RESEARCH PAPER

Quantitative Analysis of Ventilation–Perfusion Mismatch and Its Impact On Pulmonary Gas Exchange Efficiency Using V/Q Ratio and Alveolar–Arterial Oxygen Gradient

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Received: 17th Mar, 2026 | Revised: 29th Mar, 2026 | Accepted: 19th Apr, 2026 | Available Online: 5th May, 2026

ABSTRACT

Objective: To describe ventilation perfusion (V/Q) mismatch, and measure its direct effect on efficiency of pulmonary gas exchange by regional V/Q ratios and the alveolararterial oxygen gradient (A-a DO 2) in patients with chronic and acute respiratory diseases.

Material and Methods: The prospective observational cohort study was carried out in the tertiary care respiratory centers. The subjects (n=312) were stratified into four groups on the basis of clinical diagnosis: healthy (n=78), mild restrictive disease (n=76), obstructive airway disease (n=82), and interstitial lung disease (n=76). Two isotope protocols were used to perform quantitative V/Q SPECT/CT imaging. PaO2 and PaCO2 were given by the analysis of arterial blood gas, which allowed calculating A-a DO2. Multivariate linear regression, ANOVA and receiver operating characteristic (ROC) curve analyses were used to evaluate the relationships between V/Q heterogeneity indices and the impairment of gas exchange. The p-value was defined as $p < 0.05$.

Results: The V/Q heterogeneity had significant differences amongst disease cohorts ($p < 0.001$). The coefficient of variation (CV) of regional V/Q ratios strongly correlated with A-a DO_2 ($r = 0.74$, $p < 0.001$). Multivariate analysis found V/Q mismatch severity, physiological dead space fraction, and diffusion capacity (DLCO) to be independent predictors of high A-a gradients ($p < 0.05$ each). The A-a DO 2 threshold of 24.3 mmHg showed a sensitivity of 86% and a specificity of 79% in the detection of clinically significant V/Q mismatch (AUC = 0.89, $p < 0.001$).

Conclusion: The quantitative V/Q ratio analysis is a strong physiological correlate of dysfunctional pulmonary gas exchange. The A-a oxygen gradient is a good, non-invasive surrogate endpoint of the severity of V/Q mismatch. Combination of quantitative imaging and conventional gas exchange measures improves diagnostic accuracy and risk stratification in respiratory diseases.

Keywords: V/Q Ratio, Ventilation-Perfusion Mismatch, Alveolar-Arterial Oxygen Gradient, Quantitative Spect/Ct, Hypoxemia, Respiratory Physiology.

How to cite this article: Palwasha, Pasha H H, Akhter S, Talpur A, Rafique S, Aqeel S., Quantitative Analysis of Ventilation–Perfusion Mismatch and Its Impact On Pulmonary Gas Exchange Efficiency Using V/Q Ratio and

Alveolar–Arterial Oxygen Gradient. Int J Drug Deliv Technol. 2026;16(5): 1190-1196; Doi: 10.25258/Ijddt.16.5.111

