickening fraction was calculated as (thickness at end-inspiration – thickness at end-expiration) / thickness at end-expiration × 100.

Development of the RVDS10 Score

Ten binary criteria, spanning RV contractility, pulmonary afterload, respiratory mechanics, functional capacity and clinical symptomatology, were selected for inclusion based on (i) evidence-based support from international guidelines [11, 12], (ii) physiological relevance to RV contractility and/or afterload, (iii) reproducibility in routine clinical practice, and (iv) independent association with group status in this cohort. Each criterion contributed one point when abnormal: TAPSE <17 mm; S′ (TDI) <9.5 cm/s; FAC <35%; PASP >30 mmHg; diaphragmatic excursion <35 mm; 6MWD <400 m; exertional desaturation ≥4%; FEV_1 <60% predicted; CAT >20; and mMRC ≥2. The resulting RVDS10 score ranges from 0 to 10 and was stratified into three risk categories: low risk (0–2 points), moderate risk (3-5 points), and high integral risk (≥6 points).

Statistical Analysis

Data were entered in Microsoft Excel 2019 and analyzed with IBM SPSS Statistics v.26.0. Continuous variables are presented as mean ± standard deviation or median (interquartile range) according to distribution, assessed by the Shapiro – Wilk test. Two independent groups were compared with Student's t-test for normally distributed variables and the Mann – Whitney U-test otherwise. Categorical variables are presented as n (%) and compared with Pearson's χ^2 test (or Fisher's exact test for small expected cell counts). Associations between continuous variables were assessed with the Pearson correlation coefficient (r). Diagnostic performance of individual predictors and of the RVDS10 score was evaluated by sensitivity, specificity, and the area under the receiver-operating-characteristic curve (AUC), using the binary outcome of high integral risk (RVDS10≥6). A two-sided p-value <0.05 was considered statistically significant throughout.

■ RESULTS

Participant Flow and Baseline Characteristics

Of 178 individuals screened, 150 met eligibility criteria and were enrolled: 100 in the COPD group and 50 in the control group. The COPD group comprised 54 men and 46 women with a mean age of 63.2±9.7 years; the control group comprised 28 men and 22 women with a mean age of 60.3±8.1 years (p>0.05). Groups were well matched for anthropometric and baseline hemodynamic parameters (Table 1), confirming that subsequent between-group differences can reasonably be attributed to COPD-related cardiopulmonary remodeling rather than demographic confounding.

The comorbidity profile did not differ significantly between groups for obesity, arterial hypertension, prior cerebrovascular events, depression, gout, diabetes mellitus, chronic kidney disease, dyslipidemia, smoking history, ischemic heart disease or anemia (all p>0.05; Table 2), and the frequency of cardiovascular and cardiometabolic pharmacotherapy was likewise comparable (all p>0.05; Table 3), arguing against a major confounding influence of comorbid burden or background medication on the cardiopulmonary findings below.

Electrocardiographic Findings

Classical electrocardiographic markers of right-heart strain (P-pulmonale, R/S ratio inversion, atrial fibrillation) did not differ significantly between groups (all p>0.05; Table 4), indicating that standard 12-lead ECG lacks sufficient sensitivity to detect early or subclinical right ventricular dysfunction in this population when used in isolation.

Table 1
Baseline demographic, anthropometric and hemodynamic characteristics

Parameter	Control (n=50)	COPD (n=100)	p
Age, years	60.3±8.1	63.2±9.7	>0.05
Sex, male/female, n	28/22	54/46	–
Height, m	1.68±0.07	1.67±0.06	0.837
Weight, kg	74.79±10.80	76.17±9.93	0.625
Body mass index, kg/m^2	26.71±4.41	27.35±4.06	0.395
Systolic BP, mmHg	134.28±16.12	136.57±13.21	0.387
Diastolic BP, mmHg	85.26±9.31	83.35±9.93	0.250
Mean arterial pressure, mmHg	100.46±9.60	102.24±10.70	0.305
Heart rate, bpm	81.32±9.43	79.37±9.50	0.237
Ponderal index, kg/m^3	16.01±3.01	16.42±2.80	0.421
Body surface area, m^2	1.84±0.13	1.85±0.11	0.848

Notes: Data are mean ± SD unless otherwise indicated; BP – blood pressure.

Functional Exercise Capacity

The 6MWT revealed a marked reduction in exercise tolerance among COPD patients: 6MWD was 459.36±79.84 m in the COPD group versus 584.84±61.04 m in controls (p<0.001; Table 5). Notably, calculated physical workload (Watts) did not differ significantly between groups (60.30±14.50 vs 60.94±13.46 W, p=0.814), suggesting that COPD patients expended comparable mechanical effort for a substantially shorter walked distance – a pattern consistent with combined ventilatory and circulatory limitation rather than simple deconditioning. Exertional desaturation ≥4% occurred in 33.0% of COPD patients versus 24.0% of controls, a difference that did not reach statistical significance in isolation (p=0.345).

Table 2
Comorbidity profile by study group

Comorbidity	Control (n=50)	COPD (n=100)	p
Obesity	28 (56.0%)	59 (59.0%)	0.726
Arterial hypertension	28 (56.0%)	56 (56.0%)	1.000
Prior cerebrovascular event	17 (34.0%)	34 (34.0%)	1.000
Depression	12 (24.0%)	20 (20.0%)	0.573
Gout	5 (10.0%)	14 (14.0%)	0.487
Diabetes mellitus	14 (28.0%)	26 (26.0%)	0.794
Chronic kidney disease	10 (20.0%)	15 (15.0%)	0.439
COPD (defining criterion)	0 (0.0%)	100 (100.0%)	<0.001
Dyslipidemia	24 (48.0%)	47 (47.0%)	0.908
Current/former smoking	28 (56.0%)	53 (53.0%)	0.728
Ischemic heart disease	25 (50.0%)	54 (54.0%)	0.772
Myocardial infarction, history	6 (12.0%)	14 (14.0%)	0.734
Anemia	8 (16.0%)	18 (18.0%)	0.760

Notes: Data are n (%). Groups were comparable for all comorbidities except the COPD diagnosis that defined group membership.

