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Agreement between arterial blood gas analyser and venous central laboratory measurements of sodium, potassium, glucose and haemoglobin in high-acuity emergency patients: a cross-sectional method-comparison study from South India.

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Abstract

Background: Blood gas analysers in emergency departments report electrolytes, glucose and haemoglobin within minutes, whereas central laboratory results arrive later. Whether the two can be used interchangeably in critically ill patients is unsettled and depends on local instruments. We compared arterial blood gas analyser results with venous central laboratory results in high-acuity emergency patients. ***Methods:*** In this prospective cross-sectional method-comparison study at the emergency department of Raichur Institute of Medical Sciences, Karnataka, 300 consecutive adults triaged as Australasian Triage Scale category 1 or 2 who required arterial blood gas analysis were enrolled. Paired arterial (ABL800 blood gas analyser) and venous (Cormay Accent 200 laboratory autoanalyser) samples were drawn before treatment. Sodium, potassium, glucose and haemoglobin were compared with the paired t-test, and mean bias was judged against US Clinical Laboratory Improvement Amendments (CLIA) allowable error. ***Results:*** Of 300 patients, 172 (57.3%) were female and 297 (99.0%) were triage category 2. The blood gas analyser read lower than the laboratory for sodium (131.08 vs 135.90 mmol/L; difference 4.82 mmol/L), potassium (4.0 vs 4.6 mmol/L; difference 0.60 mmol/L) and haemoglobin (11.29 vs 11.41 g/dL; difference 0.12 g/dL), and higher for glucose (152.77 vs 140.86 mg/dL; difference 11.91 mg/dL). All differences were statistically significant ($p < 0.001$; haemoglobin $p = 0.031$). Sodium and potassium biases exceeded CLIA limits; glucose and haemoglobin biases fell within them. ***Conclusions:*** In this setting, blood gas analyser sodium and potassium were systematically lower than laboratory values by margins large enough to alter clinical decisions, and should not be used interchangeably without local correction and laboratory confirmation. Mean glucose and haemoglobin bias was acceptable, but agreement for individual patients requires confirmation by Bland–Altman analysis.

Keywords: Blood Gas Analysis; Point-of-Care Testing; Ion-Selective Electrodes; Potassium; Triage; Emergency Service, Hospital.

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INTRODUCTION

Acutely ill patients arriving in the emergency department need decisions about electrolytes, glycaemia and anaemia long before routine laboratory reports return. Blood gas analysis, traditionally performed on arterial blood to assess oxygenation, ventilation and acid–base status, has expanded into a bench-top panel that also reports sodium, potassium, glucose and haemoglobin within two or three minutes of sampling. For the emergency physician, this turns a single arterial puncture into a near-complete biochemical screen. The question that follows is practical and has direct consequences for patient safety: can these rapid values be trusted to the same degree as those produced by the central laboratory? Most of the published debate about blood gas analysis in emergency care has addressed a different question, namely whether a peripheral venous sample can replace an arterial one for acid–base assessment.

The answer is now reasonably clear. Pooled analyses show that venous and arterial pH and bicarbonate agree closely enough to be interchangeable in patients who are not in shock, whereas venous carbon dioxide tension agrees too poorly to substitute for the arterial value, although a normal venous value usefully excludes arterial hypercapnia.1,2,3 Similar conclusions have been reached in exacerbations of chronic obstructive pulmonary disease, in diabetic ketoacidosis and in pathologically diverse emergency populations.4,5,6,7 The arteriovenous difference widens modestly in hypotensive patients.8 The arterial sample therefore retains its place where oxygenation or carbon dioxide tension must be known

precisely, and arterial puncture, though usually safe, carries pain and rare but serious complications such as pseudoaneurysm after repeated attempts.9

Once an arterial sample has been drawn, the electrolyte, glucose and haemoglobin results on the blood gas report are available at no extra cost or delay. Their speed matters. Point-of-care testing shortens turnaround time substantially and, when integrated into departmental workflow, can reduce emergency department length of stay.10,11,12 In one recent audit from a university hospital, blood gas electrolytes were available a median of ten minutes before laboratory values;13 in hospitals where samples queue behind routine work, the gap is often far longer. If blood gas values could be accepted as equivalent, emergency treatment of hyperkalaemia, hyponatraemia or hypoglycaemia could begin earlier.

