Cureus
Part of SPRINGER NATURE

Open Access Original Article

Comparison of Venous Blood Gas Analysis With Arterial Blood Gas Analysis in Patients Presenting to the Emergency Department With Acute-Onset Dyspnea: A Cross-Sectional Study

Review began 11/05/2026
Review ended 11/18/2026
Published 11/23/2026

© **Copyright** 2026
Gangdev et al. This is an open access article distributed under the terms of the Creative Commons Attribution License CC-BY 4.0., which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

DOI: 10.7759/cureus.120162

Anand Gangdev [1], Bharat Bhardwaj [2], Naman Agrawal [3], Anmol Chanda [4], Rinku Meena [5]

1. Emergency Medicine, CU Shah Medical College and Hospital, Surendranagar, IND 2. Emergency Medicine, All India Institute of Medical Sciences, Rishikesh, IND 3. Emergency Medicine, All India Institute of Medical Sciences, Raipur, IND 4. Emergency Medicine, All India Institute of Medical Sciences, New Delhi, IND 5. Emergency Medicine, National Institute of Medical Sciences (NIMS) University, Jaipur, IND

Corresponding author: Anand Gangdev, anandgangdev7@gmail.com

Abstract

Introduction: Blood gas analysis is central to the assessment of critically ill patients in the emergency department (ED). Arterial blood gas (ABG) sampling, although the gold standard, is painful and carries procedural risks. This study aimed to determine the correlation and agreement of peripheral venous blood gas (VBG) parameters (pH, pCO_2, HCO_3^-, and lactate) with ABG analysis in a diverse cohort of ED patients presenting with acute-onset dyspnea and to evaluate the diagnostic accuracy of VBG-based acid-base diagnosis.

Methods: This was a prospective cross-sectional study conducted at the ED of a tertiary care hospital in northern India over 12 months (December 2021 to November 2022). Adult patients (⩾18 years) presenting with acute-onset dyspnea (onset <7 days) who required blood gas analysis were enrolled. Simultaneous peripheral VBG (cubital vein) and ABG (radial artery) samples were obtained within five minutes of each other. Correlation was assessed using the intraclass correlation coefficient (ICC) and agreement using Bland-Altman limits of agreement (LOA). Subgroup analysis was performed between shock and non-shock groups. Diagnostic accuracy of VBG-based acid-base classification was assessed against ABG as the gold standard.

Results: Of 255 patients, 44 (17.3%) had shock and 211 (82.7%) did not. VBG correlated strongly with ABG for pH (r = 0.98), pCO_2 (0.96), HCO_3^- (0.97), and lactate (0.98), including in shock (r = 0.95-0.98). Mean ABG-VBG differences were small: 0.031 for pH, -4.43 mmHg for pCO_2, -1.52 mmol/L for HCO_3^-, and -0.37 mmol/L for lactate. The 95% Bland-Altman LOA were -0.015 to 0.077 for pH, -13.30 to 4.44 mmHg for pCO_2, -4.63 to 1.60 mmol/L for HCO_3^-, and -1.36 to 0.61 mmol/L for lactate. The proportion of paired observations within the LOA was 96.5% for pH, 94.1% for pCO_2, 94.9% for HCO_3^-, and 94.1% for lactate. VBG sensitivity and specificity were 96.6% and 83.3% for metabolic acidosis, 100% and 89.3% for respiratory acidosis, 77.3% and 99.1% for metabolic alkalosis, and 66.1% and 98.6% for respiratory alkalosis. Venous lactate ⩾2.4 mmol/L predicted arterial hyperlactatemia (defined as arterial lactate ⩾2 mmol/L), with an area under the receiver operating characteristic curve (AUROC) of 0.996 (95% CI 0.992-1.000), sensitivity of 95.3%, and specificity of 99.3%.

Conclusion: VBG shows excellent correlation with ABG in ED patients with dyspnea, including those in shock; the differences between ABG and VBG values were small but statistically significant. VBG may be a practical alternative for initial assessment. However, VBG was less sensitive for alkalotic disorders, and ABG remains necessary when alkalosis is suspected or when precise pCO_2 or oxygenation assessment is required.

Categories: Emergency Medicine
Keywords: acid-base disorders, arterial blood gas, bland-altman analysis, dyspnea, emergency department, venous blood gas

Introduction

Dyspnea is one of the most common and potentially life-threatening chief complaints encountered in emergency departments (EDs) worldwide, arising from a broad differential spanning cardiac, pulmonary, metabolic, and other systemic disorders, making rapid, accurate diagnostic triage essential [1,2]. In the United States, dyspnea accounts for three to four million ED visits annually, representing up to 50% of patients admitted to acute tertiary hospitals [2,3]. At our institution, 7.8% of all ED visits in 2020 were for breathlessness, underscoring its clinical and operational significance in the Indian context.

Arterial blood gas (ABG) analysis remains the reference standard for evaluating ventilatory function, acid-base balance, and oxygenation in critically ill patients. However, arterial puncture is technically demanding,

causes substantial procedural discomfort, and can be complicated by hematoma, arterial thrombosis, pseudoaneurysm formation, and, rarely, compartment syndrome, especially with repeated attempts [4-6]. Arterial puncture remains one of the more painful bedside procedures performed in acute care, prompting ongoing efforts to identify effective analgesic strategies [7]. In resource-limited, high-volume EDs in India, routine ABG sampling for all dyspneic patients is further constrained by staffing and time pressures. Peripheral venous blood gas (VBG) sampling, which can be obtained simultaneously with routine intravenous cannulation, offers a safer, faster, and more patient-friendly alternative and has been increasingly adopted as a first-line triage tool in emergency medicine [8,9].

A substantial body of evidence supports close agreement between VBG and ABG for pH and bicarbonate across varied populations, but findings for pCO_2 and lactate have been less consistent [10,11]. Disease-specific studies have reported variable concordance in chronic obstructive pulmonary disease (COPD) exacerbation [12,13], diabetic ketoacidosis (DKA) [14], and sepsis or septic shock [15-18], with several authors cautioning that pCO_2 agreement narrows and lactate bias widens as physiological derangement worsens. Agreement in hemodynamically unstable or shocked patients has been particularly debated, with some studies reporting preserved concordance and others reporting clinically important divergence during circulatory failure [19]. A single-center intensive care study has examined multi-parameter arteriovenous blood gas agreement [20], but, to our knowledge, no study has assessed VBG utility in a diverse, unselected dyspneic ED population in India, nor evaluated VBG-based acid-base classification alongside a lactate-based screening threshold for hyperlactatemia [21,22].

The primary objective of this study was to determine the correlation and agreement of VBG-derived pH, pCO_2, HCO_3^-, and lactate with their ABG counterparts in patients presenting with acute-onset dyspnea. The secondary objectives were to (i) compare this correlation and agreement between patients with and without shock; (ii) evaluate the diagnostic accuracy of VBG-based acid-base classification against ABG-based classification; and (iii) determine the optimal VBG lactate cut-off for predicting arterial hyperlactatemia.