Table 3
Cardiovascular and cardiometabolic pharmacotherapy by study group

Medication class	Control (n=50)	COPD (n=100)	p
Allergic rhinitis therapy	12 (24.0%)	25 (25.0%)	0.893
Beta-blockers	13 (26.0%)	26 (26.0%)	1.000
ACE inhibitors	20 (40.0%)	42 (42.0%)	0.947
Calcium-channel blockers	8 (16.0%)	12 (12.0%)	0.662
Mineralocorticoid-receptor antagonists	12 (24.0%)	23 (23.0%)	0.891
Diuretics	18 (36.0%)	29 (29.0%)	0.474
Nitrates	3 (6.0%)	5 (5.0%)	1.000
Statins	12 (24.0%)	18 (18.0%)	0.501
SGLT2 inhibitors	1 (2.0%)	1 (1.0%)	1.000
Antiplatelet agents	10 (20.0%)	20 (20.0%)	1.000

Notes: Data are n (%); ACE – angiotensin-converting enzyme; SGLT2 – sodium-glucose cotransporter-2.

Table 4
Electrocardiographic findings by study group

ECG finding	Control (n=50)	COPD (n=100)	p
P-pulmonale	9 (18.0%)	18 (18.0%)	1.000
R/S ratio >1 (V1)	9 (18.0%)	14 (14.0%)	0.522
R/S ratio <1 (V6)	3 (6.0%)	7 (7.0%)	1.000
Atrial fibrillation	1 (2.0%)	7 (7.0%)	0.270

Notes: Data are n (%); ECG – electrocardiogram.

Table 5
Six-minute walk test results by study group

Parameter	Control (n=50)	COPD (n=100)	p
6MWD, m	584.84±61.04	459.36±79.84	<0.001
Physical workload, W	60.94±13.46	60.30±14.50	0.814
Desaturation ≥4%, n (%)	12 (24.0%)	33 (33.0%)	0.345

Notes: Data are mean ± SD or n (%); 6MWD – six-minute walk distance.

Spirometric Findings

Spirometry confirmed a predominantly obstructive ventilatory pattern in the COPD group (Table 6). Measured FVC (3.16±0.73 vs 4.25±0.43 L) and FEV_1 (2.19±0.72 vs 3.90±0.35 L) were markedly reduced compared with controls (both p<0.001), and the FEV_1/FVC ratio fell from 79.71±2.79% in controls to 57.78±7.64% in the COPD group (p<0.001), confirming significant, fixed airflow obstruction.

Table 6
Core spirometric indicators by study group

Parameter	Control (n=50)	COPD (n=100)	p
FVC, measured, L	4.25±0.43	3.16±0.73	<0.001
FEV_1, measured, L	3.90±0.35	2.19±0.72	<0.001
FEV_1/FVC, %	79.71±2.79	57.78±7.64	<0.001

Notes: Data are mean ± SD; FVC – forced vital capacity; FEV_1 – forced expiratory volume in 1 second.

Table 7
Diaphragm ultrasonographic parameters by study group

Parameter	Control (n=50)	COPD (n=100)	p
Thickness at end-inspiration, cm	0.29±0.04	0.28±0.04	0.411
Thickness at end-expiration, cm	0.17±0.04	0.17±0.04	0.906
Thickening fraction, %	72.36±28.98	70.20±29.22	0.606
Thickening ratio	1.72±0.29	1.70±0.29	0.717
Contraction slope, cm/s	2.57±0.51	2.55±0.58	0.846
Inspiratory time, s	1.68±0.40	1.70±0.45	0.742
Excursion <35 mm, n (%)	20 (40.0%)	42 (42.0%)	0.815

Notes: Data are mean ± SD or n (%). No isolated diaphragmatic index differed significantly between groups; see section 3.5 and 3.8 for the corresponding correlation and multiparametric analysis.

Diaphragm Ultrasonography

Diaphragmatic ultrasound indices – thickness at end-expiration and end-inspiration, thickening fraction, contraction slope and inspiratory time – did not differ significantly between the COPD and control groups when compared in isolation (all p>0.05; Table 7), and the proportion of participants falling below the pre-specified 35-mm excursion threshold was similar in both groups (42.0% in COPD vs 40.0% in controls, p=0.815). This finding, consistent with the correlation analysis reported in section 3.8, indicates that diaphragmatic ultrasonographic parameters do not independently discriminate COPD status; their diagnostic value instead emerges only when interpreted jointly with cardiac and respiratory-functional data within a multiparametric model (see sections 3.8–3.9).

