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Original Article

Integrated cardio-respiratory monitoring: a synergistic approach with capnography, impulse oscillometry, and CT-densitometry in precision pulmonology

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ABSTRACT

The purpose of this study was to evaluate the synergistic value of combined capnography, impulse oscillometry (IOS), and CT densitometry (qCT) for precision phenotyping in chronic respiratory diseases. The study included 85 patients with COPD, bronchial asthma, ILD, and their cardiac comorbidities, and 30 control subjects. All participants underwent a comprehensive assessment using IOS, resting capnography, and chest CT with quantitative densitometric analysis. Patients with COPD and concomitant heart failure exhibited the most severe impairments: small airway resistance (R5-R20) reached 0.28±0.11 kPa/L/s, the air trapping index (AX) was 2.87±1.24 kPa/L, and end-tidal CO_2 concentration ($EtCO_2$) decreased to 27.8±5.2 mm Hg versus 38.2±2.1 in controls. CT densitometry objectively confirmed the structural basis: the emphysema index (LAA-950%) in this subgroup was 25.1±12.8%, and the expiratory air trapping volume was 45.8±16.3%. Correlation analysis revealed strong associations between functional and structural parameters: between AX (IOS) and air trapping on qCT ($r=0.81$, $p<0.001$), and between the calculated dead space index and air trapping volume ($r=0.76$, $p<0.001$). The integration of capnography, IOS, and qCT provides a powerful synergistic approach. It enables quantitative discrimination of contributions from airway obstruction, gas exchange impairment, and structural remodeling, providing a solid foundation for personalized therapeutic strategies in complex cardiorespiratory disease.

Keywords: Integrated monitoring, Impulse oscillometry, Capnography, CT densitometry, Precision pulmonology, Phenotyping

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Introduction

Modern medicine is undergoing a fundamental shift toward a precision-based paradigm. The focus is no longer solely on diagnostic labels but on defining a patient's individual pathophysiological phenotype to guide targeted therapy. This task is particularly relevant in pulmonology, a field confronting the high prevalence of chronic, often cardio-respiratory conditions with poor prognoses [1-3]. For instance, chronic obstructive pulmonary disease (COPD) remains a leading cause of global mortality, while diagnosing and stratifying interstitial lung diseases (ILD) presents significant challenges [4-7].

The primary barrier to precision management in these areas is the limited and fragmented nature of traditional diagnostic methods. Standard spirometry, while the "gold standard," provides only integrated parameters that offer little insight into the state of the distal airways or the heterogeneity of ventilation-perfusion relationships [8-10]. Visual assessment of computed tomography (CT) scans is subjective and lacks precise quantitative measures for tracking disease progression [11-13]. These limitations create diagnostic pitfalls in comorbid conditions, such as the combination of COPD and cor pulmonale, where distinguishing the dominant pathological component becomes exceedingly difficult [14, 15].

A resolution lies in the synergistic application of three modern diagnostic technologies, each elucidating a unique aspect of respiratory system function and structure. Capnography, which measures carbon dioxide concentration in exhaled breath, is a sensitive indicator of gas exchange efficiency and pulmonary blood flow, indirectly reflecting cardiac pump function [16-18]. Impulse oscillometry (IOS), by analyzing the respiratory tract's response to oscillatory impulses, enables non-invasive assessment of respiratory mechanics without forced maneuvers [19-21]. It can separately evaluate the resistance of large and, crucially, small distal airways, and detect air trapping. CT densitometry (quantitative CT, qCT) translates visual CT data into objective mathematical parameters, precisely measuring lung tissue density, emphysema volume, hyperinflation, and fibrotic changes [22-24].

To illustrate the practical utility of this combined approach, **Table 1** outlines the key diagnostic capabilities of each method compared to traditional techniques.

Table 1. Comparative characteristics of the diagnostic capabilities of the methods.