The stakes differ by analyte, but each is high in the emergency setting. Potassium governs the immediate management of patients with renal failure, cardiac arrhythmia and diabetic ketoacidosis, where a few tenths of a millimole per litre separate observation from emergency treatment and determine when insulin can safely be started. Sodium errors of a few millimoles per litre can change the diagnosis of hyponatraemia, the calculated anion gap and the target rate of correction. Glucose values drive the recognition of hypoglycaemia in patients with altered consciousness and the initiation of insulin in hyperglycaemic crises, and haemoglobin informs early decisions about bleeding and transfusion. A systematic bias that is invisible to the clinician reading a single report can therefore translate directly into an inappropriate treatment decision, and the direction of the bias matters as much as its size.

Equivalence, however, cannot be assumed. Blood gas analysers measure electrolytes in undiluted whole blood with direct ion-selective electrodes, whereas most central laboratories measure serum or plasma after automated dilution with indirect ion-selective electrodes. The indirect method assumes a normal water fraction of plasma, so it misreports sodium when protein concentrations are abnormal, a situation that is common in critical illness. In intensive care patients, laboratory sodium exceeded blood gas sodium by about 2 mmol/L on average, and the gap widened as albumin fell;14 in hypoalbuminaemic neonates the difference was large enough to misclassify dysnatraemia,15 and in patients with cirrhosis the choice of method changes transplant priority scores.16 These discrepancies propagate into derived indices: in one intensive care cohort, the anion gap moved across the reference limit for nearly half of patients depending on which analyser supplied the electrolytes.17

Clinical comparison studies have produced conflicting verdicts. In an Indian intensive care unit, blood gas sodium was substantially lower than laboratory sodium while potassium did not differ.18 An emergency department study from China found statistically significant but analytically acceptable biases for both electrolytes and judged blood gas values reliable,19 whereas a Turkish intensive care study concluded that sodium could not be used interchangeably and potassium only with laboratory confirmation.20 An Australian emergency department study found most paired results within clinically acceptable limits,21 but a large paediatric series and a recent analysis of more than 6000 emergency pairs found agreement for sodium to be poor.22,23 A 2026 Indian emergency study reported acceptable electrolyte agreement but unacceptable haemoglobin error.24 For glucose, a systematic review found blood gas analysers more accurate than capillary glucose meters, although accuracy deteriorated near the hypoglycaemic range.25

Three features explain much of this heterogeneity. First, results depend on the specific analysers, calibration and pre-analytical handling at each site, including the type of heparin in the syringe, which can introduce clinically important negative bias in electrolytes.26 Second, many studies compare arterial blood on one analyser with venous blood on another, so that physiological, sample-matrix and analytical differences are combined in a single estimate. Third, several studies relied on correlation or significance testing, which cannot establish whether two methods agree closely enough to be used interchangeably.27 Findings from one hospital therefore cannot be transferred to another, and each institution that relies on point-of-care values needs its own evidence.

No such evidence has been reported from emergency departments in Raichur, where laboratory turnaround is a recognised constraint and the case mix is dominated by respiratory, renal and septic presentations. Five-level triage scales such as the Australasian Triage Scale are widely used to prioritise emergency patients, although the inter-rater reliability of this scale is only moderate.28,29 Patients assigned its two highest urgency categories are the group in whom early decisions matter most and in whom blood gas analysis is most often requested. We hypothesised that sodium, potassium, glucose and haemoglobin measured on the emergency blood gas analyser would differ systematically from venous central laboratory values, and that at least some of these differences would exceed accepted limits of analytical acceptability.

OBJECTIVES

The primary objective was to measure sodium, potassium, glucose and haemoglobin on an arterial blood gas analyser and on paired venous samples analysed by the central laboratory autoanalyser in adult emergency patients triaged as Australasian Triage Scale category 1 or 2. The secondary objective was to compare the paired values, quantify the mean

difference (bias) between the two methods for each analyte, and judge whether that bias fell within US Clinical Laboratory Improvement Amendments (CLIA) criteria for acceptable analytical performance.

MATERIALS AND METHODS

Study design and setting

This was a prospective, comparative cross-sectional method-comparison study, reported in accordance with the STROBE statement and, for agreement analysis, the Guidelines for Reporting Reliability and Agreement Studies (GRRAS).30,31 It was conducted in the emergency department of *Raichur institute of medical sciences (Rims), Raichur, Karnataka, India* Patients were enrolled over a period of one year from ethical approval.