Symptomatic Burden (CAT and mMRC)

Both symptom instruments showed markedly greater burden in COPD patients (Table 8). Mean CAT score was 19.86±6.45 in the COPD group versus 10.66±4.12 in controls (p<0.001), with clinically significant symptomatic impact (CAT >20) present in 44.0% of

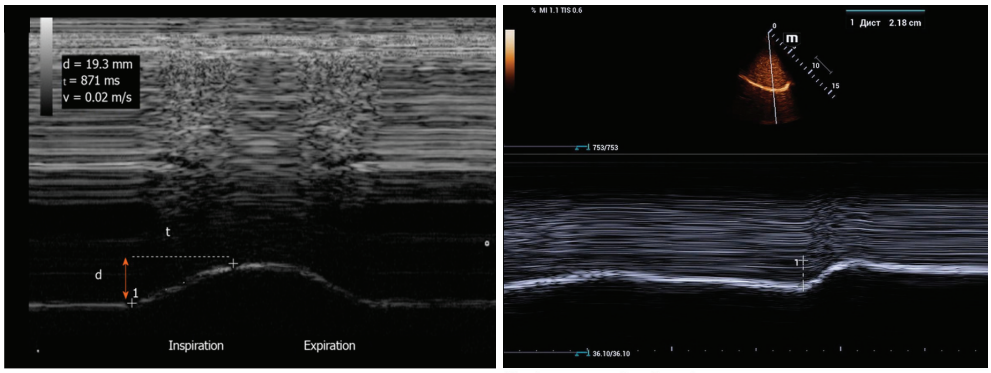


Fig. 1. Representative diaphragm ultrasonographic images from the study cohort (patient identifiers removed): A – M-mode measurement of diaphragm thickness at the zone of apposition during inspiration and expiration (d=19.3 mm; t=871 ms); B – M-mode measurement of diaphragmatic excursion via the subcostal approach (distance = 2.18 cm)

Table 8
Symptom-burden questionnaire results by study group

Parameter	Control (n=50)	COPD (n=100)	p
CAT score, points	10.66±4.12	19.86±6.45	<0.001
CAT >20 (severe impact), n (%)	0 (0.0%)	44 (44.0%)	<0.001
mMRC grade (mean)	0.44±0.50	2.07±0.83	<0.001
mMRC grade 0, n (%)	28 (56.0%)	0 (0.0%)	–
mMRC grade 1, n (%)	22 (44.0%)	31 (31.0%)	–
mMRC grade 2, n (%)	0 (0.0%)	31 (31.0%)	–
mMRC grade 3, n (%)	0 (0.0%)	38 (38.0%)	–
mMRC ≥2 (severe dyspnea), n (%)	0 (0.0%)	69 (69.0%)	<0.001

Notes: Data are mean±SD or n (%); CAT – COPD Assessment Test; mMRC – modified Medical Research Council dyspnea scale.

COPD patients and none of the controls (p<0.001). mMRC grade distribution likewise diverged sharply: 69.0% of COPD patients had clinically significant dyspnea (mMRC ≥2) versus none of the controls (p<0.001).

Echocardiographic Remodeling of the Left and Right Heart

Echocardiography revealed the most pronounced and clinically consequential between-group differences (Table 9). Left-ventricular end-diastolic and end-systolic diameters, left-ventricular mass index, and left-atrial diameter, volume index and area were all significantly greater in the COPD group (all p<0.001), alongside a higher E/e' ratio, indicating that left-heart diastolic function was also affected – consistent with the systemic cardiovascular burden of chronic hypoxemia and sustained afterload.

The right heart, however, showed the most consistent and clinically pivotal remodeling. RV basal and mid-cavity diameters were significantly enlarged in COPD (43.16±4.38 vs 37.08±3.07 mm and 37.34±2.72 vs 32.45±1.94 mm, respectively; both p<0.001), while right-ventricular systolic function was uniformly reduced: FAC fell from 39.31±4.48% in controls to 32.66±6.35% in COPD (p<0.001), TAPSE from 20.07±1.30 to 15.35±1.48 mm (p<0.001), and S' from 10.93±1.03 to 8.62±1.07 cm/s (p<0.001), while the Tei index rose from 0.35±0.04 to 0.45±0.05 (p<0.001), confirming impaired global RV performance (Figure 3). Pulmonary hemodynamics were correspondingly elevated: PASP (36.64±5.70 vs 28.45±2.71 mmHg), mean pulmonary artery pressure (mPAP, 21.91±4.17 vs 17.24±1.71 mmHg) and pulmonary vascular resistance (PVR, 2.42±0.51 vs 1.59±0.30 Wood units) were all significantly higher in COPD (all p<0.001). The TAPSE/PASP coupling ratio fell to 0.29±0.05 in the most severe disease stratum, signaling progressive uncoupling of RV contractility from pulmonary afterload. Right-atrial area was modestly higher in COPD (19.14±2.10 vs 18.08±1.65 cm^2, p=0.002), while RA diameter, RA volume and inferior vena cava parameters did not differ significantly, suggesting that RV remodeling in this cohort preceded overt right-atrial and systemic venous decompensation.

Representative echocardiographic images corresponding to the four key right-heart parameters are shown in Figure 2.

Disease-Severity Stratification and Correlation Analysis

Stratifying the COPD group by modified GOLD severity (mild-moderate, GOLD I–II, n=42; severe, GOLD III, n=38; very severe, GOLD IV, n=20) showed a step-wise deterioration

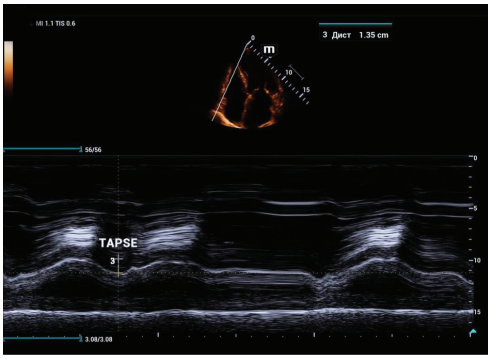
Table 9
Echocardiographic remodeling of the left and right heart