INTRODUCTION

The pulmonary system is a highly optimized gas exchange interface with the correct matching of regional ventilation (V) and perfusion (Q) making sure that oxygen uptake and carbon dioxide elimination is efficient. In physiological states, the lung is almost perfect with a V/Q ratio of about 0.8 and the heterogeneity is reduced by gravitational, regional and micro vascular control.[1]Nevertheless, this balance is disrupted by a variety of cardiopulmonary pathologies that causes ventilation–perfusion (V/Q) mismatch, which is the most common mechanism of hypoxemia in clinical practice.To be able to diagnose, prognosticate and treat V/Q mismatch with specific and effective therapeutic interventions, it is critical to understand the quantitative nature of the relationship between V/Q mismatch and pulmonary gas exchange efficiency.[2]There are two main pathophysiological patterns of ventilation-perfusion mismatch: low V/Q units (perfusion is higher than ventilation), and high V/Q units (ventilation is higher than perfusion).Low V/Q areas lead to direct venous admixture and arterial hypoxemia, whereas high V/Q areas cause physiological deadspace, decreasing ventilatory effectiveness, but not decreasing PaO2 per se, except in severe cases.[3]These mismatches are spatially distributed and have quite different magnitudes among disease states. In chronic obstructive pulmonary disease (COPD), there is heterogeneous low V/Q zones with hyperinflated high V/Q compartments produced by airway obstruction and emphysematous destruction. Conversely, interstitial lung diseases (ILD) and pulmonary fibrosis mainly affect diffusion and decrease capillary recruitment, establishing microvascular shunt-like physiology in the presence of relatively intact ventilation.[4,5]Acute disorders like pulmonary embolism, acute respiratory distress syndrome (ARDS), and pneumonia are further examples of how sensitive the V/Q heterogeneity is as rapid regional changes may lead to acute hypoxemic respiratory failure.[6,7]A number of studies have tried to fill this gap but due to methodological inconsistencies, generalizability is constrained. Initial studies used planar scintigraphy or low-resolution SPECT, which were subject to partial volume effects, and not anatomically co-registered. Recent work has also employed positron emission tomography (PET) using oxygen-15 or nitrogen-13, which has better quantitative sensitivity, but is limited by cost, half-life of the radiotracer used, and clinical availability.[8,9]Moreover, the majority of clinical research has involved cohorts involving individual diseases, and fail to compare the outcomes with each other in order to develop universal thresholds of gas exchange impairment. Lack of standardized quantitative V/Q reporting frameworks also interferes with the cross study validation and meta-analytic synthesis.[10,11]

The clinical implications of accurately quantifying V/Q mismatch extend beyond diagnostic refinement. In critical care, identifying patients with predominant low V/Q versus high V/Q physiology can guide mechanical ventilation strategies, prone positioning, and vasodilator therapy. In chronic respiratory disease, longitudinal tracking of V/Q heterogeneity may predict disease progression, exercise intolerance, and response to pharmacological or surgical interventions. Additionally, integrating quantitative V/Q data with machine learning algorithms holds promise for personalized risk stratification and dynamic treatment optimization.[12,13] However, these applications require robust validation against established physiological benchmarks, with the A-a gradient serving as an accessible, clinically entrenched reference standard.

Our primary objective is to establish the quantitative relationship between V/Q heterogeneity metrics and A-a gradient elevation. Secondary objectives include identifying independent predictors of gas exchange impairment, determining diagnostic thresholds for clinically significant mismatch, and evaluating the utility of A-a DO_2 as a non-invasive surrogate for quantitative V/Q analysis. By integrating advanced imaging physiology with conventional gas exchange metrics, this research aims to refine diagnostic algorithms, enhance pathophysiological understanding, and support evidence-based clinical management in respiratory medicine.

MATERIAL AND METHODS

This prospective, multicenter observational cohort study was conducted between January 2024 and December 2025. Adults aged 18–75 years were recruited from outpatient pulmonary clinics, inpatient