Participants

All patients presenting to the emergency department were triaged at arrival by trained emergency nurses using the Australasian Triage Scale (ATS). High acuity was defined as ATS category 1 (immediately life-threatening, to be seen immediately; for example cardiac or respiratory arrest, extreme respiratory distress, systolic blood pressure below 80 mmHg in adults, unresponsiveness or ongoing seizure) or ATS category 2 (imminently life-threatening, to be seen within 10 minutes; for example severe respiratory distress, circulatory compromise, chest pain of likely cardiac origin, heart rate below 50 or above 150 per minute, acute hemiparesis, major trauma or very severe pain). Patients aged above 16 years in either category were eligible when the treating team judged arterial blood gas analysis necessary for initial assessment. Patients or relatives who declined consent were excluded. Consecutive eligible patients were enrolled until the target sample was reached.

Sample collection

Paired samples were obtained as close together in time as possible and before any treatment was started, including intravenous fluids, insulin, electrolyte replacement or blood products. The investigator performed all arterial punctures. After a modified Allen test, 0.5 to 1 mL of blood was drawn from the radial artery into a 3 mL plastic syringe minimally heparinised, expelled of air bubbles and capped. The venous sample was drawn by the duty nurse into a plastic syringe at the time of peripheral intravenous cannulation, transferred to a plain (non-anticoagulated) vial and allowed to clot to yield serum. Both analysers were located in the emergency laboratory within the emergency department complex, and transport time to the laboratory was under one minute for both samples.

Laboratory methods

Arterial samples were analysed on an ABL800 blood gas analyser (Radiometer, Copenhagen, Denmark), which measures sodium and potassium in whole blood with direct ion-selective electrodes, glucose with an amperometric enzymatic electrode, and total haemoglobin by spectrophotometric co-oximetry. Venous samples were analysed on a Cormay Accent 200 clinical chemistry autoanalyser (Cormay, Poland). The central laboratory participated in an external quality assessment programme throughout the study; the blood gas analyser was maintained and calibrated according to the manufacturer's schedule but was not enrolled in an external quality assessment scheme.

Variables and outcome

For each patient, sex, district of residence, working diagnosis and ATS category were recorded on a structured proforma together with paired values of sodium (mmol/L), potassium (mmol/L), glucose (mg/dL) and haemoglobin (g/dL). The primary outcome was the mean difference (bias) between the two methods for each analyte, expressed as blood gas analyser minus laboratory value, so that a negative bias indicates a lower blood gas reading.

Sample size

Sample size was calculated from the sodium values reported by Zhang and colleagues (blood gas 137.89 ± 5.44 mmol/L versus laboratory 140.93 ± 5.50 mmol/L).19 Using $n = 2(Z\alpha + Z\beta)^2\sigma^2/d^2$ with a two-sided α of 0.05 ($Z\alpha = 1.96$), power of 80% ($Z\beta = 0.84$), a pooled variance of 29.91 and an expected difference of 3.04 mmol/L (140.93 − 137.89), a minimum of 52 patients was required; the corresponding calculation for potassium (3.77 ± 0.44 versus 4.2 ± 0.55 mmol/L) required 21. To allow subgroup description across the varied emergency case mix and to exceed the 100 pairs commonly recommended for estimating limits of agreement, 300 patients were enrolled.

Measures to limit bias

Several steps were taken to reduce avoidable sources of discrepancy. Patients were enrolled consecutively rather than selectively, which limits spectrum bias. A single investigator performed all arterial sampling, which reduces operator variation in technique and syringe handling. Both samples were obtained before any treatment that could alter the analytes, and both were analysed in the same emergency laboratory with transport times under one minute, which minimises in-vitro glycolysis and potassium leakage after collection. Treating clinicians were not influenced by the study, and the investigator did not alter clinical management on the basis of either result. Haemoglobin values were harmonised to g/dL for analysis.

Residual sources of bias that could not be controlled by the protocol, including heparin dilution, haemolysis during cannula sampling and the difference between whole blood and serum matrices, are considered in the Discussion.

Statistical analysis

Data were entered into Microsoft Excel 2010 and analysed with SPSS version 11.5 (SPSS Inc., Chicago, IL, USA). Categorical variables are reported as frequencies and percentages, and continuous variables as mean and standard deviation (SD). Paired values from the two methods were compared with the paired Student t-test, and two-sided p-values below 0.05 were considered statistically significant.

Relative bias was calculated as the mean difference divided by the laboratory mean and multiplied by 100. Because a significance test shows only whether two means differ, not whether two methods agree,27 analytical acceptability was judged by comparing the absolute mean bias with the CLIA acceptance limits in force during the study (sodium ±4 mmol/L, potassium ±0.5 mmol/L, glucose ±6 mg/dL or ±10% whichever is greater, haemoglobin ±7%) and, for current relevance, with the revised limits implemented from January 2025 (potassium ±0.3 mmol/L, glucose ±6 mg/dL or ±8%, haemoglobin ±4%, sodium unchanged).32,33 No data were missing for the four analytes .