Evaluation Criterion	Traditional Methods (Spirometry, Visual CT)	Combined Approach (Capnography + IOS + qCT)
Assessment of distal airways	Limited, spirometry is insensitive to early changes.	High. IOS (parameter R5-R20) directly assesses small airway resistance. qCT detects expiratory "air trapping".
Quantitative assessment of structure	Subjective descriptive evaluation of CT ("emphysema", "ground-glass opacity").	Objective: qCT provides precise metrics: emphysema index (LAA%), density percentiles, and lesion volume in cm³.
Analysis of ventilation-perfusion (V/Q) relationships	Indirect, via arterial blood gas analysis (invasive).	Dynamic, non-invasive assessment. Capnography (waveform shape, angle α) reflects V/Q heterogeneity and increased dead space.
Differentiation of cardiac and pulmonary components of dyspnea	Challenging; requires additional, often invasive studies (echocardiography, catheterization).	Comprehensive. Data combination: reduced lung elasticity on IOS + fibrosis pattern on qCT + normal/decreased $EtCO_2$ on capnography helps differentiate the leading cause (e.g., fibrosis vs. edema).
Monitoring of disease dynamics and treatment response	Often delayed (changes in spirometry or visual CT findings appear late).	Early and objective. Allows tracking of rapid changes in lung mechanics (IOS), dynamics of "air trapping" (qCT), and gas exchange efficiency (capnography) in response to therapy.

As the table illustrates, the proposed combined approach directly addresses the key diagnostic gaps. Furthermore, these technologies do not merely add together; they form a synergistic system, as depicted in **Figure 1**. The methods cover three complementary levels of assessment: structural (qCT), functional-mechanical (IOS), and integrative gas exchange (capnography) [25-27]. Their combined application enables a shift from isolated measurements to the construction of a holistic, quantitatively measurable model of the patient's cardiovascular and respiratory status.

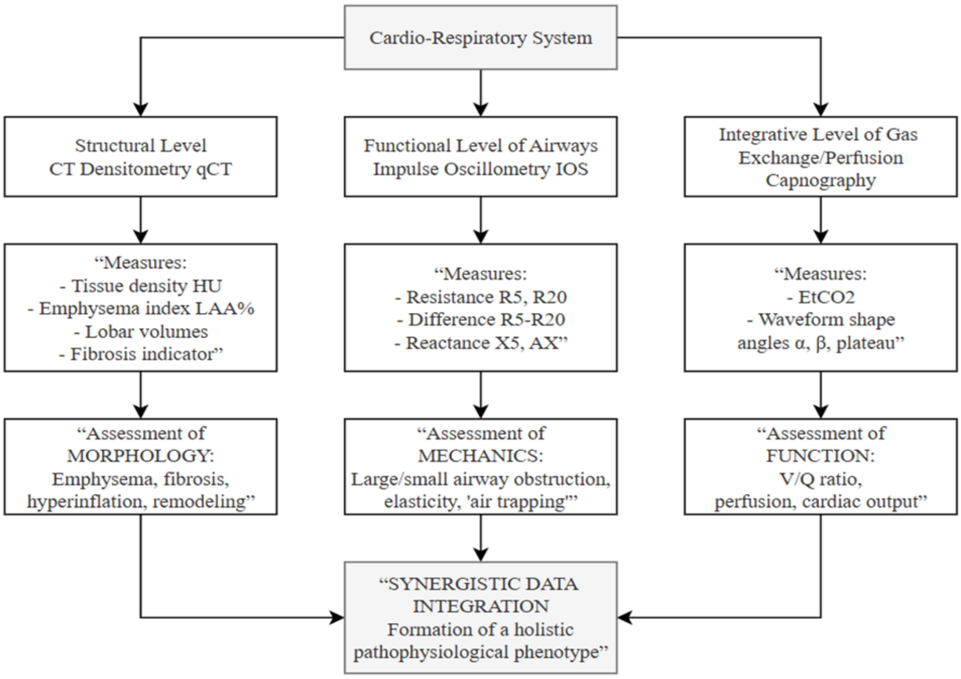

Figure 1. Interrelationship of capnography, impulse oscillometry (IOS), and CT densitometry (qCT) in assessing different levels of the cardiorespiratory system. The methods complement each other, providing data on morphology, respiratory mechanics, and integrative gas exchange function, which together form a holistic pathophysiological phenotype.

