Title
Prospective correlation of arterial vs venous blood gas measurements in trauma patients ☆,☆☆

Journal
The American journal of emergency medicine, 30(8)

ISSN
1532-8171

Authors
Rudkin, Scott E.
Kahn, Christopher A.
Oman, Jennifer A.
et al.

Publication Date
2011-12-12

Peer reviewed

Published in final edited form as:
Am J Emerg Med. 2012 October ; 30(8): 1371–1377. doi:10.1016/j.ajem.2011.09.027.

Prospective correlation of arterial vs venous blood gas measurements in trauma patients☆,☆☆

Scott E. Rudkin, MD, MBA[a,*], **Christopher A. Kahn, MD, MPH**[a], **Jennifer A. Oman, MD, MBA**[a], **Matthew O. Dolich, MD**[b], **Shahram Lotfipour, MD, MPH**[a], **Stephanie Lush, RN**[b], **Marla Gain, RN**[a], **Charmaine Firme**[a], **Craig L. Anderson, MPH, PhD**[a], and **Mark I. Langdorf, MD, MHPE**[a]

[a]Department of Emergency Medicine, University of California, Irvine, CA 92868, USA

[b]Division of Trauma, Department of Surgery, University of California, Irvine, CA 92868, USA

Abstract

Objective—The objective of this study is to assess if venous blood gas (VBG) results (pH and base excess [BE]) are numerically similar to arterial blood gas (ABG) in acutely ill trauma patients.

Methods—We prospectively correlated paired ABG and VBG results (pH and BE) in adult trauma patients when ABG was clinically indicated. A priori consensus threshold of clinical equivalence was set at ± less than 0.05 pH units and ± less than 2 BE units. We hypothesized that ABG results could be predicted by VBG results using a regression equation, derived from 173 patients, and validated on 173 separate patients.

Results—We analyzed 346 patients and found mean arterial pH of 7.39 and mean venous pH of 7.35 in the derivation set. Seventy-two percent of the paired sample pH values fell within the predefined consensus equivalence threshold of ± less than 0.05 pH units, whereas the 95% limits of agreement (LOAs) were twice as wide, at −0.10 to 0.11 pH units. Mean arterial BE was −2.2 and venous BE was −1.9. Eighty percent of the paired BE values fell within the predefined ± less than 2 BE units, whereas the 95% LOA were again more than twice as wide, at −4.4 to 3.9 BE units. Correlations between ABG and VBG were strong, at $r^2 = 0.70$ for pH and 0.75 for BE.

Conclusion—Although VBG results do correlate well with ABG results, only 72% to 80% of paired samples are clinically equivalent, and the 95% LOAs are unacceptably wide. Therefore, ABG samples should be obtained in acutely ill trauma patients if accurate acid-base status is required.

1. Introduction

An arterial blood gas (ABG) provides important information for critically ill patients. The base excess (BE) is a useful predictor of serious injury in trauma patients in general (BE less than −4) [1] and elderly patients in particular (BE less than −2), and aggressive critical care management is indicated [2,3]. Tissue hypoperfusion causes release of lactic acid leading to metabolic acidosis. The base deficit is calculated from the pH of the ABG and provides an

☆Presented at American College of Emergency Physicians Research Forum, 2006, New Orleans, LA.

☆☆Supported by a grant from the Emergency Medicine Foundation, Emergency Medicine Basic Research Skills, and the General Clinical Research Center, University of California, Irvine, supported by the National Institute of Health research grant M01 RR00827 from the National Center for Research Resources.

*Corresponding author. srudkin@uci.edu (S.E. Rudkin).

objective measure of the degree of hypoperfusion. As a patient is successfully resuscitated, base deficit improves, becoming less negative. The Advanced Trauma Life Support Course, offered by the American College of Surgeons (ACS), advises that "adequate resuscitation is best assessed by improvement in physiologic parameters, such as …ABG levels…rather than the qualitative assessment done during the primary survey. Actual values…should be obtained as soon as is practical after…the primary survey and periodic reevaluation is prudent"[4].