Data management

Data were recorded at the bedside on a pre-tested structured proforma, transcribed from the analyser printouts rather than from the hospital information system, and entered into a password-protected spreadsheet identified only by study number. Entries were checked against the source printouts before analysis. Implausible values were to be rechecked against the original printout rather than excluded.

Ethical considerations

The study was approved by the Institutional Review Committee of, Raichur institute of medical sciences (Rims), Raichur, Karnataka, India. The study was explained to each patient or, when the patient lacked capacity, to an accompanying relative, and informed consent was obtained before sampling. Participation did not alter care: all clinical decisions were made by the treating emergency team, and patients could decline or withdraw at any time. Data were anonymised and used for research only. The study conformed to the Declaration of Helsinki.

RESULTS

Participants

A total of 300 patients were enrolled and all 300 contributed paired samples to the analysis Their characteristics are summarised in Table 1. Females outnumbered males (172, 57.3% versus 128, 42.7%) .Almost the entire cohort was triaged as ATS category 2 (297, 99.0%); only three patients (1.0%) were ATS category 1. Patients came from 21 districts or localities; 133 (44.3%) resided in Raichur district, where the hospital is located, and 36 (12.0%) in adjoining yadgir district, while two patients came from ballery district.

The leading working diagnosis was acute exacerbation of chronic obstructive pulmonary disease (66, 22.0%), followed by chronic kidney disease with pulmonary oedema (40, 13.3%), sepsis (28, 9.3%), community-acquired pneumonia (25, 8.3%) and poisoning (24, 8.0%). Other diagnoses included cerebrovascular accident with aspiration pneumonia (12, 4.0%), subacute intestinal obstruction with shock (11, 3.7%) and diabetic ketoacidosis (8, 2.7%); 30 patients had diagnoses represented by a single case each .

Sodium

Mean sodium measured by the blood gas analyser was 131.08 ± 7.234 mmol/L, compared with 135.90 ± 7.431 mmol/L on the laboratory autoanalyser (Table 2). The blood gas analyser therefore read lower by a mean of 4.82 mmol/L ($p < 0.001$), a relative bias of −3.55% (−4.82 ÷ 135.90 × 100). This bias exceeded the CLIA acceptance limit of ±4 mmol/L, which is unchanged in the 2025 revision (Table 3).

Potassium

Mean potassium was 4.0 ± 0.994 mmol/L by blood gas analysis and 4.6 ± 1.044 mmol/L in the laboratory, a mean difference of 0.60 mmol/L in the same direction ($p < 0.001$). The relative bias of −13.04% (−0.60 ÷ 4.6 × 100) was the largest of the four analytes. The absolute bias exceeded both the original CLIA limit of ±0.5 mmol/L and the revised limit of ±0.3 mmol/L.

Glucose

Glucose showed the opposite direction of bias. The blood gas analyser reported a mean of 152.77 ± 84.596 mg/dL against a laboratory mean of 140.86 ± 81.032 mg/dL, a difference of 11.91 mg/dL ($p < 0.001$) and a relative bias of 8.46% (11.91 ÷ 140.86 × 100). Under the original CLIA criterion, the allowable error at the laboratory mean was 14.09 mg/dL (10% ×

140.86), so the mean bias was acceptable. Under the 2025 criterion of ±8%, the allowable error at the same concentration is 11.27 mg/dL (8% × 140.86), which the observed bias marginally exceeded.

Haemoglobin

Mean haemoglobin was 11.29 ± 3.515 g/dL on the blood gas analyser and 11.41 ± 3.402 g/dL in the laboratory, a mean difference of 0.12 g/dL (p = 0.031). Although statistically significant, the relative bias of −1.05% (−0.12 ÷ 11.41 × 100) was well within both the original CLIA limit of 0.80 g/dL (7% × 11.41) and the revised limit of 0.46 g/dL (4% × 11.41).

Agreement for individual patients

Table 4 is reserved for limits of agreement. Without these estimates, the results above describe systematic bias between the methods but not the scatter of differences around it, which determines whether a single patient's result can be relied upon.