Materials And Methods

Study design and setting

This was a single-center, hospital-based prospective cross-sectional study conducted in the Trauma and Emergency Medicine Department of All India Institute of Medical Sciences (AIIMS), Rishikesh, Uttarakhand, India, a tertiary academic medical center in northern India. The study was approved by the Institutional Ethics Committee, AIIMS Rishikesh (Letter No. AIIMS/IEC/21/614; approval date: 26/11/2021). Written informed consent was obtained from all participants or their designated attendants prior to enrollment.

Study duration and population

Consecutive adult patients (⩾18 years) presenting to the ED with acute-onset dyspnea (symptom onset <7 days) from December 2021 to November 2022 were screened for eligibility. A time-bound consecutive sampling method was used over 12 months. The minimum sample size required to detect a correlation coefficient of 0.705 ($\alpha = 0.05$, power = 0.80) was 14; 255 patients were enrolled and analyzed. The enrolled sample was also adequate for the Bland-Altman agreement analysis. The standard error of each Bland-Altman limit of agreement is approximately $\sqrt{(3s^2/n)}$, where s is the SD of the paired differences [23]. Based on this approximation, a sample of 100 pairs, a commonly recommended sample size for agreement studies, provides an approximate 95% precision of ±0.34 s for each limit of agreement. In comparison, the 255 pairs included in the present study provide an improved precision of approximately ±0.21 s.

Inclusion and exclusion criteria

Patients were included if they were ⩾18 years of age and presented with acute-onset dyspnea requiring blood gas analysis (including diagnoses such as COPD exacerbation, asthma, acute respiratory distress syndrome (ARDS), pneumonia, congestive cardiac failure, pulmonary embolism, DKA, and sepsis). Patients were excluded if they (i) presented in cardiac or respiratory arrest; (ii) had a contraindication to arterial blood sampling; (iii) required immediate endotracheal intubation; (iv) had an interval >5 minutes between VBG and ABG sampling; (v) had significant hypothermia (<32°C); (vi) were pregnant; or (vii) declined consent.

Sample collection and analysis

Peripheral VBG samples were obtained from the cubital vein and ABG samples from the radial artery (following Allen's test), both using heparinized syringes. The interval between paired samples did not exceed five minutes. A tourniquet was used for venepuncture; tourniquet time was not standardized or recorded. All samples were analyzed within 15 minutes using a calibrated pHOx Ultra blood gas analyzer (Nova Biomedical, Waltham, MA, USA). Values recorded included pH, pCO_2, HCO_3^-, and lactate from both ABG and VBG.

Classification of shock and non-shock groups

Patients were classified into two groups on the basis of the blood pressure recorded at ED presentation.

Shock was defined as hypotension, i.e., Mean Arterial Pressure (MAP) <65 mmHg (shock group, n = 44); all other patients (MAP ⩾65 mmHg) were assigned to the non-shock group (n = 211). No additional criteria (such as lactate level or vasopressor requirement) were used to define the groups, and the two subgroups were not pre-specified at the time of study design.

Classification of acid-base disorders

Acid-base disorders were classified independently using ABG and VBG measurements. The ABG-based classification was considered the reference standard, against which the VBG-based classification was evaluated. Metabolic acidosis, metabolic alkalosis, respiratory acidosis, and respiratory alkalosis were defined using pre-specified pH, bicarbonate (HCO_3^-), and partial pressure of carbon dioxide (pCO_2) criteria. The same diagnostic criteria were applied to both ABG and VBG measurements. Mixed acid-base disorders were permitted, and patients with more than one concurrent disorder were classified under each applicable category.

Study outcomes

The primary outcome was the agreement between ABG and VBG values of pH, pCO_2, HCO_3^-, and lactate, expressed as the Bland-Altman bias (mean difference) with 95% limits of agreement, together with the correlation between paired values (Pearson's r). The secondary outcomes were (i) agreement and correlation between ABG and VBG values in the shock and non-shock groups; (ii) diagnostic accuracy of VBG-based classification of acid-base disorders (metabolic and respiratory acidosis and alkalosis) against ABG-based classification, expressed as sensitivity, specificity, positive predictive value (PPV), negative predictive value (NPV), accuracy, and Cohen's kappa; and (iii) the optimal VBG lactate cut-off for predicting arterial hyperlactatemia (arterial lactate ⩾2 mmol/L), determined by ROC curve analysis.

Statistical analysis

Data were entered into Microsoft Excel (Microsoft Corp., Redmond, WA, USA) and analyzed using IBM SPSS Statistics for Windows, Version 23.0 (Released 2015; IBM Corp., Armonk, NY, USA). Confidence intervals, between-group comparisons, and ROC analyses were additionally computed in Python 3 (SciPy) from the same dataset. Continuous variables were expressed as mean ± standard deviation (SD) or median (interquartile range, IQR) as appropriate, and categorical variables as frequencies and percentages (n, %). Agreement between ABG and VBG paired measurements was assessed using the Bland-Altman method, calculating bias (mean difference, ABG - VBG) with its 95% CI and 95% LOA (LOA = mean difference ± 1.96 SD of differences); the number and proportion of paired differences falling within the LOA were also reported descriptively. No clinically acceptable limits of agreement were pre-specified; the LOA were therefore interpreted descriptively. The difference between paired ABG and VBG values, in the whole cohort and separately within the shock and non-shock groups, was tested using the paired t-test (null hypothesis: mean difference = 0; reported as t-statistic with degrees of freedom (df)). The strength of the linear association between ABG and VBG values was quantified using Pearson's correlation coefficient (r), reported with its 95% CI. Linear regression was performed to derive predictive equations for ABG parameters from VBG values. The mean ABG-VBG differences of the shock and non-shock groups were compared using Welch's independent-samples t-test (reported as t-statistic with degrees of freedom). Diagnostic accuracy (sensitivity, specificity, PPV, NPV, accuracy, and Cohen's kappa) of VBG-based acid-base diagnosis was calculated against ABG as the gold standard; 95% CIs for proportions were calculated using the exact (Clopper-Pearson) method, and the 95% CI and significance of Cohen's kappa were obtained from its large-sample standard error (z-test). ROC curve analysis was used to determine the optimal cut-off of VBG lactate for predicting arterial hyperlactatemia (arterial lactate ⩾2 mmol/L), defined as the point maximizing the Youden index (sensitivity + specificity − 1); the AUROC was reported with its 95% CI, together with the positive and negative likelihood ratios and the diagnostic odds ratio at the chosen cut-off. A two-sided p-value < 0.05 was considered statistically significant.

Results

Patient characteristics

Of the 255 patients analyzed, the mean age was 54.05 ± 15.26 years (range 19-89 years), and 146 were male (n = 146; 57.3%). Most patients were triaged to the red area (n = 177; 69.4%) and arrived by government ambulance (n = 169; 66.3%). A large proportion had abnormal vital signs, including a heart rate <60 or >100 beats per minute (n = 174; 68.2%), a respiratory rate >30 breaths/min (n = 73; 28.6%), and hypotension, i.e., MAP <65 mmHg (n = 44; 17.3%). The most prevalent comorbidities were hypertension (n = 122; 47.8%), diabetes mellitus (n = 107; 42.0%), heart failure (n = 56; 22.0%), and COPD (n = 51; 20.0%). The most frequent diagnoses were lower respiratory tract infection (LRTI; n = 110; 43.1%), acute heart failure (n = 100; 39.2%), COPD exacerbation (n = 56; 22.0%), acute coronary syndrome (n = 42; 16.5%), acute-on-chronic kidney disease (n = 35; 13.7%), DKA (n = 27; 10.6%), and ARDS (n = 26; 10.2%).