However, obtaining an ABG can cause patient morbidity by arterial injury and is more painful than venous sampling [5]. If the 2 were clinically interchangeable, the venous blood gas (VBG) could be drawn simultaneously with placement of the resuscitative venous catheter. Prior studies have shown that a VBG can substitute for an ABG in diabetic ketoacidosis or acutely ill medical patients [6-8]. Correlations were high ($r = 0.97$), with mean VBG pH lower by 0.03 (range, 0-0.11) in one study and 0.056 (range, 0.07-0.35) in the other [6,7]. Although prior work has shown that VBG testing is not an accurate substitute for ABG results in mechanically ventilated trauma patients, substitution has not been assessed in the acute phase of trauma resuscitation [9].

We sought to determine (1) whether VBG measurements of blood pH and calculated BE are numerically and similar to these values in an ABG across a broad spectrum of acutely injured patients and, if not, (2) whether a linear regression equation would accurately predict ABG values from a VBG result.

2. Materials and Methods

We prospectively correlated paired ABG and VBG results (pH and BE) from a convenience sample of adult (age >18 years) trauma-designated patients at an ACS-verified level 1 trauma center, from January through September 2006. Patients were included according to the following institutional criteria, which required an ABG be drawn for patients meeting any of the following criteria:

- Systolic blood pressure less than 90 mm Hg in the emergency department (ED)
- Requiring transfusion of blood products
- Requiring endotracheal intubation
- Positive Focused Assessment of Sonography in Trauma
- Ongoing hemorrhage (declining bedside hemoglobin level)
- Pelvic fractures with disrupted ring
- Hypoxia (SpO_2 <93% on room air).
- Judgment of trauma captain

All patients older than 60 years with any of the following:

- Motor vehicle crash (MVC) greater than 30 mph
- MVC with passenger space intrusion greater than 12 in
- Passenger ejection from the vehicle
- MVC rollover
- MVC with fatality in same vehicle
- Automobile vs pedestrian injury, at any speed

Fall from greater than standing height

Systolic blood pressure less than 100 mm Hg on ED arrival

Pulse less than 60 or greater than 100 beats per minute

Once the trauma captain determined an ABG was indicated by these criteria, a VBG was drawn as soon as possible. We excluded patients if the 2 samples were drawn more than 1 hour apart.

2.1. Study setting

The level I study trauma center cared for 2141 patients during the study year. These were 75% male, with 80% categorized as moderate trauma activations vs 20% critical. Eighty-five percent were victims of blunt trauma; 13%, penetrating; and 2%, burns. *Penetrating trauma* was defined as mechanisms of gunshot wound, stabbing, or impalement. Blunt trauma included motor vehicle collision, motorcyclist, pedestrian, bicyclist, fall, or other assault. Eighteen percent of trauma victims had injury severity scores greater than 15, with 11% going directly to the operating room from the ED. Eighty-two percent of trauma patients were admitted to the hospital (23% to the intensive care unit), whereas the remainder were observed and discharged from the ED after 6 to 12 hours.

2.2. Study procedures

Patients were included if an ABG was clinically indicated. We used femoral or radial arteries, according to clinical availability, from which we drew blood samples with either a 23-gauge, 1-in (radial) or 22-gauge, 1.5-in needle (femoral). Venous samples were drawn directly from antecubital veins, before any intravenous infusion, with an 18- to 20-gauge, 1.5-in intravenous catheter. Arterial samples were drawn immediately into a standard blood gas syringe. Both samples were run on the same analyzer within 10 minutes of sample acquisition (Bayer Model 865 automatic pH/blood gas system analyzer; Tarrytown, NY). Samples were sent to laboratory via a 4-in tube-transport system. Base excess was calculated from pH and P_{co_2} results using the Henderson-Hasselbach equation. A priori, a focus group of 15 attending physicians (trauma surgery, emergency medicine [EM], critical care/pulmonology, and anesthesiology), blinded to the study hypothesis, set a consensus single threshold of less than 0.05 pHunits and a variance in BEless than 2,regardless of pH and BE values, as the greatest tolerable difference between ABG and VBG to be considered clinically equivalent.