wards, and preoperative evaluation services. Participants were stratified into four groups based on established clinical, radiological, and spirometric criteria: (1) Healthy controls (n=78) with no history of cardiopulmonary disease, normal spirometry, and normal chest imaging; (2) Mild restrictive disease (n=76) defined by total lung capacity (TLC) <80% predicted and DLCO <80% predicted without significant airway obstruction; (3) Obstructive airway disease (n=82) comprising stable COPD or asthma with FEV1/FVC <0.70 and post-bronchodilator FEV1 ≥50% predicted; (4) Interstitial lung disease (n=76) with high-resolution CT (HRCT) evidence of fibrotic or inflammatory interstitial patterns and restrictive physiology. Exclusion criteria included acute respiratory infection within 4 weeks, hemodynamic instability, pregnancy, known pulmonary embolism, prior lung resection (>1 lobe), severe anemia (Hb<10 g/dL), or inability to undergo SPECT/CT imaging. A standardized washout period of 12 hours was enforced for short-acting bronchodilators and 48 hours for long-acting agents prior to testing.Ethical Considerations and Data Governance: All procedures complied with Good Clinical Practice (GCP) guidelines. Data were anonymized using unique study identifiers and stored on encrypted, HIPAA-compliant servers. An independent data safety monitoring board reviewed adverse events quarterly. No study-related interventions altered standard clinical care.Reconstructed SPECT volumes were rigidly registered to CT datasets using mutual information algorithms. Lung parenchyma was semi-automatically segmented using threshold-based and atlas-guided techniques, excluding large airways and vessels. Voxels were classified into 1,200–1,500 anatomical segments using a standardized 18-zone model (6 axial, 3 coronal, 3 sagittal divisions per lung). Regional V/Q ratios were calculated as the ratio of normalized ventilation counts to normalized perfusion counts per voxel, with corrections for radiotracer decay, detector sensitivity, and tissue attenuation. Global and regional V/Q distributions were characterized using three validated metrics: (1) Coefficient of variation (CV = SD/mean × 100), (2) Logarithmic standard deviation of ventilation (LogSDV) and perfusion (LogSDQ), and (3) Shunt fraction (V/Q <0.1) and dead space fraction (V/Q >10). All processing was performed using commercially validated software (QV/Q Suite v3.2, MedImetrics) with inter-rater reliability assessed via intraclass correlation coefficients (ICCs).Data were compiled in a centralized REDCap database. Continuous variables were tested for normality using Shapiro–Wilk tests. Group comparisons employed one-way ANOVA with Tukey post-hoc correction or Kruskal–Wallis tests for non-parametric data. Categorical variables were analyzed using Chi-square or Fisher's exact tests. Pearson and Spearman correlations assessed relationships between V/Q metrics and A-a DO_2. Multivariable linear regression identified independent predictors of A-a gradient elevation, adjusting for age, sex, BMI, DLCO, FEV1% predicted, and comorbidities. Receiver operating characteristic (ROC) curve analysis determined optimal A-a DO_2 thresholds for detecting severe V/Q mismatch (CV >35%). Statistical significance was predefined as two-tailed $p < 0.05$. All analyses were performed using R v4.3.2 and SPSS v28.0, with 95% confidence intervals reported.A priori power calculation indicated that 280 participants would provide 90% power to detect a moderate correlation ($r = 0.40$) between CV of V/Q and A-a DO_2 at $\alpha = 0.05$. Accounting for a 10% dropout rate, 312 subjects were enrolled. Effect sizes were verified post-hoc using observed variance parameters.Imaging protocols underwent phantom-based calibration weekly. V/Q segmentation reproducibility was confirmed in a 10% random subset (ICC = 0.92, 95% CI: 0.87–0.95). ABG analyzers were calibrated daily with certified gas standards. Blinded duplicate readings were performed for 15% of datasets to ensure analytical consistency.

RESULTS

The final analytical cohort comprised 312 participants who completed all imaging, physiological, and gas exchange assessments without protocol deviation. Baseline characteristics demonstrated expected physiological gradients across cohorts, with progressive impairment in spirometric parameters, diffusion capacity, and exercise tolerance corresponding to disease severity.

Baseline characteristics demonstrate statistically significant differences across cohorts, particularly in age, BMI, FEV1, DLCO, and functional capacity ($p < 0.001$ for physiological parameters).

Table 1. Baseline Demographic and Clinical Characteristics by Study Group

Variable	Healthy (n=78)	Restrictive (n=76)	Obstructive (n=82)	ILD (n=76)	p-value
Age (years)	48.3 ± 9.1	56.7 ± 10.2	58.4 ± 11.0	61.2 ± 12.1	<0.001
Male, n (%)	42 (53.8)	41 (53.9)	49 (59.8)	44 (57.9)	0.82
BMI (kg/m²)	24.1 ± 3.2	25.8 ± 3.9	27.3 ± 4.1	26.5 ± 3.8	0.003
FEV1% pred	98.2 ± 8.5	89.4 ± 11.2	62.1 ± 14.3	78.6 ± 10.9	<0.001
DLCO % pred	101.3 ± 9.7	74.2 ± 12.4	71.8 ± 13.1	58.4 ± 11.6	<0.001
6MWD (m)	582 ± 64	491 ± 71	448 ± 83	412 ± 79	<0.001

All V/Q heterogeneity metrics differ significantly across groups ($p < 0.001$). The coefficient of variation (CV) and shunt fraction increase progressively with disease severity, indicating worsening regional mismatch.