Point-of-care ultrasound (POCUS) was performed in all patients. On lung ultrasound, the B-profile was the most common finding (n = 132; 51.8%), and left ventricular ejection fraction was reduced in 101 patients (n = 101; 39.6%). Overall, 103 patients were transferred to the intensive care unit (ICU) (n = 103; 40.4%), 85 to the

ward (n = 85; 33.3%), 52 were discharged (n = 52; 20.4%), and 15 died in the ED (n = 15; 5.9%). The baseline characteristics of the study population are presented in Table 1.

Characteristic	Value
Demographics	
Age, years, mean ± SD (range)	54.05 ± 15.26 (19-89)
Age, years, median (IQR); range	55.0 (44.0-65.5); 19-89
18-30 years, n (%)	23 (9.0%)
31-40 years, n (%)	21 (8.2%)
41-50 years, n (%)	49 (19.2%)
51-60 years, n (%)	62 (24.3%)
61-70 years, n (%)	68 (26.7%)
71-80 years, n (%)	24 (9.4%)
81-90 years, n (%)	8 (3.1%)
Male sex, n (%)	146 (57.3%)
Female sex, n (%)	109 (42.7%)
Mode of arrival and triage	
Government ambulance, n (%)	169 (66.3%)
Private ambulance, n (%)	59 (23.1%)
Hired vehicle, n (%)	27 (10.6%)
Triage category: red, n (%)	177 (69.4%)
Triage category: yellow, n (%)	78 (30.6%)
Vital signs at presentation	
Respiratory rate, breaths/min, mean ± SD	26.68 ± 7.37
Respiratory rate >30 breaths/min, n (%)	73 (28.6%)
Heart rate, beats/min, mean ± SD	107.93 ± 24.48
Heart rate <60 or >100 beats/min, n (%)	174 (68.2%)
Mean arterial pressure, mmHg, mean ± SD	74.72 ± 18.62
SpO_2, %, mean ± SD	93.23 ± 6.67
Hypotension (MAP <65 mmHg; shock group), n (%)	44 (17.3%)
No hypotension (MAP ≥65 mmHg; non-shock group), n (%)	211 (82.7%)
Comorbidities	
Hypertension, n (%)	122 (47.8%)
Diabetes mellitus, n (%)	107 (42.0%)
Heart failure, n (%)	56 (22.0%)
Chronic obstructive pulmonary disease, n (%)	51 (20.0%)
Obesity, n (%)	43 (16.9%)
Dyslipidemia, n (%)	41 (16.1%)
Chronic kidney disease, n (%)	38 (14.9%)
Coronary artery disease, n (%)	35 (13.7%)

Pulmonary tuberculosis, n (%)	27 (10.6%)
Asthma, n (%)	25 (9.8%)
Malignancy, n (%)	23 (9.0%)
Chronic liver disease, n (%)	22 (8.6%)
Interstitial lung disease, n (%)	20 (7.8%)
Cerebrovascular accident, n (%)	20 (7.8%)
Hypothyroidism, n (%)	10 (3.9%)
Hyperthyroidism, n (%)	5 (1.9%)
No comorbidity, n (%)	40 (15.7%)
Final diagnoses	
Lower respiratory tract infection, n (%)	110 (43.1%)
Acute heart failure, n (%)	100 (39.2%)
Acute-on-chronic heart failure, n (%)	49 (19.2%)
COPD exacerbation, n (%)	56 (22.0%)
Acute coronary syndrome, n (%)	42 (16.5%)
STEMI, n (%)	26 (10.2%)
NSTEMI, n (%)	16 (6.3%)
Acute-on-chronic kidney disease, n (%)	35 (13.7%)
Diabetic ketoacidosis, n (%)	27 (10.6%)
Asthma exacerbation, n (%)	26 (10.2%)
Acute respiratory distress syndrome, n (%)	26 (10.2%)
Hypertensive heart failure, n (%)	24 (9.4%)
Septic shock, n (%)	22 (8.6%)
Decompensated chronic liver disease, n (%)	22 (8.6%)
Interstitial lung disease flare, n (%)	19 (7.5%)
Cardiogenic shock, n (%)	18 (7.1%)
Acute pancreatitis, n (%)	16 (6.3%)
High-altitude pulmonary edema, n (%)	15 (5.9%)
Pneumothorax, n (%)	14 (5.5%)
Pulmonary thromboembolism, n (%)	11 (4.3%)
Panic attack, n (%)	3 (1.2%)
Point-of-care ultrasound findings	
Lung ultrasound: A-profile, n (%)	85 (33.3%)
Lung ultrasound: B-profile, n (%)	132 (51.8%)
Lung ultrasound: C-profile, n (%)	38 (14.9%)
Lung ultrasound: pleural shredding, n (%)	60 (23.5%)
Left ventricular function: normal, n (%)	154 (60.4%)
Left ventricular function: reduced (mild, moderate or severe), n (%) - eye balling method	101 (39.6%)
Mildly reduced, n (%)	63 (24.7%)
Moderately reduced, n (%)	23 (9.0%)

Severely reduced, n (%)	15 (5.9%)
Disposition from the emergency department	
Intensive care unit, n (%)	103 (40.4%)
Ward, n (%)	85 (33.3%)
Discharged, n (%)	52 (20.4%)
Died in the ED, n (%)	15 (5.9%)

TABLE 1: Baseline characteristics of the study population (N = 255)

COPD: chronic obstructive pulmonary disease, ED: emergency department, STEMI: ST-segment elevation myocardial infarction, NSTEMI: non-ST-segment elevation myocardial infarction, MAP: mean arterial pressure.

Percentages are calculated using the total study population (N = 255) as the denominator. Comorbidities and final diagnoses are not mutually exclusive (a patient could have more than one).

Correlation and agreement between ABG and VBG parameters

Table 2 summarizes the correlation and Bland-Altman agreement for all paired parameters across the full cohort. The differences between paired ABG and VBG values were small but statistically significant for all four parameters (paired t-test, df = 254, all $p < 0.001$).