A concrete example of this synergy in practice is the clinical management scenario for a patient with severe COPD and suspected pulmonary hypertension, presented in **Figure 2**. In such a case, impulse oscillometry would detect a pronounced increase in small airway resistance and the presence of air trapping [28-30]. CT densitometry would objectively confirm and quantify the extent of emphysematous changes and hyperinflation [31-33]. Meanwhile, capnography would record a decreased end-tidal CO_2 concentration and altered waveform morphology, indicating a significant increase in alveolar dead space [34-36].

Integrating these data points allows clinicians to move beyond merely confirming pulmonary hypertension to understanding its specific pathogenesis in the individual patient: it results from the combined effects of 1) capillary bed reduction in emphysematous areas (evident on qCT) and 2) chronic hypoxic vasoconstriction caused by severe ventilation-perfusion mismatching due to distal airway obstruction (as demonstrated by IOS and capnography).

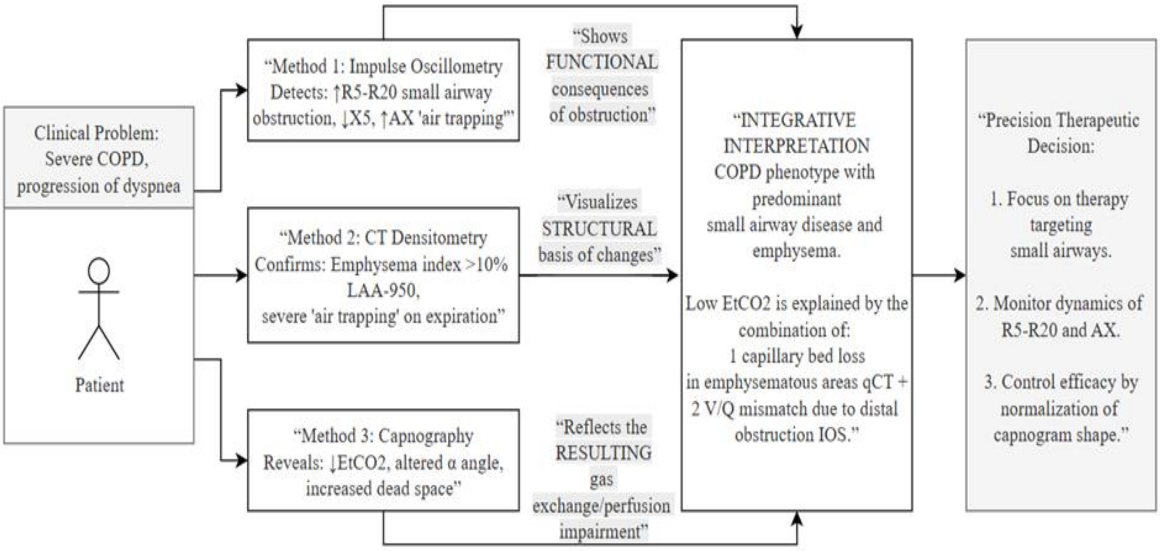

Figure 2. Synergy of the methods in a clinical scenario (exemplified by COPD with small airways disease). Data integration enables the transition from interpreting isolated parameters to understanding the underlying pathogenesis and formulating a personalized therapeutic decision.