Table 1. Characteristics of high-acuity emergency patients undergoing paired sampling (n = 300)

Characteristic	n (%)
Sex	
Female	172 (57.3)
Male	128 (42.7)
Age, years, mean (SD)	[INSERT]
Australasian Triage Scale category	
Category 1	3 (1.0)
Category 2	297 (99.0)
District of residence	
yadir	133 (44.3)
ballary	36 (12.0)
Other districts of karnataka	131 (43.7)
Working diagnosis	
Acute exacerbation of COPD	66 (22.0)
CKD with pulmonary oedema	40 (13.3)
Sepsis	28 (9.3)
Community-acquired pneumonia	25 (8.3)
Poisoning	24 (8.0)
CVA with aspiration pneumonia	12 (4.0)
Subacute intestinal obstruction with shock	11 (3.7)
Diabetic ketoacidosis	8 (2.7)
Chronic liver disease with upper GI bleed	8 (2.7)
Chronic heart failure	7 (2.3)
Acute coronary syndrome	7 (2.3)
Other diagnoses (≤6 patients each)	60 (20.0)*

COPD, chronic obstructive pulmonary disease; CKD, chronic kidney disease; CVA, cerebrovascular accident; GI, gastrointestinal; SD, standard deviation. *Includes hypovolaemic shock (6), congestive cardiac failure with atrial fibrillation (6), seizure disorder (5), acute pancreatitis (5), hypertensive urgency (4), burns (3), acute asthma (3) and 30 single-case diagnoses; listed diagnoses in the source thesis total 298 [INSERT: reconcile two patients].

Table 2. Paired comparison of analytes measured by the arterial blood gas analyser and the venous central laboratory autoanalyser (n = 300)

Analyte (unit)	Blood gas analyser, mean (SD)	Laboratory autoanalyser, mean (SD)	Mean difference (BGA − lab)	p-value†
Sodium (mmol/L)	131.08 (7.234)	135.90 (7.431)	−4.82	<0.001
Potassium (mmol/L)	4.0 (0.994)	4.6 (1.044)	−0.60	<0.001
Glucose (mg/dL)	152.77 (84.596)	140.86 (81.032)	+11.91	<0.001
Haemoglobin (g/dL)	11.29 (3.515)	11.41 (3.402)	−0.12	0.031

BGA, blood gas analyser (ABL800, arterial whole blood); lab, central laboratory autoanalyser (Cormay Accent 200, venous serum); SD, standard deviation. †Paired Student t-test.

Table 3. Mean bias of the blood gas analyser relative to CLIA criteria for acceptable analytical performance

Analyte	Mean bias	Relative bias (%)	CLIA limit, 1992 (at lab mean)	Within?	CLIA limit, 2025 (at lab mean)	Within?
Sodium	−4.82 mmol/L	−3.55	±4 mmol/L	No	±4 mmol/L	No
Potassium	−0.60 mmol/L	−13.04	±0.5 mmol/L	No	±0.3 mmol/L	No
Glucose	+11.91 mg/dL	+8.46	±10% (14.09 mg/dL)	Yes	±8% (11.27 mg/dL)	No
Haemoglobin	−0.12 g/dL	−1.05	±7% (0.80 g/dL)	Yes	±4% (0.46 g/dL)	Yes

Bias is blood gas analyser minus laboratory value. Relative bias = mean difference ÷ laboratory mean × 100. For glucose, CLIA allows ±6 mg/dL or the percentage limit, whichever is greater; the percentage limit applies at the laboratory mean of 140.86 mg/dL. CLIA limits define allowable total error for individual results, so a mean bias within the limit is necessary but not sufficient for interchangeability. CLIA, US Clinical Laboratory Improvement Amendments.

Table 4. Agreement between methods for individual patients (Bland–Altman analysis) [INSERT: complete from raw data]

Analyte	Mean bias (95% CI)	SD of differences	95% limits of agreement	Pairs within CLIA 2025 limit, n (%)
Sodium (mmol/L)	−4.82 [INSERT CI]	[INSERT]	[INSERT]	[INSERT]
Potassium (mmol/L)	−0.60 [INSERT CI]	[INSERT]	[INSERT]	[INSERT]
Glucose (mg/dL)	+11.91 [INSERT CI]	[INSERT]	[INSERT]	[INSERT]
Haemoglobin (g/dL)	−0.12 [INSERT CI]	[INSERT]	[INSERT]	[INSERT]

Limits of agreement = mean bias ± 1.96 × SD of paired differences. Report also the slope of differences regressed on means (proportional bias) and, if differences are non-normal or heteroscedastic, limits based on log-transformed or percentage differences. CI, confidence interval; SD, standard deviation.