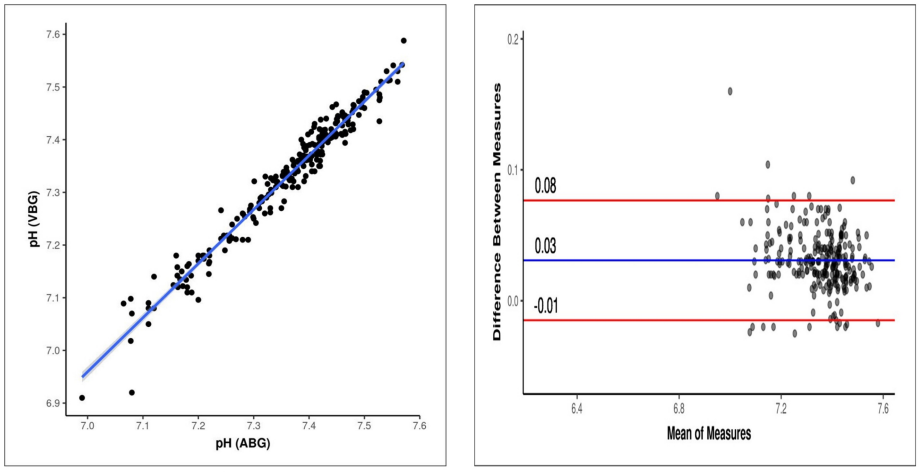
Parameter	ABG, mean ± SD	VBG, mean ± SD	Mean difference, ABG - VBG (95% CI)	95% LOA	Paired t-test: t (p-value)	Pearson r (95% CI)
pH	7.37 ± 0.11	7.34 ± 0.12	0.031 (0.028-0.034)	-0.015 to 0.077	21.12 (<0.001)	0.98 (0.97-0.98)
pCO_2 (mmHg)	33.57 ± 16.70	38.00 ± 17.76	-4.43 (-4.99 to -3.88)	-13.30 to 4.44	-15.65 (<0.001)	0.96 (0.95-0.97)
HCO_3^- (mmol/L)	18.82 ± 6.83	20.34 ± 7.22	-1.52 (-1.71 to -1.32)	-4.63 to 1.60	-15.22 (<0.001)	0.97 (0.96-0.98)
Lactate (mmol/L)	2.34 ± 2.16	2.71 ± 2.42	-0.37 (-0.44 to -0.31)	-1.36 to 0.61	-11.84 (<0.001)	0.98 (0.97-0.98)

TABLE 2: Correlation and Bland-Altman agreement between ABG and VBG parameters (N = 255)

ABG: arterial blood gas, VBG: venous blood gas, LOA: limits of agreement (mean difference ± 1.96 SD).

Mean difference = ABG - VBG. Paired t-test (df = 254) tests for no difference between ABG and VBG; all Pearson correlations had $p < 0.001$.

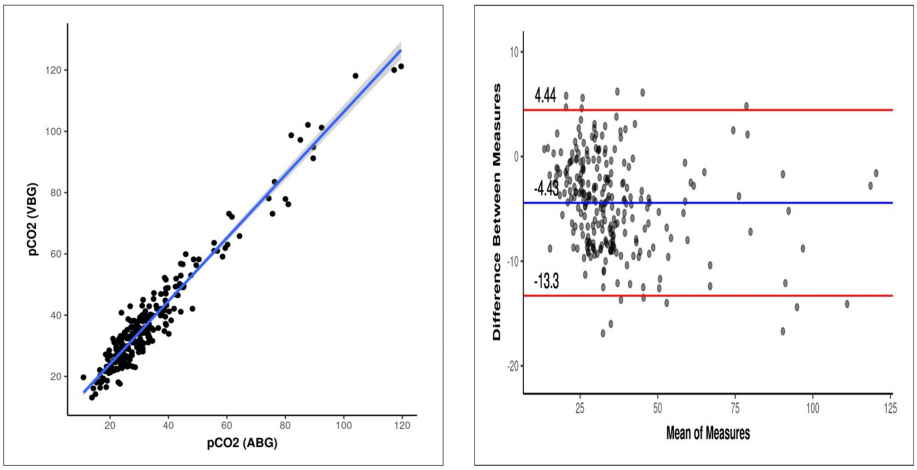
Excellent correlation was observed between ABG and VBG for pH, pCO_2, HCO_3^-, and lactate (Pearson r = 0.96-0.98; all $p < 0.001$; Table 2). The mean differences (ABG - VBG) were small but statistically significant for all four parameters: 0.031 (95% CI 0.028-0.034) for pH, -4.43 mmHg (95% CI -4.99 to -3.88) for pCO_2, -1.52 mmol/L (95% CI -1.71 to -1.32) for HCO_3^-, and -0.37 mmol/L (95% CI -0.44 to -0.31) for lactate (paired t-test, all $p < 0.001$; Table 2). Corresponding regression equations for predicting ABG values from VBG values are also provided in Figures 1-3.



A B

FIGURE 1: Correlation and agreement between arterial blood gas (ABG) and venous blood gas (VBG) pH (N = 255)

(A) Scatter plot showing the correlation between ABG and VBG pH values (Pearson r = 0.98, p < 0.001). The blue line is the fitted linear regression line of VBG on ABG. Regression equation for predicting ABG from VBG: pH[ABG] = 0.497 + 0.936 × pH[VBG]. (B) Bland-Altman plot showing agreement between ABG and VBG pH. Mean difference (bias, ABG - VBG) = 0.03; 95% limits of agreement = -0.01 to 0.08; 246 of 255 (96.5%) paired differences lay within the limits of agreement. The solid blue horizontal line is the mean difference (bias, ABG − VBG) and the two solid red horizontal lines are the upper and lower 95% limits of agreement (mean difference ± 1.96 SD of the differences); each dot is one paired observation.



A B

FIGURE 2: Correlation and agreement between arterial blood gas (ABG) and venous blood gas (VBG) pCO_2, mmHg (N = 255)

(A) Scatter plot showing the correlation between ABG and VBG pCO_2 values (Pearson r = 0.96, p < 0.001). The blue line is the fitted linear regression line of VBG on ABG. Regression equation for predicting ABG from VBG: pCO_2[ABG] = -0.992 + 0.909 × pCO_2[VBG]. (B) Bland-Altman plot showing agreement between ABG and VBG pCO_2. Mean difference (bias, ABG - VBG) = -4.43 mmHg; 95% limits of agreement = -13.30 to 4.44 mmHg; 240 of 255 (94.1%) paired differences lay within the limits of agreement. The solid blue horizontal line is the mean difference (bias, ABG - VBG) and the two solid red horizontal lines are the upper and lower 95% limits of agreement (mean difference ± 1.96 SD of the differences); each dot is one paired observation.

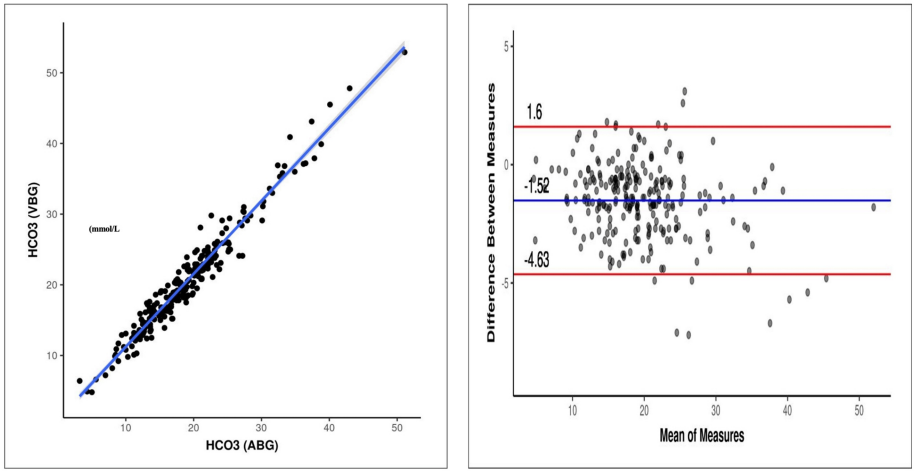


FIGURE 3: Correlation and agreement between arterial blood gas (ABG) and venous blood gas (VBG) HCO_3^-, mmol/L (N = 255).