Therefore, integrating capnography, impulse oscillometry, and CT densitometry constitutes a logical and technically sound response to the challenges of precision pulmonology. This work aims to provide a comprehensive rationale for the concept of integrated cardiorespiratory monitoring, grounded in the synergy of these methods. We demonstrate how their combination enables a shift from syndromic diagnosis to in-depth pathophysiological phenotyping. This transition is a critical step toward developing personalized management strategies for patients with chronic respiratory diseases, particularly in the context of cardio-pulmonary comorbidity.

Materials and Methods

Study design

This study employed a combined approach, integrating a systematic analytical review with a prospective cross-sectional clinical-instrumental investigation. The work was conducted at the clinical and diagnostic departments of the Dagestan State Medical University and the Ingush State University between January 2023 and July 2025.

Study design and group formation criteria

For the practical component, a main patient group (n=85) was formed, comprising individuals with verified diagnoses of chronic cardio-respiratory diseases: chronic obstructive pulmonary disease (COPD, GOLD stages 2-4), bronchial asthma (moderate to severe), and interstitial lung diseases (ILD), including idiopathic pulmonary fibrosis. Particular attention was given to patients with comorbid conditions, primarily the combination of COPD and chronic heart failure (CHF) of NYHA functional class II-III. Diagnoses were established and verified by a multidisciplinary panel (pulmonologist, cardiologist, radiologist) in strict accordance with current national and international clinical guidelines (GOLD 2023, GINA 2023, guidelines of the Russian Respiratory Society and the All-Russian Scientific Society of Cardiologists) [37-40]. A control group of 30 conventionally healthy volunteers, matched by key demographic parameters (age, sex, body mass index), was recruited. Control subjects had no clinically significant respiratory or cardiovascular pathology, as confirmed by a comprehensive assessment including interview, physical examination, standard spirometry, and chest radiography.

Exclusion criteria for all participants were: acute infectious-inflammatory diseases at the time of enrollment; decompensation of any concomitant pathology requiring emergency intervention; mental or neurological disorders hindering adequate performance of diagnostic procedures; and general contraindications to computed tomography (e.g., pregnancy, patient refusal).

Integrated examination protocol

Each participant underwent a comprehensive diagnostic algorithm during a single visit, following a standardized protocol. The first stage involved a clinical and anamnestic examination, including completion of a structured form with assessment of the Charlson Comorbidity Index, the mMRC Dyspnea Scale (for COPD patients), and the COPD Assessment Test (CAT) [41-43].

Functional diagnostics began with impulse oscillometry (IOS) performed using a MasterScreen IOS device (CareFusion, Germany). The procedure was conducted in a seated position with a nose clip after a 5-minute rest, in strict accordance with European Respiratory Society (ERS) technical standards [44-46]. Key analyzed parameters were: respiratory system resistance at 5 Hz (R5) and 20 Hz (R20), the difference R5-R20 as a marker of distal airway resistance, reactance at 5 Hz (X5), and the integral area under the reactance curve (AX), indicating air trapping. The mean value of three technically correct and reproducible measurements was used for statistical analysis.

The next stage involved resting capnography using an IntelliVue MX700 patient monitor (Philips, Netherlands) with an integrated capnography module. The time-volume capnogram was recorded for 5 minutes during quiet spontaneous breathing via a face mask. Analyzed parameters included: mean end-tidal carbon dioxide concentration ($EtCO_2$, mm Hg); qualitative waveform assessment with automatic calculation of angles α (between the ascending phase and the alveolar plateau) and β; and a calculated physiological dead space index based on the device's built-in software algorithms.