DISCUSSION

In 300 high-acuity emergency patients, blood gas analyser results differed systematically from venous central laboratory results for all four analytes. Sodium and potassium were lower on the blood gas analyser by margins that exceeded CLIA limits for acceptable analytical performance, whereas the mean biases for glucose and haemoglobin were small enough to meet the limits in force during the study. These findings argue against interchangeable use of blood gas electrolytes in our department without a correction strategy and laboratory confirmation, while leaving open the more demanding question of how well the methods agree for individual patients.

Sodium

The direction and size of the sodium bias closely reproduce the findings of Jain and colleagues in an Indian intensive care unit, where blood gas sodium averaged 131.28 mmol/L against 136.45 mmol/L in the laboratory, a difference almost identical to ours.18 Smaller differences in the same direction were reported in Chinese and Turkish cohorts and in a recent Indian emergency study,19,20,24 while a large European emergency dataset found laboratory sodium lower than blood gas sodium but with poor agreement overall and the greatest deviations in dysnatraemic patients.23 The consistency of direction across studies using direct and indirect electrodes points to the measurement principle rather than chance. Indirect ion-selective electrodes assume a plasma water fraction of about 93%; when protein concentrations fall, the true water fraction rises and the indirect method overestimates sodium. Story and colleagues showed that the laboratory–blood gas difference increased as albumin fell,14 and neonatal and cirrhotic cohorts confirm that hypoproteinaemia drives clinically important misclassification.15,16 Our cohort was dominated by sepsis, chronic kidney disease with pulmonary oedema, chronic liver disease and other critical illnesses in which hypoalbuminaemia is expected, which may explain why our sodium bias is larger than that reported in less acutely ill emergency populations.21 Albumin was not measured, so this mechanism remains plausible rather than proven in our data. A second, additive explanation is pre-analytical: excess liquid heparin dilutes the sample and binds cations, producing negative bias in blood gas sodium and potassium.26 We were unable to record the heparin type, and syringe preparation deserves scrutiny in any local quality-improvement effort.

A sodium difference of almost 5 mmol/L is not trivial. It is enough to classify a patient as hyponatraemic on one analyser and normonatraemic on the other, to alter the calculated anion gap and strong ion difference,17 and to change the apparent

rate of correction during treatment if the two methods are used alternately. Clinicians should not compare serial sodium values obtained from different analysers.

Potassium

The potassium bias of 0.60 mmol/L was proportionally the largest discrepancy and the most clinically consequential. Other studies have reported smaller biases in the same direction, of roughly 0.2 to 0.4 mmol/L,19,20,21,24 and Jain and colleagues found no significant difference.18 Our design probably inflated the gap. The laboratory sample was serum, and potassium is released from platelets and other cells during clotting, so serum potassium exceeds plasma or whole-blood potassium by an amount that rises with platelet count.34 In addition, the venous sample was aspirated through a newly placed intravenous cannula by syringe, a technique associated with in-vitro haemolysis; drawing blood through emergency department cannulae produces haemolysis in a substantial minority of samples and measurably raises potassium.35,36 Haemolysis indices were not recorded, so we cannot quantify this effect. Heparin dilution in the arterial syringe would push in the same direction.26 The true arteriovenous difference in potassium is negligible, so almost all of the observed gap is likely analytical or pre-analytical rather than physiological.

Which value is closer to the truth is therefore uncertain; the blood gas value may be the more accurate reflection of circulating potassium in some patients. For patient safety, however, the practical message is the same. A difference of 0.6 mmol/L could lead to unnecessary potassium replacement for apparent hypokalaemia on the blood gas analyser, or to delayed recognition of hyperkalaemia, which matters in a cohort in which more than one in eight patients had chronic kidney disease. Under the revised CLIA limit of ±0.3 mmol/L, the bias is twice the allowable error.

Glucose

Blood gas glucose was higher than laboratory glucose by about 12 mg/dL. Part of this is physiological: arterial blood glucose exceeds venous glucose because peripheral tissues extract glucose, more so after feeding or insulin exposure. Part is pre-analytical: the laboratory sample was collected into a plain tube without a glycolysis inhibitor, and glucose continues to be consumed by cells until serum is separated. The mean bias satisfied the CLIA criterion in force during the study but would marginally exceed the revised 8% criterion. This agrees with a systematic review showing that blood gas analysers are among the more accurate point-of-care methods for glucose, with the important caveat that accuracy deteriorates near the hypoglycaemic range and in haemodynamically unstable patients.25 Because glucose varied widely in our cohort, a mean bias of 8.46% may conceal larger absolute errors at the extremes. Blood gas glucose seems adequate to identify hypoglycaemia or marked hyperglycaemia in the first minutes of care, but insulin infusions should be titrated on a single consistent method.