Table 2. Quantitative V/Q Heterogeneity Indices by Disease Group

Metric	Healthy	Restrictive	Obstructive	ILD	p-value
Mean V/Q ratio	0.81 ± 0.09	0.76 ± 0.11	0.89 ± 0.14	0.72 ± 0.10	<0.001
V/Q CV (%)	18.4 ± 3.2	26.7 ± 4.8	34.2 ± 5.1	38.6 ± 5.9	<0.001
LogSDV	0.21 ± 0.04	0.29 ± 0.05	0.36 ± 0.06	0.39 ± 0.05	<0.001
Shunt fraction (%)	1.8 ± 0.6	3.4 ± 1.1	5.1 ± 1.4	6.8 ± 1.7	<0.001
Dead space fraction (%)	1.0			± 1.8	1

The correlation between V/Q heterogeneity (CV) and A-a DO_2 strengthens with disease severity, reaching $r = 0.79$ in ILD and $r = 0.71$ in obstructive disease.

Table 3. Correlation Between V/Q CV and Alveolar–Arterial Oxygen Gradient

Group	n	Pearson r	95% CI	p-value
Healthy	78	0.31	0.09–0.50	0.004
Restrictive	76	0.58	0.41–0.71	<0.001
Obstructive	82	0.71	0.58–0.81	<0.001
ILD	76	0.79	0.67–0.87	<0.001
Pooled	312	0.74	0.68–0.80	<0.001

Multivariable modeling identifies V/Q CV as the strongest independent predictor of A-a DO_2 elevation ($\beta = 0.48$, $p < 0.001$), followed by DLCO reduction and dead space fraction. Age retains modest significance, likely reflecting physiological senescence of pulmonary capillary beds.

Table 4. Multivariable Linear Regression Predictors of Elevated A-a DO_2

Predictor	β Coefficient	SE	95% CI	p-value
V/Q CV (%)	0.48	0.07	0.34–0.62	<0.001
DLCO% pred	-0.31	0.08	-0.47 to -0.15	<0.001
Dead space fraction (%)	0.22	0.06	0.10–0.34	<0.001
Age (years)	0.14	0.05	0.04–0.24	0.008
FEV1% pred	-0.09	0.05	-0.19 to 0.01	0.072
BMI	0.06	0.04	-0.02 to	0.131

Predictor	β Coefficient	SE	95% CI	p-value
(kg/m²)			0.14	

An A-a DO_2 threshold of 24.3 mmHg demonstrates excellent diagnostic accuracy for detecting severe V/Q mismatch, with an AUC of 0.89 ($p < 0.001$).

Table 5. Diagnostic Performance of A-a DO_2 for Identifying Severe V/Q Mismatch (CV >35%)

Metric	Value	95% CI	p-value
Optimal threshold (mmHg)	24.3	22.1–26.5	<0.001
Sensitivity (%)	86.2	81.4–90.1	<0.001
Specificity (%)	79.4	74.2–83.8	<0.001
AUC	0.89	0.85–0.93	<0.001
Positive LR	4.18	3.21–5.44	<0.001
Negative LR	0.17	0.11–0.26	<0.001

High sensitivity and favorable likelihood ratios support its clinical utility as a rapid screening tool. The negative likelihood ratio (0.17) indicates that a normal A-a gradient effectively rules out clinically significant mismatch, reinforcing its value in triage and longitudinal monitoring.

DISCUSSION

This study provides a comprehensive quantitative assessment of ventilation–perfusion mismatch and its direct impact on pulmonary gas exchange efficiency, establishing robust correlations between imaging-derived V/Q heterogeneity indices and the alveolar–arterial oxygen gradient. Our findings demonstrate that regional V/Q dispersion, quantified via the coefficient of variation and shunt/dead space fractions, is the predominant physiological determinant of A-a DO_2 elevation across diverse respiratory pathologies. The strong multivariable association ($\beta = 0.48$, $p < 0.001$) confirms that V/Q mismatch supersedes traditional spirometric and anthropometric parameters in explaining arterial hypoxemia.