(A) Scatter plot showing the correlation between ABG and VBG HCO_3^- values (Pearson r = 0.97, p < 0.001). The blue line is the fitted linear regression line of VBG on ABG. Regression equation for predicting ABG from VBG: HCO_3^-[ABG] = 0.049 + 0.923 × HCO_3^-[VBG]. (B) Bland-Altman plot showing agreement between ABG and VBG HCO_3^-. Mean difference (bias, ABG - VBG) = -1.52 mmol/L; 95% limits of agreement = -4.63 to 1.60 mmol/L; 242 of 255 (94.9%) paired differences lay within the limits of agreement. The solid blue horizontal line is the mean difference (bias, ABG - VBG) and the two solid red horizontal lines are the upper and lower 95% limits of agreement (mean difference ± 1.96 SD of the differences); each dot is one paired observation.

Correlation and agreement in shock vs non-shock patients

Forty-four patients (17.3%) were classified into the shock group (hypotension, MAP <65 mmHg) and 211 (82.7%) into the non-shock group. Correlation and agreement remained robust in both groups for all parameters, as shown in Table *3* (Pearson r = 0.951-0.979 in the shock group and 0.973-0.983 in the non-shock group; all p < 0.001). Within each group, ABG and VBG values differed significantly (paired t-test, all p < 0.001). The mean ABG-VBG difference was numerically larger in the shock group for pH (0.038 vs 0.029), pCO_2 (-6.56 vs -3.99 mmHg), and lactate (-0.55 vs -0.34 mmol/L) and similar for HCO_3^- (−1.53 vs −1.51 mmol/L). The between-group difference was statistically significant only for pCO_2 (Welch t = -2.99, df = 55, p = 0.004) and not for pH (p = 0.12), HCO_3^- (p = 0.93), or lactate (p = 0.14). The absolute between-group difference in bias for pCO_2 was small (2.6 mmHg) and was not considered clinically significant for ventilatory assessment.

Parameter	Group	ABG, mean ± SD	VBG, mean ± SD	Mean difference, ABG - VBG (95% CI)	95% LOA	Paired t-test: t (df); p	Pearson r (95% CI)	Shock vs non-shock: comparison of mean differences
pH	Shock	7.31 ± 0.11	7.27 ± 0.13	0.038 (0.027-0.048)	-0.029 to 0.105	7.31 (43); <0.001	0.971 (0.947-0.984)	Welch t = 1.57 (df 49), p = 0.12
	No shock	7.38 ± 0.11	7.35 ± 0.11	0.029 (0.027-0.032)	−0.010 to 0.069	21.23 (210); <0.001	0.983 (0.978-0.987)	
pCO_2 (mmHg)	Shock	30.97 ± 13.90	37.52 ± 16.45	-6.56 (-8.19 to -4.93)	-17.06 to 3.95	-8.11 (43); <0.001	0.951 (0.912-0.973)	Welch t = -2.99 (df 55), p = 0.004
	No shock	34.11 ± 17.20	38.10 ± 18.06	-3.99 (-4.56 to -3.42)	-12.25 to 4.26	-13.76 (210); <0.001	0.973 (0.964-0.979)	
HCO_3^- (mmol/L)	Shock	16.04 ± 6.93	17.58 ± 7.28	-1.53 (-1.99 to -1.08)	-4.47 to 1.40	-6.80 (43); <0.001	0.979 (0.962-0.989)	Welch t = -0.09 (df 66), p = 0.93
	No shock	19.40 ± 6.68	20.91 ± 7.09	-1.51 (-1.73 to -1.29)	-4.67 to 1.65	-13.61 (210); <0.001	0.974 (0.966-0.980)	
Lactate (mmol/L)	Shock	4.18 ± 3.34	4.73 ± 3.85	-0.55 (-0.84 to -0.27)	-2.39 to 1.28	-3.91 (43); <0.001	0.976 (0.956-0.987)	Welch t = -1.51 (df 45), p = 0.14
	No shock	1.95 ± 1.58	2.29 ± 1.74	-0.34 (-0.38 to -0.29)	-1.01 to 0.34	-14.16 (210); <0.001	0.983 (0.977-0.987)	

TABLE 3: ABG-VBG agreement in shock (n = 44) and non-shock (n = 211) patients

ABG: arterial blood gas, VBG: venous blood gas, LOA: limits of agreement (mean difference ± 1.96 SD).

Shock group (n = 44): MAP <65 mmHg; non-shock group (n = 211): MAP ≥65 mmHg. Mean difference = ABG − VBG. Paired t-test (df = n - 1) tests for no difference between ABG and VBG within each group; all Pearson correlations had p < 0.001. Shock vs non-shock: comparison of the mean ABG - VBG difference between the two groups (Welch's independent-samples t-test; t, df and p-value shown).

Diagnostic performance of VBG-based acid-base diagnosis

Table 4 presents the diagnostic accuracy of VBG-based classification of acid-base disorders against ABG-based diagnosis as the gold standard. Using ABG as the reference standard, VBG-based classification correctly identified metabolic acidosis with 96.6% (113/117) sensitivity and 83.3% (115/138) specificity (accuracy 228/255, 89.4%; Cohen's κ = 0.789), respiratory acidosis with 100% (50/50) sensitivity and 89.3% (183/205) specificity (accuracy 233/255, 91.4%; κ = 0.765), and metabolic alkalosis with 77.3% (17/22) sensitivity and 99.1% (231/233) specificity (accuracy 248/255, 97.3%; κ = 0.814). Performance was comparatively lower for respiratory alkalosis with 66.1% (74/112) sensitivity and 98.6% (141/143) specificity (accuracy 215/255, 84.3%; κ = 0.670). The 95% CIs for all measures are given in Table 4.

Disorder	Sensitivity, % (95% CI)	Specificity, % (95% CI)	PPV, % (95% CI)	NPV, % (95% CI)	Accuracy, % (95% CI)	Cohen's κ (95% CI)
Metabolic acidosis	96.6 (91.5-99.1)	83.3 (76.0-89.1)	83.1 (75.7-89.0)	96.6 (91.6-99.1)	89.4 (85.0-92.9)	0.789 (0.715-0.864)
Respiratory acidosis	100.0 (92.9-100.0)	89.3 (84.2-93.2)	69.4 (57.5-79.8)	100.0 (98.0-100.0)	91.4 (87.2-94.5)	0.765 (0.674-0.856)
Metabolic alkalosis	77.3 (54.6-92.2)	99.1 (96.9-99.9)	89.5 (66.9-98.7)	97.9 (95.1-99.3)	97.3 (94.4-98.9)	0.814 (0.681-0.948)
Respiratory alkalosis	66.1 (56.5-74.7)	98.6 (95.0-99.8)	97.4 (90.8-99.7)	78.8 (72.0-84.5)	84.3 (79.3-88.6)	0.670 (0.580-0.760)
Arterial hyperlactatemia (venous lactate ≥2.4 mmol/L vs arterial lactate ≥2 mmol/L)	95.3 (89.4-98.5)	99.3 (96.3-100.0)	99.0 (94.7-100.0)	96.7 (92.5-98.9)	97.6 (94.9-99.1)	-

TABLE 4: Diagnostic performance of peripheral VBG for acid-base disorder classification and of venous lactate for arterial hyperlactatemia (ABG as gold standard; N = 255)

ABG: arterial blood gas, VBG: venous blood gas, PPV: positive predictive value, NPV: negative predictive value.