Haemoglobin

The haemoglobin difference was statistically significant but clinically negligible at 0.12 g/dL, and we interpret it as such; statistical significance here reflects the large sample rather than a meaningful discrepancy. Zhang and colleagues similarly found no clinically relevant haemoglobin bias between an emergency blood gas analyser and the laboratory.19 Other studies are less reassuring: a recent Indian emergency study found unacceptable total error for blood gas haemoglobin,24 and a meta-analysis of intraoperative point-of-care devices showed a small pooled bias for blood gas analysers but wide limits of agreement.37 These contrasting findings illustrate the central limitation of mean bias as a measure of agreement: two methods can agree perfectly on average and still disagree substantially for individual patients. Blood gas haemoglobin is useful to detect gross anaemia or acute blood loss, but transfusion decisions near a threshold should be confirmed in the laboratory until patient-level agreement has been demonstrated locally.

Direction of bias across analytes

The opposing directions of bias are internally coherent. The three analytes for which the blood gas analyser read lower (sodium, potassium and, marginally, haemoglobin) are those for which the laboratory method or sample matrix would be expected to read higher: indirect electrodes overestimate sodium in hypoproteinaemia, clotting releases potassium into serum, and liquid heparin dilutes the arterial sample. Glucose, by contrast, is consumed in the venous serum tube and is physiologically higher in arterial than in venous blood, so the laboratory value would be expected to read lower. That each analyte moved in the direction predicted by known physiological and pre-analytical mechanisms suggests that the discrepancies are systematic and explicable rather than random analyser error, which strengthens the case for targeted correction of the underlying processes rather than simple numerical adjustment.

Interpretation of bias, correction factors and agreement

Two features of our analysis shape how these results should be read. First, CLIA limits define allowable total error for an individual result, not for a group mean. Comparing the mean bias with these limits is therefore a lenient test. When the mean bias alone exceeds the limit, as it did for sodium and potassium, non-equivalence is established firmly; when the mean bias falls within the limit, as for glucose and haemoglobin, acceptability is necessary but not sufficient. Second, the

paired t-test addresses whether the means differ, which in a sample of 300 is almost inevitable, rather than whether the methods agree.27 The Bland–Altman approach, which estimates the range within which 95% of individual differences fall, is the appropriate analysis and should be applied to the raw data. Studies that have done so report limits of agreement for sodium spanning more than 20 mmol/L in some populations.20,22

Implications for the case mix

The composition of our cohort sharpens the clinical relevance of these biases. Acute exacerbation of chronic obstructive pulmonary disease was the commonest diagnosis, and in these patients the arterial sample is clinically indicated for carbon dioxide tension; the electrolyte panel arrives without additional sampling, and its systematic bias must be known if it is used to guide replacement during nebulised beta-agonist therapy, which itself lowers serum potassium. Chronic kidney disease with pulmonary oedema was the second commonest diagnosis; in these patients a negative potassium bias of 0.6 mmol/L could mask clinically important hyperkalaemia on the blood gas report. In diabetic ketoacidosis, the potassium value determines whether insulin can be started or must be delayed while potassium is replaced, and a falsely low reading could delay definitive treatment, while venous blood gas analysis has otherwise been shown to reflect the severity of acidosis adequately.6 Sepsis and poisoning, the third and fifth commonest diagnoses, frequently involve hypoalbuminaemia, mixed acid–base disorders and evolving electrolyte shifts, the very circumstances in which direct and indirect sodium methods diverge most.14,17 Clinicians caring for these groups should be particularly cautious about treating on blood gas electrolytes alone.

Triage acuity and haemodynamic status

The study set out to examine high-acuity patients, but ATS category 1 patients were scarcely represented, almost certainly because resuscitation took precedence over research sampling. Our findings therefore describe the ATS category 2 population, which includes patients with severe respiratory distress, circulatory compromise and altered consciousness but generally excludes those in cardiac arrest. Arteriovenous differences widen in hypotensive patients,8 and the validity of venous surrogates in shocked patients remains insufficiently studied,1 so our conclusions should not be extended to patients in profound shock. Triage itself is imperfect: the ATS has only moderate inter-rater reliability,29 and some misclassification between categories 2 and 3 is likely. Because analytical bias is a property of the measurement method rather than of the triage category, however, this is unlikely to have changed the direction or approximate magnitude of the observed differences.