Parameter	Control (n=50)	COPD (n=100)	p
Left heart			
LV end-diastolic diameter, mm	49.92±2.03	54.69±2.73	<0.001
LV end-systolic diameter, mm	31.95±2.16	38.36±2.98	<0.001
LV mass index, g/m²	94.47±11.25	124.42±14.82	<0.001
LVEF (Simpson), %	60.24±2.34	51.84±5.09	<0.001
Left atrial diameter, mm	36.59±2.04	40.77±3.81	<0.001
Left atrial volume index, mL/m²	29.75±3.14	38.95±5.04	<0.001
E/e' (lateral)	8.05±0.93	10.86±1.94	<0.001
Right heart and pulmonary hemodynamics			
RV basal diameter, mm	37.08±3.07	43.16±4.38	<0.001
RV mid-cavity diameter, mm	32.45±1.94	37.34±2.72	<0.001
TAPSE, mm	20.07±1.30	15.35±1.48	<0.001
S' (TDI), cm/s	10.93±1.03	8.62±1.07	<0.001
FAC, %	39.31±4.48	32.66±6.35	<0.001
Tei (myocardial performance) index	0.35±0.04	0.45±0.05	<0.001
Right atrial area, cm²	18.08±1.65	19.14±2.10	0.002
PASP, mmHg	28.45±2.71	36.64±5.70	<0.001
mPAP, mmHg	17.24±1.71	21.91±4.17	<0.001
PVR, Wood units	1.59±0.30	2.42±0.51	<0.001
TAPSE/PASP ratio (RV-PA coupling)†	0.71±0.09	0.29±0.05	<0.001

Notes: Data are mean ± SD. †TAPSE/PASP is shown for the control group and for the GOLD IV (very severe) COPD stratum, illustrating the extent of RV-pulmonary arterial uncoupling at advanced disease severity. LV – left ventricle; LVEF – left-ventricular ejection fraction; RV – right ventricle; TAPSE – tricuspid annular plane systolic excursion; TDI – tissue Doppler imaging; FAC – fractional area change; PASP – pulmonary artery systolic pressure; mPAP – mean pulmonary artery pressure; PVR – pulmonary vascular resistance.

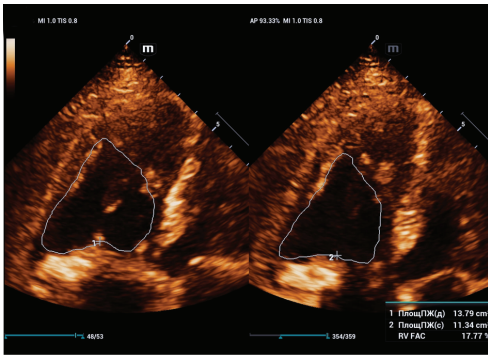
of RV systolic indices with worsening airflow obstruction: TAPSE fell from 18.2±1.9 mm (GOLD I-II) to 15.7±2.4 mm (GOLD III) to 13.8±2.1 mm (GOLD IV), with a parallel decline in S' (9.7 to 8.1 to 6.9 cm/s) and FAC (38.1% to 32.4% to 29.8%; Table 10). Baseline clinical and hemodynamic markers (age, systolic and diastolic blood pressure, heart rate) also increased significantly across strata (all $p<0.001$), as did spirometric obstruction (FEV_1/FVC 0.84±0.03, 0.64±0.02 and 0.37±0.07 across the control, mild-moderate/severe and very-severe strata respectively; all $p<0.001$).

Correlation analysis linked pulmonary function directly to RV performance: FEV_1 (%) correlated moderately with TAPSE ($r=0.58$, $p<0.01$) and with S' ($r=0.53$, $p<0.01$), and TAPSE correlated with 6MWD ($r=0.55$). Symptomatic burden tracked RV function as well – TAPSE was significantly lower in patients with CAT≥20 (15.3±2.5 vs 17.9±2.3 mm, $p<0.01$). In contrast, diaphragmatic excursion correlated only weakly with FEV_1 ($r=0.205$, $p=0.041$) and showed no significant correlation with 6MWD ($r=–0.018$, $p=0.862$), TAPSE ($r=0.096$, $p=0.344$) or S' ($r=0.034$, $p=0.740$), reinforcing that diaphragmatic mechanics contribute complementary rather than redundant information to right-heart assessment.



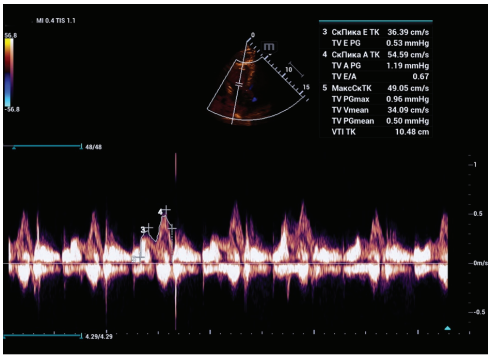
TAPSE, M-mode

A



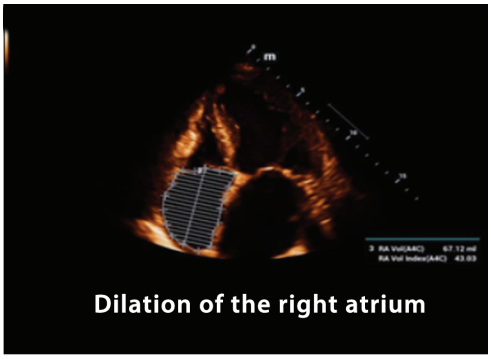
RV fractional area change (FAC)

B



Tricuspid regurgitant jet (PASP estimation)

C



Right atrial dilation, apical 4-chamber

D

Fig. 2. Representative echocardiographic images from the study cohort (patient identifiers removed): A – M-mode measurement of TAPSE; B – Planimetric tracing of RV end-diastolic and end-systolic area for fractional area change calculation (RV FAC = 17.77%); C – Continuous-wave Doppler of the tricuspid regurgitant jet used to estimate PASP; D – Apical four-chamber view demonstrating right atrial dilation