Research assistants directly observing the resuscitation collected times of ABG and VBG draw, physiologic data (initial blood pressure, pulse oximetry, and heart rate), and indicators of patient severity (Glasgow Coma Scale score, trauma and injury severity scores) as well as intubation status, need for blood products, and Focused Assessment of Sonography in Trauma results. We defined elderly patients as those older than 65 years. A priori, we planned to determine whether our prediction equation applied more closely for patients with markers of significant pulmonary injury: need for endotracheal intubation or pneumothorax or hemothorax. The study was approved by the local institutional review board with waiver of informed consent for resuscitation research.

2.3. Statistical analysis

To determine that we would find that at least 95% of the ABG-VBG pairs were clinically equivalent if 99% were truly so, we calculated a sample size of 173 for each of the planned derivation and validation sets. This sample would also provide 90% power to detect 90% equivalence in the more acutely ill subgroup (BE −4 or less or −2 or less in the elderly), assuming that 30% of patients would fall within this group [1]. To allow for 10% loss of

patients due to missing data, we planned to enroll 384 patients, split equally into derivation and validation groups.

Previous studies have shown that ABG and VBG values are not numerically equivalent [6-8]. We hypothesized that ABG and VBG would be found to be linearly related. Thus, we used linear regression to predict ABG pH and BE from the corresponding VBG results. This approach requires a derivation set to estimate the coefficients of the regression equation and a validation set to determine the accuracy of the prediction.

Data were entered into a spreadsheet (Excel; Microsoft Corporation, Redmond, WA) and analyzed with Stata (version 10.1; Stata Corporation, College Station, TX). Because, by chance, the first half of the data included more subjects with BE −4 or less or −2 or less in the elderly, we randomly divided the data into derivation and validation sets by sorting on a random number generated by Stata. We report 95% confidence intervals (95% CI) for proportions and 95% limits of agreement (LOAs) to describe the difference between predicted and observed values [7]. The linear relationship of the ABG values to the VBG values of the derivation data set was summarized with the square of the Pearson product-moment correlation coefficient (r^2).

We then graphed the differences between observed and predicted arterial pH and BE values, vs the observed venous values (Bland-Altman), and plotted against lines reflecting the consensus-derived limits of clinical equivalence. Goodness of fit for patients with markers of significant pulmonary injury and for 5 ABG-VBG interval subgroups was assessed by applying Levene's robust test of equality of variances to the differences between predicted and observed values [9-11].

3. Results

We collected samples on 385 patients, excluded 29 (7.5%) with incomplete data (ABG, 2; VBG, 27), 10 (2.5%) patients with greater than 1 hour between samples, and one where times were not recorded, leaving 346 (89.9% of enrolled patients) for analysis. These represented 25.6% of trauma activation patients during the study period.

Injury severity scores were similar between those subjects who had complete data and only an ABG result; average injury severity score was 17.59. Average patient age was 42 years (range, 18-100 years). The age distribution was 23%, 18 to 24 years; 58%, 25 to 64 years; and 19%, older than 65 years. Twenty-two percent were female. Mechanism of injury was 83% blunt trauma, 16% penetrating, and 1% burns. There were no statistically significant differences in these parameters between the study patients and the overall population of trauma patients in our center. There were no statistically significant differences between the derivation and validation patients for the following factors: age, sex, mechanism or injury, time between ABG and VBG draws, or arterial or venous pH or BE. Fourteen percent were intubated or had a pneumothorax or hemothorax, markers of significant pulmonary injury. The median time between ABG and VBG draws was 10 minutes (interquartile range, 5-17 minutes). Thirty-four percent were within 5 minutes, 20% were 6 to 10 minutes apart, 27% were 11 to 20 minutes apart, and 19% were 21 to 60 minutes apart.