The lactate row refers to the Youden-optimal VBG cut-off (≥2.4 mmol/L) against arterial lactate ≥2 mmol/L (Cohen's kappa not applicable). 95% CIs for sensitivity, specificity, PPV, NPV, and accuracy were calculated using the exact (Clopper-Pearson) method; 95% CIs for Cohen's kappa were calculated from its large-sample standard error. All Cohen's kappa values were statistically significant ($p < 0.001$).

For venous lactate as a screening tool for arterial hyperlactatemia (⩾2 mmol/L; 107 of 255 patients (42.0%) had arterial lactate ⩾2 mmol/L), the optimal VBG cut-off, defined by the maximum Youden index (J = 0.947), was ⩾2.4 mmol/L (AUROC = 0.996, 95% CI: 0.992-1.000, $p < 0.001$; Figure *4*). At this threshold, VBG-based screening achieved 95.3% (102/107) sensitivity (95% CI: 89.4%-98.5%), 99.3% (147/148) specificity (95% CI: 96.3%-100.0%), PPV 99.0% (102/103; 95% CI: 94.7%-100.0%), NPV 96.7% (147/152; 95% CI: 92.5%-98.9%), and diagnostic accuracy 97.6% (249/255; 95% CI: 94.9%-99.1%). The positive likelihood ratio was 141.1 (95% CI: 20.0-995.4), the negative likelihood ratio 0.05 (95% CI: 0.02-0.11), and the diagnostic odds ratio 2999 (95% CI: 345-26052). At a cut-off of ⩾2.5 mmol/L, sensitivity was 92.5% (99/107) and specificity 99.3% (147/148); at the arterial threshold itself (⩾2.0 mmol/L), sensitivity was 100% (107/107) and specificity 88.5% (131/148).

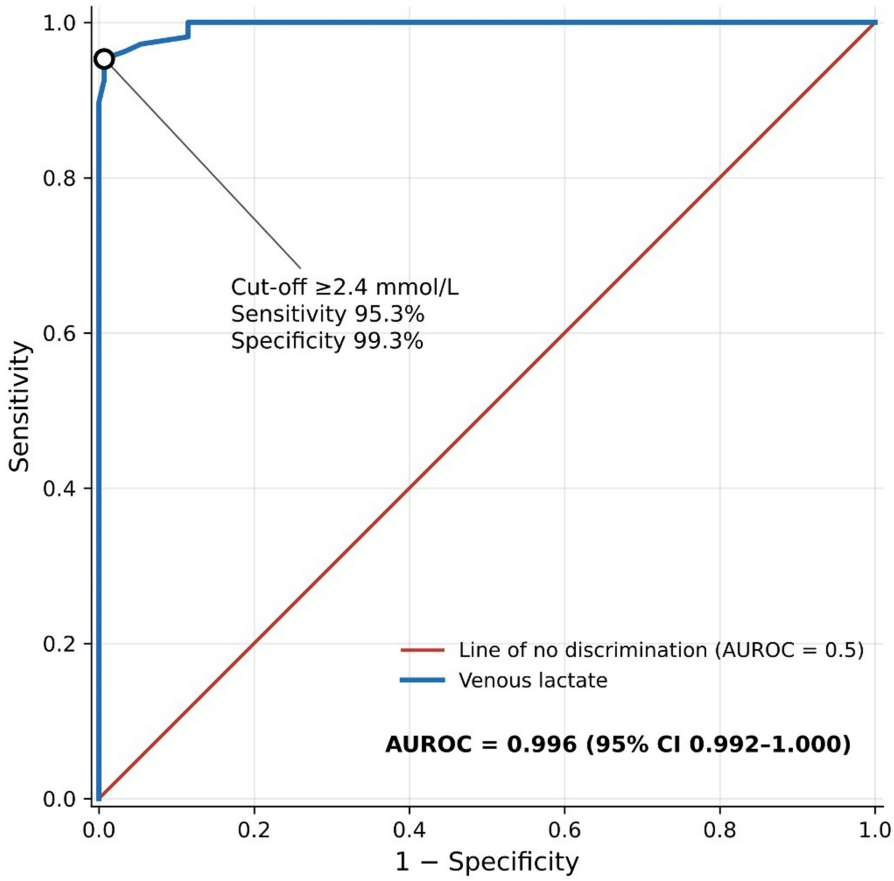


FIGURE 4: Receiver operating characteristic (ROC) curve of venous lactate for predicting arterial hyperlactatemia (arterial lactate ≥2 mmol/L) (N = 255)

The blue line is the ROC curve of venous lactate for predicting arterial hyperlactatemia (arterial lactate ≥2 mmol/L), and the red diagonal line is the line of no discrimination (AUROC = 0.5). The marked point is the Youden-optimal cut-off of ≥2.4 mmol/L (sensitivity 95.3%, 102/107; specificity 99.3%, 147/148). The AUROC was 0.996 (95% CI 0.992-1.000; p < 0.001).

AUROC: area under the receiver operating characteristic curve.

Discussion

This study demonstrates that peripheral VBG parameters show excellent correlation and small mean differences compared with ABG values in a broad, heterogeneous cohort of ED patients with acute-onset dyspnea. To our knowledge, this is the largest single-center study comparing ABG and VBG in an unselected dyspneic ED population in India, and one of the few to concurrently assess the diagnostic accuracy of VBG-based acid-base classification alongside a validated lactate-based screening threshold.

Our findings for pH are consistent with international evidence. The mean ABG-VBG pH difference of 0.03 matches Zeserson et al. (0.03, correlation coefficient 0.94) [10] and the meta-analysis by Byrne et al. (mean difference 0.03 pH units) [8]. Our correlation coefficient of 0.98 compares favorably with White et al. (correlation coefficient >0.85) [15] and the externally validated conversion-model study by Jörg et al., confirming that simple venous-to-arterial adjustment models perform acceptably in undifferentiated ED patients [11]. The LOAs ranged from -0.01 to 0.08 pH units, indicating relatively narrow differences between arterial and venous pH measurements. However, as clinically acceptable LOAs were not prespecified, the clinical interchangeability of the two measurements cannot be conclusively established.