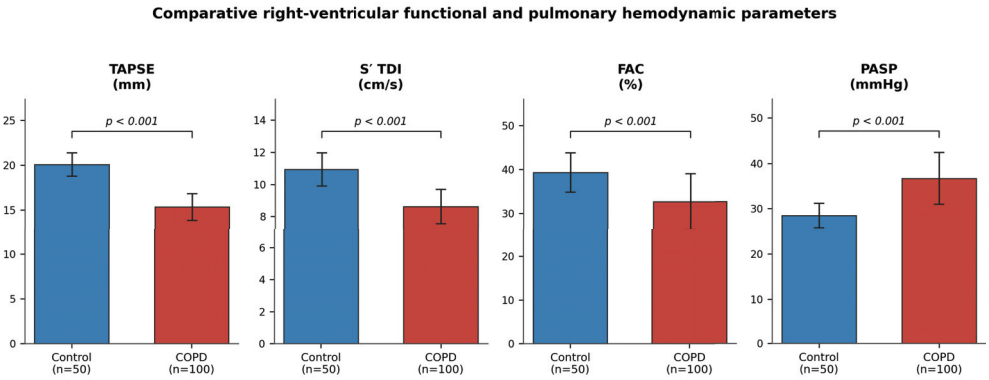


Fig. 3. Comparative right-ventricular functional and pulmonary hemodynamic parameters (mean±SD) in the control and COPD groups. Brackets show the between-group p-value for each parameter

Table 10
Clinical, hemodynamic, spirometric and right-ventricular parameters stratified by GOLD severity

Parameter	GOLD I–II (n=42)	GOLD III (n=38)	GOLD IV (n=20)	p
Age, years	52.20±5.42	52.20±7.40	64.35±8.82	<0.001
Systolic BP, mmHg	116.80±2.97	125.90±3.81	129.02±2.54	<0.001
Diastolic BP, mmHg	76.05±2.67	80.00±2.66	75.78±2.75	<0.001
Heart rate, bpm	76.15±7.65	83.70±2.94	87.13±4.56	<0.001
FEV_1/FVC	0.84±0.03*	0.64±0.02	0.37±0.07	<0.001
FEV_1, % predicted	82.85±1.87*	67.90±10.24	36.25±2.12	<0.001
FVC, L	4.46±0.28*	3.09±0.20	2.29±0.10	<0.001
TAPSE, mm	18.2±1.9	15.7±2.4	13.8±2.1	n.a.†
S' (TDI), cm/s	9.7	8.1	6.9	n.a.†
FAC, %	38.1	32.4	29.8	n.a.†

Notes: Data are mean ± SD. * Control-group reference row shown for the spirometric parameters. † Between-stratum p-values were not computed for these three echocardiographic indices in the source dataset (descriptive stratification only); the consistent step-wise deterioration is nonetheless notable and is discussed qualitatively in the text. BP – blood pressure.

RVDS10 Criterion Frequencies

Each of the ten RVDS10 criteria was substantially more frequent in the COPD group than in controls (Table 11), with the largest between-group differences observed for PASP >30 mmHg (87.0% vs 16.0%), mMRC ≥2 (69.0% vs 0.0%), S'<9.5 cm/s (61.0% vs 6.0%) and TAPSE <17 mm (60.0% vs 2.0%; all p<0.001). Exertional desaturation ≥4% and diaphragmatic excursion <35 mm did not reach statistical significance as isolated categorical criteria (p=0.257 and p=0.815, respectively), consistent with the correlation findings above; both were nonetheless retained in the integral score because of their physiological relevance and their contribution to overall model discrimination (section 3.11).

RVDS10 Score Distribution and Internal Validity

The mean RVDS10 score was 5.56±1.24 in the COPD group versus 0.78±0.62 in controls (p<0.001). By risk category, 80.0% of COPD patients fell into the high-integral-risk stratum

Table 11
Frequency of individual RVDS10 threshold criteria by study group

RVDS10 criterion	Control (n=50)	COPD (n=100)	p
TAPSE <17 mm	1 (2.0%)	60 (60.0%)	<0.001
S' <9.5 cm/s	3 (6.0%)	61 (61.0%)	<0.001
FAC <35%	12 (24.0%)	49 (49.0%)	0.006
Tei index ≥0.43†	2 (4.0%)	61 (61.0%)	<0.001
PASP >30 mmHg	8 (16.0%)	87 (87.0%)	<0.001
6MWD <400 m	0 (0.0%)	27 (27.0%)	<0.001
Desaturation ≥4%	12 (24.0%)	33 (33.0%)	0.257
Diaphragm excursion <35 mm	20 (40.0%)	42 (42.0%)	0.815
FEV_1 <60% predicted	0 (0.0%)	40 (40.0%)	<0.001
CAT >20	0 (0.0%)	44 (44.0%)	<0.001
mMRC ≥2	0 (0.0%)	69 (69.0%)	<0.001

Notes: Data are n (%). † The Tei index is reported here as a supplementary, exploratory correlate; it is not one of the ten scored RVDS10 criteria (see Table 12/section 2.5).

Table 12
Representative clinical vignettes illustrating RVDS10 scoring

Parameter	Patient 1	Patient 2	Patient 3
TAPSE, mm	16 (1 pt)	18 (0 pt)	16.5 (1 pt)
S', cm/s	8.9 (1 pt)	10.1 (0 pt)	9.1 (1 pt)
FAC, %	38 (0 pt)	36 (0 pt)	34 (1 pt)
PASP, mmHg	35 (1 pt)	29 (0 pt)	32 (1 pt)
Diaphragm excursion, mm	30 (1 pt)	36 (0 pt)	34 (1 pt)
6MWD, m	360 (1 pt)	420 (0 pt)	410 (0 pt)
Desaturation ≥4%	yes (1 pt)	no (0 pt)	no (0 pt)
CAT, points	22 (1 pt)	18 (0 pt)	21 (1 pt)
mMRC, grade	2 (1 pt)	1 (0 pt)	2 (1 pt)
FEV_1, % predicted	55 (1 pt)	68 (0 pt)	62 (0 pt)
RVDS10 total	9 – high risk	0 – low risk	7 – high risk

Notes: pt – point(s) assigned for the given criterion.