It is instructive to present results of the validation set in 3 ways:

1. Graphic relationship between predicted ABG and VBG for pH using Bland-Altman plots (Fig. 1) and BE (Fig. 2);
2. Proportion of predicted ABG results from VBG pH and BE results that fall within our predetermined “clinically equivalent” thresholds (Table 1); and

3. Predicted ABG results based on VBG results across a wide range of possible pH values (Table 2).

3.1. pH results

In the derivation set, mean arterial pH was 7.40, whereas mean venous pH was 7.35. The predictive equation obtained from linear regression on the derivation set was arterial pH of 1.09 + 0.86 (venous pH). The arterial and venous values were closely related ($r^2 = 0.70$). The accuracy of the predicted arterial pH in the validation data set is shown in Fig. 1 and summarized in Table 1. Only 72% of the subjects fit within the predefined acceptable range of ±0.05 pH units, and the 95% LOA was unacceptably wide at −0.11 to 0.10 pH units. In Table 1, similarity of observed ABG values to those predicted by linear regression from VBG values was shown. Both pH and BE values are stratified by the injury severity marker, low BE (even for pH value reporting). Rightmost column presented to consider possibility that consensus-derived clinically equivalent range is too narrow and that ±0.10 pH units or less and ±4 BE units or less may be more appropriate as clinically equivalent.

Across the clinically relevant range of pH values, the ability of the regression equation to predict ABG (arterial pH) results from VBG (venous pH) results varies, from close agreement with alkalemia to wide divergence with increasing acidemia, as shown in Table 2. Only with pH values greater than 7.4 does the regression equation predict ABG arterial pH within our a priori “clinically equivalent” range.

The distribution of the differences between the predicted and observed arterial pH values by time between the ABG and VBG blood draw is shown in Fig. 3. The fit of the predicted values did not differ between all these time categories ($P = .85$), nor was the difference significant for ABG-VBG intervals greater than or equal to 15 minutes vs shorter ($P = .96$). The predicted pH values fit the validation data set better for subjects with a normal or positive BE than for more severely injured patients with BE −4 or less for all patients and −2 or less in the elderly (Table 1; $P = .03$). Predictive accuracy did not vary by significant pulmonary injury (need for tube thoracostomy or endotracheal intubation), sex, or mechanism of trauma. For Figs. 3 and 4, the time intervals are not evenly distributed (eg, every 5 minutes) due to declining sample sizes with prolonged intervals.

3.2. Base excess results

In the derivation set, mean arterial BE was −2.2, whereas mean venous BE was −1.9. The predictive equation obtained by linear regression on the derivation set was arterial BE = −0.87 + 0.78 (venous BE). The arterial and venous values were closely related ($r^2 = 0.75$). The arterial and venous values of BE in the validation data set are shown in Fig. 2. The fit of the predicted values of arterial BE to the validation set is shown in Fig. 4 and summarized in Table 1. Only 80% of the subjects fit within the predefined “clinically equivalent” range of ±2 BE units or less; the 95% LOA was unacceptably wide at −3.9 to 4.4 BE units.

The distribution of the differences between the predicted and observed arterial BE by time between the ABG and VBG blood draw is shown in Fig. 4. The fit of the predicted values did not differ between all these time categories ($P = .20$), nor did it differ for intervals greater than or equal to 15 minutes vs shorter ($P = .22$). The predicted values fit the validation data set better for subjects with a normal or high BE than for more severely injured patients with a low BE (Table 1; $P < .0001$) but did not meet our definition of clinical equivalence in either group. The ability of the regression equation to predict arterial BE from venous BE was worse for patients with a marker of significant pulmonary injury than for those without such a marker (95% LOA, −5.7 to 5.0 and −3.6 to 4.3, respectively; $P = .006$). Predictive accuracy did not vary by sex or mechanism of trauma.