For pCO_2, we found a strong correlation (r = 0.96) with 94.1% of observations within the LOA (-13.30 to 4.44 mmHg). Prior studies have noted less satisfactory pCO_2 agreement in COPD, where a 2022 meta-analysis found persistent arteriovenous differences [12]. Our results are comparable to Zeserson et al. (mean difference 4.8 mmHg, correlation coefficient 0.93) [10] and support venous pCO_2 as a reasonable estimate of

arterial pCO_2 in ED triage, provided hypercarbia is confirmed with ABG when decisions depend critically on the exact value [11,24].

Bicarbonate and lactate showed the tightest agreement in our study (r = 0.97 and 0.98, respectively). By comparison, the Arterio-VENouS intra-Subject agreement for blood gases (AVENSIS) study by Nanjayya et al., which compared central venous with arterial samples in intensive care patients, found acceptable agreement for pH but poor agreement for pCO_2 [20]. For lactate, our findings agree with recent literature: Jose et al. found good agreement between arterial and venous lactate in sepsis [18], and Velissaris et al. and Mahmoodpoor et al. similarly found strong correlation, with modest bias, between arterial and venous lactate in septic and critically ill cohorts [16,17]; and a 2025 retrospective analysis by Li et al. confirmed a strong linear relationship between arterial and venous lactate, with wider agreement limits at higher concentrations, echoing our finding that concordance narrows with worsening physiological derangement [22]. These findings support the use of venous HCO_3^- and lactate as estimates of their arterial counterparts in most ED patients, although the lactate limits of agreement (-1.36 to 0.61 mmol/L) mean that individual values close to a decision threshold should be interpreted with caution.

The persistence of robust correlation in hypotensive patients (n = 44, MAP <65 mmHg) is particularly notable. While mean differences were slightly greater in shock patients (pH difference 0.038 vs 0.029; pCO_2 difference 6.56 vs 3.99 mmHg; the between-group difference was statistically significant only for pCO_2), Pearson r remained ⩾0.95 for all parameters. This is reassuring considering the classic observation by Adrogue et al. that arteriovenous acid-base concordance can deteriorate in severe circulatory failure [25], although more recent data from Rudkin et al. found no significant relationship between systolic blood pressure and the arterial-peripheral venous difference for pH, pCO_2, or bicarbonate [19].

The high sensitivity of VBG for metabolic acidosis (96.6%) and respiratory acidosis (100%) is clinically meaningful, as missed acidemia carries the greatest potential for harm. VBG was less sensitive for alkalotic disorders (respiratory alkalosis 66.1%, metabolic alkalosis 77.3%), reflecting its tendency to slightly underestimate pH and overestimate pCO_2 relative to ABG. Clinicians should exercise caution using VBG to exclude alkalotic disorders and maintain a low threshold for confirmatory ABG when clinical suspicion for alkalosis is high.

The excellent diagnostic performance of venous lactate (AUROC = 0.996) at a cut-off of ⩾2.4 mmol/L for predicting arterial hyperlactatemia (sensitivity 95.3%, specificity 99.3%) is consistent with the systematic review by van Tienhoven et al. [21] and with Niczewski et al., who found arterial and venous lactate similarly predictive of short-term mortality in dyspneic ED patients [26], and Li et al., who reported near-identical prognostic accuracy for arterial and venous lactate despite imperfect numerical agreement [22]. These data support venous lactate as a practical screening tool for tissue hypoperfusion in resource-constrained EDs, though absolute values should be interpreted cautiously at higher concentrations. Because the cut-off was derived and evaluated in the same dataset, its performance requires confirmation in an independent cohort.

From a practical standpoint in the Indian context, where ED overcrowding, limited personnel, and high patient volumes often preclude routine ABG sampling, peripheral VBG may serve as a pragmatic alternative for initial patient assessment and triage. This approach reduces the risk of procedural complications such as hematoma, pseudoaneurysm, and compartment syndrome [5,6], the substantial pain associated with arterial puncture that has prompted trials of analgesic strategies [7], and needlestick injuries. A prehospital cohort by Walther et al. further showed that blood gas analysis (pH, lactate, and CO_2) obtained outside the hospital carries prognostic information for seven-day mortality [27]. Complementary bedside tools such as point-of-care lung ultrasound have also been shown to accelerate diagnosis in dyspneic ED patients [28], and integrating such low-resource strategies alongside VBG may further streamline emergency dyspnea pathways, an approach also explored by Körver et al., who found that VBG combined with pulse oximetry could inform ED disposition decisions without ABG confirmation in most patients with respiratory complaints [9].

This study has several limitations. First, only a single paired ABG-VBG sample was obtained per patient, precluding assessment of temporal reproducibility. Second, the tourniquet time during VBG collection was not standardized or recorded. Third, the single-center design at a tertiary academic hospital in northern India may limit generalizability to other healthcare settings with different patient populations and disease spectra. Fourth, ED diagnoses were based on the treating physician's assessment at the time of presentation and may not have reflected the definitive inpatient diagnosis. Fifth, patients with multiple concurrent diagnoses could not always be classified into discrete subgroups. The shock and non-shock subgroups were not prespecified, shock was defined solely by blood pressure criteria at presentation (MAP <65 mmHg), so the subgroup findings are more susceptible to confounding and should be considered exploratory. Lastly, the VBG lactate cut-off was derived and evaluated in the same sample without external validation, which may overestimate its diagnostic performance.

Conclusions

In adult patients presenting to the ED with acute-onset dyspnea, peripheral VBG correlated strongly with ABG for pH, pCO_2, HCO_3^-, and lactate, and the mean differences between the two were small but

statistically significant for all four parameters ($p < 0.001$). However, strong correlation and a small average bias do not guarantee agreement in an individual patient: the limits of agreement were narrow for pH but wide for pCO_2 and lactate. VBG-based classification was highly sensitive for metabolic and respiratory acidosis but less sensitive for metabolic and respiratory alkalosis, which it may miss. A venous lactate of ≥2.4 mmol/L identified arterial hyperlactatemia with high accuracy, although this cut-off requires external validation. Correlation remained strong in patients with shock. Peripheral VBG is therefore a practical, safer, and less invasive first-line test for the initial evaluation of dyspneic ED patients, particularly in resource-constrained settings, and is well suited to screening for acidosis and hyperlactatemia. ABG remains necessary when alkalosis is suspected, when an exact pCO_2 value will guide management, and when oxygenation must be assessed.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Anand Gangdev, Bharat Bhardwaj, Naman Agrawal

Acquisition, analysis, or interpretation of data: Anand Gangdev, Rinku Meena, Anmol Chanda

Drafting of the manuscript: Anand Gangdev, Rinku Meena, Anmol Chanda, Naman Agrawal

Critical review of the manuscript for important intellectual content: Bharat Bhardwaj, Naman Agrawal

Supervision: Bharat Bhardwaj, Naman Agrawal

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. Institutional Ethics Committee, All India Institute of Medical Sciences, Rishikesh issued approval AIIMS/IEC/21/614. **Animal subjects:** All authors have confirmed that this study did not involve animal subjects or tissue. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