(≥6 points) and 20.0% into the moderate-risk stratum (3–5 points); no COPD patient scored in the low-risk range. All control participants fell into the low-risk category (Figure 4).

Component validity was supported by significant correlations between the total RVDS10 score and its constituent continuous variables: TAPSE (r=–0.72), S' (r=–0.68), Tei index (r=+0.70), FAC (r=–0.63), 6MWD (r=–0.61), diaphragmatic excursion (r=–0.55) and FEV_1 (r=-0.48; all $p<0.001$, except FEV_1, $p<0.01$), demonstrating a coherent multi-domain correlation structure across cardiac, respiratory and functional axes.

Three representative clinical vignettes illustrate score application (Table 12). Patient 1 (TAPSE 16 mm, S' 8.9 cm/s, FAC 38%, PASP 35 mmHg, diaphragmatic excursion 30 mm, 6MWD 360 m, desaturation ≥4%, CAT 22, mMRC 2, FEV_1 55%) scored 9/10, corresponding to high integral risk. Patient 2 (TAPSE 18 mm, S' 10.1 cm/s, FAC 36%, PASP 29 mmHg, excursion 36 mm, 6MWD 420 m, no significant desaturation, CAT 18, mMRC 1, FEV_1 68%) scored 0/10, corresponding to low risk. Patient 3 (TAPSE 16.5 mm, S' 9.1 cm/s, FAC 34%, PASP 32 mmHg, excursion 34 mm, 6MWD 410 m, no significant desaturation, CAT 21, mMRC 2, FEV_1 62%) scored 7/10, again corresponding to high integral risk, illustrating that even patients with only borderline abnormality on any single parameter can accumulate clinically meaningful integral risk.

Diagnostic Performance

Among individual predictors, FAC <35% showed the highest discriminative capacity for high integral risk (sensitivity 69.8%, specificity 74.5%, AUC=0.741), followed by S' <9.5 cm/s (AUC=0.649), TAPSE <17 mm (AUC=0.646), and diaphragmatic excursion <35 mm (AUC=0.643; Table 13, Figure 5). FEV_1 <60% alone performed poorly as a discriminator (AUC=0.441) despite high specificity (95.7%), underscoring that spirometric severity alone does not reliably flag right-heart involvement. At its internally optimized threshold (≥4 abnormal criteria), the RVDS10 algorithm separated the pre-defined high-integral-risk category (RVDS10 ≥6) with 100% sensitivity and specificity (AUC=1.000); because this reflects the mathematical consistency of the score against a threshold derived from the same criteria and cohort rather than an externally validated discrimination, it should be interpreted as evidence of internal coherence rather than proof of superiority

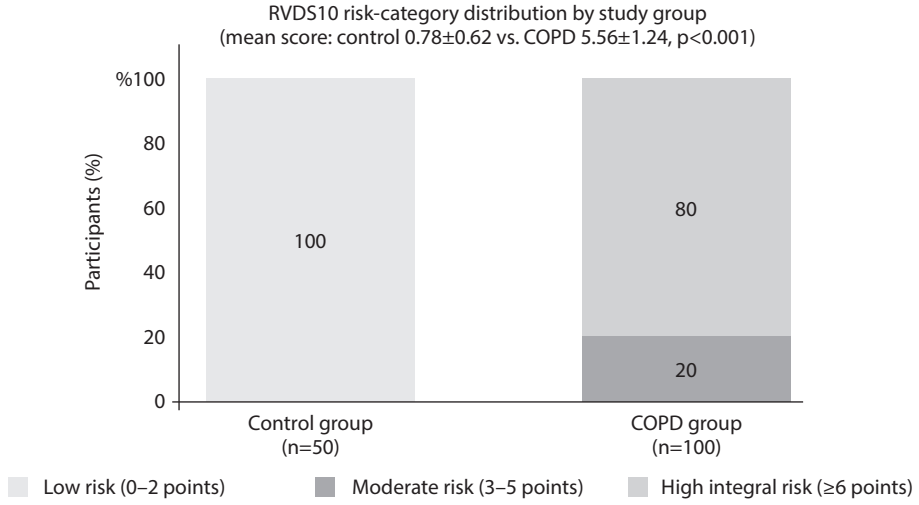


Fig. 4. Distribution of RVDS10 risk categories by study group

over any single marker, pending external validation (see section 4, Limitations). Optimal cut-points identified by ROC analysis were RVDS10 ≥6 (sensitivity 1.00, specificity 1.00 for the high-risk category by definition), FAC ≤32.4% (sensitivity 0.698, specificity 0.745) and TAPSE ≤15.9 mm (sensitivity 0.774, specificity 0.532).