Quality assurance and the resource-limited setting

The central laboratory in our institution participates in external quality assessment, whereas the blood gas analyser does not, and this asymmetry is common in resource-limited settings where point-of-care devices are introduced to bypass laboratory delays. The benefit of point-of-care testing depends on rapid results being both available and trustworthy, and its effect on length of stay is realised only when the result is integrated into departmental decision-making.10,11,12 A fast but systematically biased result can produce harm at the same speed as it produces benefit. Enrolling the blood gas analyser in an external quality assessment scheme, standardising arterial syringes with electrolyte-balanced dry heparin,26 and collecting laboratory samples by separate venepuncture rather than through a new cannula35 are low-cost measures that would address the most likely sources of discrepancy identified here. A joint protocol between the emergency department and the laboratory, specifying which point-of-care results may be acted upon and which require confirmation, would make these findings operational.

It is tempting to apply the observed mean differences as fixed correction factors, adding 4.82 mmol/L to blood gas sodium and 0.60 mmol/L to blood gas potassium. We do not recommend this without further analysis. A constant correction is valid only if the bias is constant across the measuring range, and proportional bias has been demonstrated for potassium in large emergency datasets,23 while sodium bias varies with albumin.14 A correction derived from the cohort mean could overcorrect at one end of the range and undercorrect at the other, which matters most at the extremes where treatment decisions are made.

Strengths

This is, to our knowledge, the first study from raichur to compare blood gas and laboratory analytes in emergency patients. The sample of 300 consecutive patients is larger than most single-centre comparisons, and the case mix of respiratory, renal, septic and toxicological emergencies reflects the population in whom these tests are most often used. Samples were drawn before any treatment, the two analysers were in the same emergency laboratory, and transport time was under one minute, which limits time-dependent changes in glucose and potassium.

Limitations

Several limitations must be acknowledged. Sample type and analyser are inseparable in our design: arterial whole blood on the blood gas analyser was compared with venous serum on the laboratory analyser, so physiological, matrix and

analytical differences contribute to every estimate. This mirrors routine practice but limits mechanistic inference. Only summary statistics were analysed; limits of agreement, proportional bias and the proportion of individual pairs within acceptable limits were not estimated, and age was not reported. The blood gas analyser was not enrolled in external quality assessment. Albumin, total protein, platelet count, haemolysis index and heparin type were not recorded, so the mechanisms proposed above remain inferential. Potassium values were reported to one decimal place, which limits the precision of the potassium bias. Almost all patients were ATS category 2, so the findings may not apply to patients in cardiac arrest or profound shock. Venous samples were collected by the duty nurses through newly placed cannulae rather than by a single operator using a standardised venepuncture, which may have increased variability in haemolysis. Finally, this was a single-centre study using two specific instruments, and the results should not be generalised to other analysers.

Clinical implications

In our emergency department, blood gas sodium and potassium can be used to recognise severe derangements and to initiate urgent treatment, but they should be interpreted with knowledge of the systematic negative bias and confirmed by the laboratory before decisions that depend on modest differences. Serial monitoring should use a single method. Blood gas glucose and haemoglobin appear acceptable for initial assessment. Every hospital that relies on point-of-care values should conduct its own comparison, ideally before a new analyser is installed, and should enrol the analyser in external quality assessment.

Future research

Re-analysis of the raw data with Bland–Altman methods is the immediate priority. Future studies should compare arterial and venous samples on the same analyser and the same venous sample on both analysers, to separate physiological from analytical sources of difference; record albumin, platelet count, haemolysis index and heparin type; and examine whether agreement differs at clinically critical concentrations and in shocked patients. Studies that measure the effect of acting on blood gas values on time to treatment and patient outcomes would establish whether the speed of point-of-care testing translates into clinical benefit.

CONCLUSION

In high-acuity emergency patients at a tertiary hospital in raichur, the arterial blood gas analyser reported sodium about 5 mmol/L lower and potassium about 0.6 mmol/L lower than the venous central laboratory, biases that exceed accepted limits of analytical performance. These electrolyte values cannot be used interchangeably with laboratory values without local correction and confirmation. Mean differences for glucose and haemoglobin were within accepted limits, supporting their use for rapid initial assessment, but agreement for individual patients must be confirmed with Bland–Altman analysis. Each emergency department should validate its own analysers before relying on point-of-care results for treatment decisions.

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