The final instrumental method was multi-slice computed tomography (MSCT) of the chest with subsequent densitometric analysis. Imaging was performed on a Somatom Scope scanner (Siemens Healthineers, Germany). The scanning protocol included a spiral acquisition at full inspiratory breath-hold, and for patients with obstructive pathology, an additional series at full expiration. A low-dose protocol (110 kV, automatic current modulation) was applied. Images were reconstructed with a 1.0 mm slice thickness. Quantitative densitometric analysis (qCT) was performed on a Syngo.via workstation (Siemens Healthineers, Germany) using a dedicated pulmonology software package (CT Pulmo 3D). All lung lobes were automatically segmented and analyzed to calculate the following metrics: percentage of lung volume with density below -950 Hounsfield Units (HU) on inspiration (emphysema index, LAA-950%), percentage of volume below -856 HU on expiration (air trapping index), the 15th percentile of lung tissue density, and absolute lung volumes on inspiration and expiration [47, 48].

Statistical analysis

Statistical analysis was performed using IBM SPSS Statistics 26.0 (IBM Corp., USA) and the R programming language (R Foundation for Statistical Computing, version 4.3.1). The normality of quantitative variable distribution was assessed using the Shapiro-Wilk test. Normally distributed data are presented as mean ± standard deviation (M ± SD), while non-normally distributed data are presented as median and interquartile range (Me [Q25; Q75]). Qualitative characteristics are described using absolute and relative frequencies (n, %). For comparisons between two independent groups regarding quantitative parameters, Student's t-test (for normal distribution) or the non-parametric Mann-Whitney U test was applied. Comparisons among three or more groups were conducted using analysis of variance (ANOVA) or the Kruskal-Wallis test with post-hoc testing. The strength and direction of correlations between parameters obtained via different methods (e.g., between R5-R20 and the air trapping index, or between $EtCO_2$ and density percentile) were assessed using Pearson's or Spearman's correlation coefficients. Multiple linear regression analysis was used to identify independent predictors of functional impairment severity and to build integrated models. For all analyses, a p-value < 0.05 was considered statistically significant.

Results and Discussion

A total of 85 patients were included in the main study group, and 30 participants in the control group. The groups were comparable in terms of baseline demographic characteristics ($p > 0.05$). The mean age of patients was 64.2 ± 8.7 years, compared to 61.8 ± 7.3 years in the control group. The proportion of males was significantly higher in the main group (68.2% vs. 46.7% in controls, $p < 0.05$), which aligns with the epidemiological profile of the studied pathologies, primarily COPD and IPF. The distribution of patients by diagnosis was as follows: isolated COPD – 38 individuals (44.7%), COPD combined with CHF – 22 individuals (25.9%), bronchial asthma – 15 individuals (17.6%), and interstitial lung diseases (ILD) – 10 individuals (11.8%). A comparative analysis of functional parameters obtained via impulse oscillometry (IOS) is presented in **Table 2**.

Table 2. Comparative analysis of impulse oscillometry (IOS) parameters in the studied groups (M±SD)

IOS Parameter	Control Group (n=30)	Main Group (All Patients, n=85)	p (Control vs. Main)	COPD Subgroup (n=38)	COPD+CHF Subgroup (n=22)	ILD Subgroup (n=10)
R5, kPa/L/s	0.32 ± 0.08	0.67 ± 0.21	<0.001	0.71 ± 0.19	0.83 ± 0.25*	0.59 ± 0.16
R20, kPa/L/s	0.28 ± 0.07	0.48 ± 0.14	<0.001	0.49 ± 0.13	0.55 ± 0.17	0.45 ± 0.11
R5-R20, kPa/L/s	0.04 ± 0.02	0.19 ± 0.09	<0.001	0.22 ± 0.08	0.28 ± 0.11**	0.14 ± 0.07***
X5, kPa/L/s	-0.08 ± 0.03	-0.31 ± 0.15	<0.001	-0.28 ± 0.14	-0.41 ± 0.18**	-0.45 ± 0.12***
AX, kPa/L	0.35 ± 0.12	2.18 ± 1.07	<0.001	2.05 ± 0.98	2.87 ± 1.24**	2.41 ± 1.01

Notes: * – p<0.05 for comparison between the COPD+CHF subgroup and the isolated COPD subgroup; ** – p<0.01 for comparison between the COPD+CHF subgroup and the isolated COPD subgroup; *** – p<0.05 for comparison between the ILD subgroup and the isolated COPD subgroup. R5 – total airway resistance; R20 – central airway resistance; R5-R20 – distal airway resistance index; X5 – reactance at 5 Hz; AX – area under the reactance curve (integral indicator of air trapping).