References

1. Berliner D, Schneider N, Welte T, Bauersachs J: The differential diagnosis of dyspnea. Dtsch Arztebl Int. 2016, 113:834-45. 10.3238/arztebl.2016.0834
2. Parshall MB, Schwartzstein RM, Adams L, et al.: An official American Thoracic Society statement: update on the mechanisms, assessment, and management of dyspnea. Am J Respir Crit Care Med. 2012, 185:435-52. 10.1164/rccm.201111-2042ST
3. Prekker ME, Feemster LC, Hough CL, et al.: The epidemiology and outcome of prehospital respiratory distress. Acad Emerg Med. 2014, 21:543-50. 10.1111/acem.12380
4. Patel KN, Gandhi SP, Sutariya HC: Radial artery pseudoaneurysm: a rare complication after a single arterial puncture for blood-gas analysis. Indian J Crit Care Med. 2016, 20:622-6. 10.4103/0972-5229.192066
5. Garner O, Nunna K, Braun A: Development of compartment syndrome after radial artery puncture in a patient with acute hypoxemic respiratory failure due to COVID-19. Case Rep Med. 2022, 2022:8241057. 10.1155/2022/8241057
6. Ikuta A, Kubo S, Osakada K, et al.: Predictors of success and puncture site complications in the distal radial approach. Heart Vessels. 2023, 38:147-56. 10.1007/s00380-022-02152-6
7. Yasuo S, Hayashi M, Suda C, Kataoka Y, Taito S, Imai E, Sazanami K: Efficacy of local anesthesia for radial artery puncture pain: a systematic review and network meta-analysis. Cureus. 2024, 16:e64682. 10.7759/cureus.64682
8. Byrne AL, Bennett M, Chatterji R, Symons R, Pace NL, Thomas PS: Peripheral venous and arterial blood gas analysis in adults: are they comparable? A systematic review and meta-analysis. Respirology. 2014, 19:168-75. 10.1111/resp.12225
9. Körver S, Eurlings MB, Merry AH, Gronenschild MH, Raijmakers MT, Latten GH: Comparison of peripheral venous and arterial blood gas in management of patients with respiratory complaints in the emergency department: a prospective observational cohort study. PLoS One. 2025, 20:e0330190. 10.1371/journal.pone.0330190
10. Zeserson E, Goodgame B, Hess JD, et al.: Correlation of venous blood gas and pulse oximetry with arterial blood gas in the undifferentiated critically ill patient. J Intensive Care Med. 2018, 33:176-81. 10.1177/0885066616652597
11. Jörg M, Öster M, Wretborn J, Wilhelms DB: Agreement of pCO(2) in venous to arterial blood gas conversion

models in undifferentiated emergency patients. Intensive Care Med Exp. 2023, 11:80. 10.1186/s40635-023-00564-w

12. Sheng F, Chen L, Lin H, Wu H: Systematic review and meta-analysis: value of venous blood gas in the diagnosis of acute exacerbation of chronic obstructive pulmonary disease in Emergency Department. Ann Palliat Med. 2022, 11:1473-81. 10.21037/apm-22-336
13. Tang S, Mei Z, Huang D, Liu L, Yang L, Yin D, Hu L: Comparative analysis of hemoglobin, potassium, sodium, and glucose in arterial blood gas and venous blood of patients with COPD. Sci Rep. 2024, 14:5194. 10.1038/s41598-024-55992-9
14. Immadisetty SK, Patange AP: Comparison of blood gas and acid-base measurement in arterial and venous blood samples in patients with diabetic ketoacidosis. Biomed Pharmacol J. 2022, 15:413-420. 10.13005/bpj/2381
15. White HD, Vazquez-Sandoval A, Quiroga PF, Song J, Jones SF, Arroliga AC: Utility of venous blood gases in severe sepsis and septic shock. Proc (Bayl Univ Med Cent). 2018, 31:269-75. 10.1080/08998280.2018.1460133
16. Mahmoodpoor A, Shadvar K, Sanaie S, Golzari SE, Parthvi R, Hamishehkar H, Nader ND: Arterial vs venous lactate: correlation and predictive value of mortality of patients with sepsis during early resuscitation phase. J Crit Care. 2020, 58:118-24. 10.1016/j.jcrc.2019.05.019
17. Velissaris D, Karamouzos V, Pantzaris ND, Kyriakopoulou O, Gogos C, Karanikolas M: Relation between central venous, peripheral venous and arterial lactate levels in patients with sepsis in the emergency department. J Clin Med Res. 2019, 11:629-34. 10.14740/jocmr3897
18. Jose JM, Cherian A, Bidkar PU, Mohan VK: The agreement between arterial and venous lactate in patients with sepsis. Int J Clin Pract. 2021, 75:e14296. 10.1111/ijcp.14296
19. Rudkin SE, Anderson CL, Grogan TR, Elashoff DA, Treger RM: Assessing acid-base status in circulatory failure: relationship between arterial and peripheral venous blood gas measurements in hypovolemic shock. J Intensive Care Med. 2020, 35:511-8. 10.1177/0885066618762335
20. Nanjayya VB, McCracken P, Vallance S, et al.: Arterio-VENouS intra subject agreement for blood gases within intensive care: the AVENSIS study. J Intensive Care Soc. 2020, 21:64-71. 10.1177/1751143719840259
21. van Tienhoven AJ, van Beers CA, Siegert CE: Agreement between arterial and peripheral venous lactate levels in the ED: a systematic review. Am J Emerg Med. 2019, 37:746-50. 10.1016/j.ajem.2019.01.034
22. Li Y, He X, Li D, Yuan X, Xu J, Yang J: Bland-Altman plot to assess the consistency of arterial and venous blood lactate in the emergency room: a retrospective cohort study. Sci Rep. 2025, 15:2143. 10.1038/s41598-025-85104-0
23. Bland JM, Altman DG: Statistical methods for assessing agreement between two methods of clinical measurement. Lancet. 1986, 1:307-310. 10.1016/S0140-6736(86)90837-8
24. Schütz N, Roth D, Schwameis M, Röggla M, Domanovits H: Can venous blood gas be used as an alternative to arterial blood gas in intubated patients at admission to the emergency department? A retrospective study. Open Access Emerg Med. 2019, 11:305-12. 10.2147/OAEM.S228420
25. Adrogué HJ, Rashad MN, Gorin AB, Yacoub J, Madias NE: Assessing acid-base status in circulatory failure . N Engl J Med. 1989, 320:1312-6. 10.1056/NEJM198905183202004
26. Niczewski M, Gawęda S, Kluszczyk P, et al.: The predictive role of lactate in the emergency department in patients with severe dyspnea. Emerg Med Int. 2024, 2024:6624423. 10.1155/2024/6624423
27. Walther LH, Lassen AT, Mogensen CB, Christensen EF, Mikkelsen S: Prehospital blood gas analyses in acute patients treated by a ground-based physician-manned emergency unit: a cohort study. Scand J Trauma Resusc Emerg Med. 2023, 31:102. 10.1186/s13049-023-01170-1
28. Ciumanghel I, Barbuta E, Ciumanghel AI, Buzincu I, Grigorasi G, Cimpoesu D: Point-of-care lung ultrasound - a rapid and reliable diagnostic tool for emergency physicians treating patients with acute dyspnea in high-volume emergency departments. Emerg Radiol. 2025, 32:329-38. 10.1007/s10140-025-02343-4