The physiological mechanisms underlying our observations align with established respiratory pathophysiology. V/Q mismatch disrupts the ideal matching of gas delivery and capillary perfusion, creating regions of inefficient gas exchange that manifest as increased physiological shunt and dead space. Low V/Q units, prevalent in fibrotic and obstructive diseases, permit poorly oxygenated blood to bypass ventilated alveoli, directly elevating the A-a gradient through venous admixture.[14] High V/Q regions, characteristic of emphysema and pulmonary vascular disease, waste ventilation without contributing to oxygenation, indirectly worsening gas exchange efficiency by increasing the work of breathing and promoting dynamic hyperinflation.[16,17] Our quantitative imaging data reveal that the magnitude of V/C heterogeneity, rather than absolute mean V/Q, drives gas exchange impairment. This distinction is critical, as global V/Q ratios can appear normal despite severe regional dispersion, masking underlying pathophysiology. The strong correlation between CV and A-a DO_2 ($r = 0.74$) underscores the necessity of spatially resolved metrics in clinical assessment.

Comparison with recent literature reinforces the validity and novelty of our findings. Prior studies utilizing PET imaging have demonstrated similar relationships between V/Q dispersion and hypoxemia, but limited accessibility and high costs restrict widespread clinical adoption.[18,19] Our methodology aligns with these recommendations, employing attenuation-corrected, voxel-based segmentation and validated heterogeneity indices. Notably, our A-a DO_2 threshold of 24.3 mmHg slightly exceeds historical reference values, likely reflecting contemporary patient demographics with higher BMI and increased comorbidity burden. Age-adjusted corrections remain essential, as physiological capillary rarefaction and reduced elastic recoil naturally elevate A-a gradients with advancing years.[20,21] Our regression model appropriately accounts for this confounder, isolating the independent contribution of V/Q mismatch.

Our findings advocate for integrating V/Q heterogeneity metrics into prognostic models. Recent machine learning applications have demonstrated that dynamic V/Q tracking predicts exacerbation risk and mortality more accurately than static PFTs.[22,23] Longitudinal deployment of our quantitative framework could enable precision respiratory medicine, guiding intervention timing and intensity.

Despite these limitations, our study establishes a robust quantitative bridge between regional V/Q physiology and global gas exchange efficiency. The

consistent statistical significance across all analytical models ($p < 0.001$ for primary endpoints) reinforces the reliability of our findings. Clinically, these results support the routine incorporation of A-a gradient assessment in respiratory evaluation, with quantitative V/Q imaging reserved for complex or refractory cases. Educational initiatives should emphasize the physiological interpretation of V/Q heterogeneity, moving beyond binary normal/abnormal classifications toward continuous, spatially aware metrics. Health economic analyses will be crucial to determine cost-effectiveness in diverse healthcare systems, particularly given the widespread availability of SPECT/CT compared to alternative modalities.

Quantitative analysis of ventilation–perfusion mismatch reveals that regional V/Q dispersion is the principal driver of alveolar–arterial oxygen gradient elevation across respiratory diseases. The A-a DO_2 serves as a highly accurate, accessible surrogate for severe mismatch, enabling rapid clinical decision-making. Integration of advanced imaging with conventional gas exchange metrics refines diagnostic precision, supports personalized therapy, and establishes a foundation for next-generation respiratory monitoring. As computational and imaging technologies continue to evolve, the quantitative V/Q framework presented here will play a central role in transforming pulmonary physiology from descriptive observation to predictive, actionable science.

CONCLUSION

This study demonstrates that quantitative ventilation–perfusion mismatch, measured via regional V/Q ratio heterogeneity, is the primary determinant of impaired pulmonary gas exchange efficiency as reflected by the alveolar–arterial oxygen gradient. An A-a DO_2 threshold of 24.3 mmHg provides excellent diagnostic accuracy for identifying clinically significant mismatch, offering a practical, non-invasive tool for clinical triage. Integration of quantitative V/Q imaging with conventional gas exchange metrics enhances pathophysiological understanding, supports disease-specific management, and establishes a foundation for precision respiratory medicine. Future longitudinal and interventional studies should leverage this framework to optimize therapeutic targeting and improve patient outcomes across acute and chronic respiratory disorders.

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