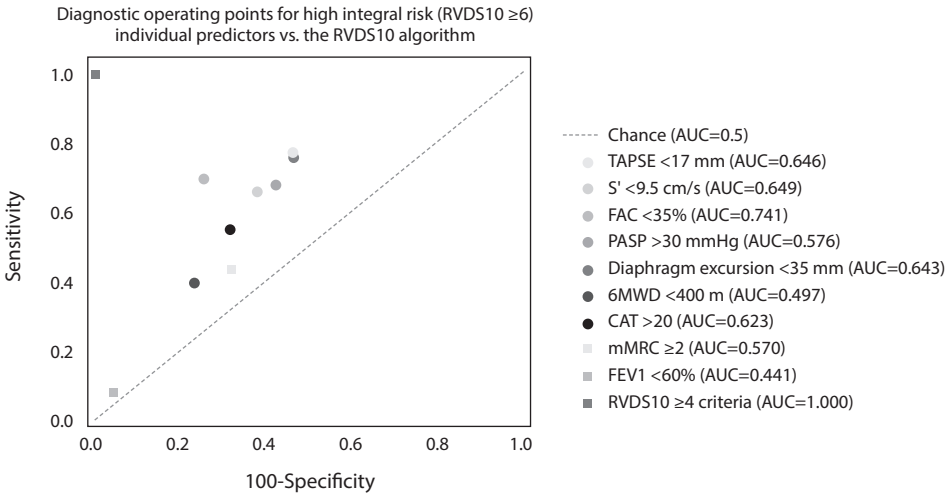
The RVDS10 Diagnostic Algorithm

Integrating the above findings, we propose a five-step clinical application pathway: 1) acquisition of all ten parameters; 2) scoring of each criterion; 3) summation of the total; 4) risk-category assignment; and 5) risk-adapted clinical action – routine surveillance for low risk, dynamic monitoring with reassessment of diaphragmatic and exercise reserve for moderate risk, and extended echocardiographic follow-up with pulmonary-hypertension work-up and intensified therapy/rehabilitation for high integral risk.

Table 13
Receiver-operating-characteristic analysis for high integral risk (RVDS10 ≥6)

Predictor	Sensitivity, %	Specificity, %	AUC
TAPSE <17 mm	77.4	53.2	0.646
S′ <9.5 cm/s	66.0	61.7	0.649
FAC <35%	69.8	74.5	0.741
PASP >30 mmHg	67.9	57.4	0.576
Diaphragm excursion <35 mm	75.5	53.2	0.643
6MWD <400 m	39.6	76.6	0.497
CAT >20	54.7	68.1	0.623
mMRC ≥2	43.4	68.1	0.570
FEV_1 <60%	7.5	95.7	0.441
RVDS10 (≥4 criteria)	100.0	100.0	1.000

Notes: AUC, area under the ROC curve. AUC=1.000 for RVDS10 reflects performance against a threshold derived from the same score in the same cohort (internal coherence) – see Discussion, Limitations.



Optimal ROC-derived cut-points

Optimal cut-point	Sensitivity	Specificity
RVDS10 ≥6	1.00	1.00
FAC ≤32.4%	0.698	0.745
TAPSE ≤15.9 mm	0.774	0.532

Fig. 5. Diagnostic operating points (sensitivity vs. 1 – specificity) for individual predictors and for the RVDS10 algorithm in identifying high integral risk (RVDS10 ≥6). The dashed line represents the line of no discrimination (AUC=0.5). The RVDS10 point reflects performance against a threshold derived from the same score in the same cohort (see Discussion, Limitations)

■ DISCUSSION

This prospective cohort study demonstrates that structural and functional remodeling of the right heart is common – and often clinically silent – even in patients with stable, ambulatory COPD, and that no single echocardiographic index reliably captures its full extent. By integrating ten criteria spanning RV contractility, pulmonary afterload, respiratory mechanics, functional capacity and symptom burden, the RVDS10 score separated COPD patients from matched controls with a mean difference of nearly five points (5.56±1.24 vs 0.78±0.62) and classified four in five COPD patients as high integral risk.

Why FAC Outperformed TAPSE?

Among individual echocardiographic parameters, FAC showed the best discriminative performance (AUC=0.741), exceeding TAPSE (AUC=0.646) and S′ (AUC=0.649). This is physiologically coherent: TAPSE and S′ each capture only the longitudinal (base-to-apex) component of RV shortening, whereas FAC integrates both longitudinal and radial contraction across the entire RV cavity [10]. As pulmonary vascular resistance rises chronically, the RV's contractile pattern shifts from a predominantly longitudinal mode toward a more global, radially-recruited pattern to preserve stroke volume [20]; single-plane longitudinal indices may therefore under-detect early or compensated dysfunction

that a global area-based index still captures [21]. This observation reinforces the rationale of the 2025 ASE recommendations for multi-parameter, rather than single-cutoff, right-heart grading [11].

Diaphragm Ultrasonography: A Complementary, not Redundant, Signal

In isolation, diaphragmatic thickness, thickening fraction and excursion did not differ significantly between COPD patients and controls in this cohort, and diaphragmatic excursion correlated only weakly with TAPSE, S′ and 6MWD. At first glance this might appear to undercut the rationale for including diaphragmatic assessment; however, we interpret it differently. First, the weak but significant correlation between diaphragmatic excursion and FEV_1 (r=0.205, p=0.041), together with a measurable individual AUC of 0.643 for predicting high integral risk, indicates that diaphragmatic mechanics do carry independent information – simply not enough to serve as a stand-alone marker. Second, the absence of correlation between diaphragmatic excursion and cardiac indices (TAPSE, S′) is itself informative: it suggests that, at least in this relatively young and physically active military cohort, respiratory-muscle mechanical failure and right-ventricular contractile failure may progress along partially independent pathophysiological tracks rather than a single linear cascade, consistent with prior descriptions of diaphragmatic remodeling as a distinct consequence of hyperinflation [8, 13]. This independence is precisely the physiological argument for a multiparametric model: a marker that is weakly correlated with – rather than redundant with – the cardiac axis adds unique discriminative information when combined with, rather than substituted for, echocardiographic parameters [14].