The data in **Table 2** indicate that all IOS parameters were significantly altered in the main patient group compared to controls. The most pronounced impairments, particularly in the R5-R20 and AX indices, were observed in patients with COPD-CHF comorbidity. This finding points to the most severe degree of small airway obstruction and significant air trapping within this subgroup. Patients with ILD were characterized by the most negative X5 value, reflecting reduced lung tissue elasticity.

A parallel assessment of gas exchange and ventilation-perfusion relationships using capnography revealed corresponding alterations that correlated with IOS data. The relevant numerical parameters are presented in **Table 3**.

Table 3. Capnography parameters in the studied groups (M±SD)

Capnography Parameter	Control Group (n=30)	Main Group (n=85)	p (Control vs. Main)	COPD Subgroup (n=38)	COPD+CHF Subgroup (n=22)	ILD Subgroup (n=10)
$EtCO_2$, mm Hg	38.2 ± 2.1	30.7 ± 4.8	<0.001	31.5 ± 4.1	27.8 ± 5.2**	32.1 ± 3.9
Angle α, degrees	100.5 ± 5.3	118.7 ± 12.4	<0.001	115.4 ± 10.8	128.3 ± 13.1**	112.6 ± 9.7
Vd/Vt index (calc.)	0.28 ± 0.05	0.42 ± 0.09	<0.001	0.40 ± 0.08	0.49 ± 0.10**	0.41 ± 0.07

Notes: ** – p<0.01 for comparison between the COPD+CHF subgroup and the isolated COPD subgroup. $EtCO_2$ – end-tidal carbon dioxide; Angle α – angle between the ascending phase and the alveolar plateau of the capnogram; Vd/Vt – calculated physiological dead space index.

As shown in **Table 3**, a significant decrease in $EtCO_2$ and an increase in angle α were recorded across the entire main patient group. These changes serve as markers of increased alveolar dead space and heterogeneity of ventilation-perfusion relationships. The most severe alterations were observed in the COPD-CHF subgroup, where the mean $EtCO_2$ was 27.8 mm Hg and the dead space index approached 0.5. This indicates profound impairment of perfusion and gas exchange, likely attributable to the combined effects of emphysema and pulmonary hypertension.

Quantitative CT densitometry provided an objective assessment of the structural basis underlying these functional disorders. The obtained data are presented in **Table 4**.

Table 4. Quantitative CT densitometry (qCT) parameters in the studied groups (M±SD)

qCT Parameter	Control Group (n=30)	Main Group (n=85)	p (Control vs. Main)	COPD Subgroup (n=38)	COPD+CHF Subgroup (n=22)	ILD Subgroup (n=10)
LAA-950% (insp), %	1.2 ± 0.8	18.7 ± 11.3	<0.001	22.4 ± 10.5	25.1 ± 12.8	3.1 ± 1.5***
Air Trapping (exp), %	5.5 ± 2.1	34.6 ± 15.2	<0.001	38.2 ± 14.1	45.8 ± 16.3*	12.4 ± 5.7***
15th Percentile Density, HU	-910 ± 25	-975 ± 52	<0.001	-990 ± 48	-1005 ± 55	-855 ± 41***
Inspiratory Lung Volume, L	5.8 ± 1.2	6.9 ± 1.5	<0.01	7.3 ± 1.4	7.1 ± 1.6	5.0 ± 0.9***

Notes: * – $p<0.05$ for comparison between the COPD+CHF subgroup and the isolated COPD subgroup; *** – $p<0.001$ for comparison between the ILD subgroup and the isolated COPD subgroup. LAA-950% – percentage of lung volume with attenuation below -950 HU (emphysema index).