Relevance to Occupational and Military Medicine

Military and other physically demanding occupational cohorts face routine, high-intensity cardiopulmonary loading, and subclinical right-heart or respiratory-muscle impairment can compromise both operational performance and long-term health. Because RVDS10 relies exclusively on equipment already available in standard functional-diagnostics departments (a spirometer, an echocardiograph, a linear ultrasound probe, and a marked walking corridor) and requires no contrast agents, invasive testing or advanced strain-imaging software, it is well suited to periodic occupational health screening and fitness-for-duty evaluation in this setting, complementing the routine spirometry- and ECG-based examinations currently used in occupational medicine.

Left-Heart Involvement

COPD patients also showed evidence of left-heart remodeling – increased LV mass index, left-atrial enlargement and a higher E/e′ ratio – despite the study's focus on the right heart. This is consistent with the broader recognition that chronic hypoxemia, systemic inflammation and elevated right-sided afterload exert biventricular effects rather than isolated right-heart consequences [6, 7], and argues against interpreting COPD-related cardiac remodeling as a purely "right-sided" phenomenon.

Comparison with Existing Literature

Our findings of reduced TAPSE, FAC and S′ and elevated PASP in COPD align with previous echocardiographic studies in this population [22, 23]. The principal distinction of the present work is methodological: rather than reporting isolated echocardiographic

abnormalities or already-established hemodynamic derangement, we integrated RV structural-functional indices with respiratory (spirometric and diaphragmatic) and whole-body functional (6MWT) markers within a single, prospectively-applied multiparametric model – an approach that, to our knowledge, has not previously been evaluated specifically in a military-occupational COPD cohort.

Strengths

This study's principal strengths are (i) prospective, standardized acquisition of five independent data domains (electrocardiographic, spirometric, echocardiographic, diaphragmatic-sonographic and functional-exercise) in every participant; (ii) echocardiographic protocol and grading aligned with the contemporary 2025 ASE right-heart recommendations [11]; (iii) a well-matched control group, minimizing confounding by age, sex, BMI and comorbidity burden; and (iv) an internally coherent correlation structure linking the composite score to each of its physiological domains.

Limitations

Several limitations warrant emphasis. First, this was a single-center study with a moderate sample size (100 COPD patients, 50 controls); external replication in larger, multi-center and non-occupational cohorts is required before broader clinical application. Second, the cross-sectional design cannot establish the temporal sequence in which cardiac, diaphragmatic and functional impairment develop, nor can it demonstrate that RVDS10 predicts future clinical events (exacerbation, hospitalization, mortality); longitudinal follow-up is needed. Third, and most importantly from a methodological standpoint, the reported AUC of 1.000 for RVDS10 reflects the mathematical consistency of the score against a risk category (RVDS10 ≥6) that was itself derived from the same criteria in the same derivation cohort; this is a measure of internal coherence, not of validated discrimination against an independent reference standard, and should not be interpreted as evidence that RVDS10 outperforms individual predictors in a clinical sense. Prospective external validation – ideally against right-heart catheterization-confirmed pulmonary hypertension or cardiac MRI-derived RV volumetrics – is an essential next step. Fourth, PASP was derived non-invasively from the tricuspid regurgitant jet and an echocardiographically estimated right atrial pressure, both of which carry inherent measurement uncertainty. Fifth, this analysis did not include RV free-wall longitudinal strain or three-dimensional RV ejection fraction, both increasingly emphasized as sensitive markers of subclinical RV dysfunction in the 2025 ASE framework [11]; incorporating strain imaging may further improve the model's sensitivity to the earliest stages of disease. Sixth, all ten RVDS10 criteria were equally weighted (1 point each) for simplicity and reproducibility rather than empirically derived through multivariable regression; formal weight optimization in a larger derivation cohort, with subsequent validation in an independent sample, may improve discriminative performance. Finally, because the cohort was drawn from a military-medical population with a relatively active occupational profile, the null finding for isolated diaphragmatic ultrasonographic differences may not generalize to older, more sedentary or more severely deconditioned COPD populations, in whom diaphragmatic contribution to functional impairment may be more pronounced.

■ CONCLUSIONS

Right ventricular structural and functional impairment is prevalent in stable COPD, extends to measurable left-heart remodeling, and is not reliably captured by any single echocardiographic parameter or by resting electrocardiography alone. FAC showed the strongest individual discriminative performance among echocardiographic indices, while diaphragmatic ultrasonographic parameters, though not independently discriminative in this cohort, contributed complementary information within an integrated model. The RVDS10 score – combining echocardiographic, hemodynamic, respiratory, functional and clinical-symptomatic domains into a single, equipment-light, 0–10 point tool – showed strong internal consistency and separated COPD patients from controls with a large effect size, supporting its potential as a structured framework for early, multiparametric risk stratification of right-heart dysfunction in COPD, including in occupational and military health-screening settings. Prospective, externally validated, multi-center studies are needed before the score can be recommended for routine clinical use.

Practical Recommendations

Based on these findings, the following recommendations are proposed for clinical practice:

1. In patients with COPD – particularly military personnel and individuals in physically demanding occupations – clinicians should consider structured, multiparametric assessment of right ventricular function rather than reliance on any single echocardiographic index.
2. A complete transthoracic echocardiographic protocol capturing TAPSE, S', FAC, PASP and the TAPSE/PASP coupling ratio should be prioritized whenever right-heart involvement is suspected.
3. Diaphragmatic excursion assessment and the 6-minute walk test should be incorporated as complementary, low-cost components of the overall functional evaluation rather than as stand-alone predictors.
4. Risk stratification using the RVDS10 framework (low risk 0–2, moderate risk 3–5, high integral risk ≥6 points) may help guide the intensity of follow-up, pulmonary-hypertension work-up, and rehabilitation planning, pending external validation of the score.

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