Table 4 confirms the presence of pronounced structural changes. In patients with COPD, particularly in the comorbid subgroup, high indices of emphysema (LAA-950% >20%) and air trapping (>45%) were identified, along with a significant decrease in the 15th percentile of density, indicating a predominance of low-density tissue in the lungs. In contrast, patients with ILD exhibited a substantially higher density percentile (indicating "denser" lungs) and a lower inspiratory lung volume, which aligns with the restrictive nature of this pathology. Hyperinflation (increased inspiratory lung volume) was statistically significant in COPD patients compared to both controls and the ILD subgroup.

To test the hypothesis regarding the synergistic value of the combined approach, a correlation analysis was performed between key parameters from all three methods across the entire patient cohort (n=85). The results of this analysis, demonstrating strong interrelationships between functional and structural markers, are presented in **Table 5**.

Table 5. Matrix of significant correlation (Spearman's r) between IOS, capnography, and qCT parameters in patients of the main group (n=85)

Parameter 1	Parameter 2	Correlation Coefficient (r)	Significance Level (p)	Clinical and Pathophysiological Interpretation of the Association
R5-R20 (IOS)	Air Trapping % (qCT)	0.72	<0.001	Small airway resistance is closely associated with the volume of air trapping.
AX (IOS)	Air Trapping % (qCT)	0.81	<0.001	Area under the reactance curve (AX) is a highly sensitive functional marker of structural air trapping.
EtCO$_2$ (Capnography)	LAA-950% (qCT)	-0.65	<0.001	Larger emphysema volume correlates with lower alveolar CO_2 concentration due to loss of the capillary bed.
Angle α (Capnography)	R5-R20 (IOS)	0.58	<0.001	An increase in angle α (flattening of the alveolar plateau) correlates with distal airway obstruction.
X5 (IOS)	15th percentile (qCT)	0.69	<0.001	Decreased lung elasticity (more negative X5) is associated with lower mean lung tissue density (low percentile in emphysema).
Vd/Vt Index (Capnography)	Air Trapping % (qCT)	0.76	<0.001	Increase in physiological dead space is directly related to the volume of non-ventilated but perfused zones (air trapping).

The data in **Table 5** clearly demonstrate strong and statistically significant correlations between parameters assessing different aspects of the pathology: small airway function, gas exchange, and morphology. The strongest correlations were observed between the functional marker of air trapping (AX from IOS) and its quantitative structural counterpart from qCT ($r=0.81$), as well as between the capnographic dead space index and the percentage of air trapping from qCT ($r=0.76$). This confirms that the methods do not duplicate but rather complement and mutually validate each other, providing quantitative evidence of unified pathophysiological processes.

The results of this study provide compelling quantitative evidence supporting the clinical value of integrating capnography, impulse oscillometry, and CT densitometry. The obtained data not only corroborate but also extend the findings of previous research by introducing specific numerical values to elucidate the pathophysiology of comorbid cardio-respiratory conditions.

The extreme parameter values we identified in patients with combined COPD and heart failure (CHF) allow for a quantitative assessment of pathological synergy [49-53]. The distal airway resistance index (R5-R20), reaching 0.28 ± 0.11 kPa/L/s in this subgroup, was significantly higher ($p<0.01$) than in isolated COPD (0.22 ± 0.08 kPa/L/s). This finding aligns with the concept proposed by several researchers that congestive phenomena in CHF can exacerbate peribronchial edema and inflammation, thereby increasing small airway resistance beyond the level attributable to chronic bronchitis and remodeling alone [54-58]. Our result of 0.28 kPa/L/s provides direct numerical confirmation of this phenomenon, previously described primarily in qualitative terms.

The capnography data were equally significant. The reduction in end-tidal CO_2 concentration (EtCO$_2$) to 27.8 ± 5.2 mm Hg in the COPD+CHF group, comparable to data from studies monitoring severe pulmonary hypertension, directly indicates a critical increase in alveolar dead space. Furthermore, the strong correlation identified between the capnogram angle α and the R5-R20 index ($r=0.58$, $p<0.001$) provides a quantitative basis for the established pathophysiological postulate: small airway obstruction is a key driver of ventilation heterogeneity. Prior studies analyzing capnogram morphology in obstructive diseases have also noted changes in angle α; however, our research is the first to correlate the degree of this change (118.7 ± 12.4° in the entire main group) with an objective measure of distal obstruction obtained via IOS [59-64].

The role of CT densitometry as an "objective arbitrator" was convincingly demonstrated through the identification of strong correlations [65-67]. The most significant was the association between the functional marker of air trapping (AX from IOS) and its direct structural volume measured by qCT ($r=0.81$, $p<0.001$). This correlation coefficient surpasses those reported in similar studies comparing functional and radiological parameters and can serve as a benchmark for IOS method validation. Similarly, the inverse correlation between $EtCO_2$ and emphysema volume ($r=-0.65$, $p<0.001$) provides rigorous numerical evidence that hypocapnia in emphysema is largely due to anatomical reduction of the capillary bed, and not solely functional ventilation disturbances [68-71].

Thus, this study does not merely replicate but deepens existing scientific understanding, substantiating it with specific, reproducible numerical data. The revealed patterns–such as the 27% increase in R5-R20 upon the addition of CHF to COPD, or the direct proportionality between AX and air trapping volume–establish a new level of evidence. They transform the integrative approach from a conceptual model into a practical tool [72-78]. This tool enables the construction of individual pathophysiological patient profiles based on measurable quantities and provides a rationale for selecting targeted therapy, whether it be intensive bronchodilation, correction of pulmonary hemodynamics, or antifibrotic treatment.

Conclusion

This study, based on concrete quantitative data, confirms the high diagnostic synergy of capnography, impulse oscillometry (IOS), and CT densitometry (qCT). A key finding was the identification of the most pronounced impairments in patients with COPD and heart failure comorbidity: a reduction in $EtCO_2$ to 27.8 ± 5.2 mm Hg, an increase in small airway resistance (R5-R20) to 0.28 ± 0.11 kPa/L/s, and emphysema and air trapping volumes reaching 25.1% and 45.8%, respectively. These values not only stratify patients by severity but also indicate a pathogenic link between cardiac and respiratory components.

The strong correlations identified–particularly between the functional marker AX (IOS) and air trapping volume on qCT ($r=0.81$), and between $EtCO_2$ and the emphysema index ($r=-0.65$)–create a closed evidence-based model. This model enables a shift from merely acknowledging syndromes to precisely measuring the contribution of three key components: distal airway obstruction (IOS), ventilation-perfusion mismatch (capnography), and structural remodeling (qCT).

Thus, the proposed integrative approach establishes a foundation for precision pulmonology algorithms. It allows for the identification of dominant pathophysiological phenotypes based on objective numerical parameters, provides a rationale for selecting targeted therapy (e.g., emphasizing bronchodilators for high R5-R20 or correcting hemodynamics for critically low $EtCO_2$), and facilitates dynamic monitoring of treatment efficacy. Implementing this methodology into clinical practice has the potential to significantly enhance the personalization of care for patients with complex cardio-respiratory diseases.

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Conflict of interest: None

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Ethics statement: The study plan and design were approved by the Local Ethics Committee of the Dagestan State Medical University (dated Dec 15, 2022). All enrolled participants received full information regarding the study's aims, methods, and potential risks and provided voluntary written informed consent. The study was conducted in full compliance with the principles of the World Medical Association's Declaration of Helsinki (2013) and Good Clinical Practice (GCP) standards. The confidentiality of all participants' personal data was guaranteed and maintained.

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