3.3. Limitations

We acknowledge several limitations. First, our "clinically equivalent" thresholds for pH and BE were arbitrarily determined by consensus of experts and are not based on any validated outcome measure. We performed a convenience sample omitting midnight to 8 AM when research assistants were not available. We excluded a minority of patients with samples drawn more than 1 hour apart. Most patients excluded for missing one blood gas value or the other were for missing VBG (n = 27). This imbalance reflects protocol violation in patients who, by clinical judgment, warranted an ABG, rather than exclusion of more severely injured patients. However, the decision to perform ABG and, hence, enroll a patient in the study was subject to unknown bias by a heterogeneous group of trauma surgeons and emergency physicians, as only 25.6% of all trauma patients were enrolled. We did not constrain the location of ABG or VBG blood draws. Although we did not find that increasing time between blood draws was statistically associated with differences between ABG and VBG values, we acknowledge the risk of a type II error due to small sample sizes. Because 21.8% of our patients in the validation set had ABG-VBG time intervals greater than 20 minutes, ongoing resuscitation could have accounted for our finding of nonequivalence. Finally, we did not follow up patients to clinical outcome nor determine if nonequivalence of these 2 acid-base parameters had a clinically important effect.

4. Discussion

Prior studies have concluded that a VBG can be used instead of an ABG with acceptable clinical accuracy for patients with diabetic ketoacidosis (DKA), general severe medical illness, and chronic obstructive pulmonary disease [6-8,12]. By contrast, Malinoski et al [8] showed that these were not interchangable for posttrauma ventilated patients in the intensive care unit [9].

However, this is the largest study comparing ABG to VBG results in acute-phase trauma patients. We found that VBG and ABG results correlate well in trauma patients ($r^2 = 0.70$ for pH and 0.75 for BE). However, their LOAs were broader than the a priori clinically equivalent ranges, and the predicted values fell outside these ranges too frequently (28% for pH and 20% for BE) to allow their clinical substitution.

Given that we did not study trends in acid base status or correlate them with clinical outcomes, we cannot say with certainty that clinical equivalence is absent. A prudent clinician, armed with this data, may choose to consider the 2 values as being more interchangeable than we did. Furthermore, it is not clear which values (ie, ABG vs. VBG) best reflect shock physiology, although one could posit that postcapillary acid-base status might better reflect tissue hypoperfusion.

Brandenburg and Dire [6] found a strong correlation ($r = 0.97$) between arterial and venous pH values with mean difference of 0.03 (range, 0-0.11) in 61 DKA patients, whereas Gennis et al [7] found a pH difference of 0.056 (-0.07 to 0.35) in a group of 184 acutely ill medical patients [8]. However, the study of Brandenberg et al [6] had a nearly 50% drop out rate when patients were not determined to be in DKA and did not report LOAs. Similary, Elborn et al [12] compared VBG with ABG samples in patients recovering from acute exacerbations of chronic obstructive pulmonary disease. He found a strong correlation between the tests ($r = 0.84$), but again, they did not report the proportion of patients whose values fell outside a clinically interchangeable range.

McGillivray et al [5] studied acutely ill pediatric patients to compare venous and capillary blood gas values. Intraclass correlation coefficients for capillary and venous pH, P_{CO_2}, and P_{O_2} were the main outcomes (0.92, 0.80, and 0.67, respectively). Although this study

demonstrated that a venous sample was very well correlated with an arterial specimen, the subject population was well perfused, and a true ABG was not performed. Again, mere correlation, in our view, is insufficient to gauge the appropriateness of clinical substituion.

The study results of Gennis et al [7] regarding pH were remarkably similar to ours [8]. They reported that 95% of their ABG pH values were predicted by their regression equation from VBG results within +0.11 pH units. Our study's 95% LOA ranges from Table 1 was −0.10 to 0.11. However, although Gennis et al [7] concluded, therefore, that the results were clinically interchangeable, our a priori definition of clinical equivalence was narrower, at ±0.05 or less. Using this definition, only 72% of values in our study were interchangeable, and therefore, VBG cannot be used to approximate ABG pH accurately enough to allow clinical substitution. Furthermore, the study population of Gennis et al was poorly described and, therefore, likely heterogeneous (including a minority of cardiac arrest patients), compared with our relatively homogenous trauma victim group.

A pilot study by Gindi et al [13] of 30 severely injured trauma patients compared near-simultaneous ABG and VBG and showed a Pearson correlation coefficient for BE of 0.922, indicating that 85% (R^2) of the variability in ABG base deficit can be accounted for by VBG results. However, this study did not report correlation data on pH nor LOAs. Although they concluded that VBG may be able to replace ABG, to substitute the venous test for the arterial, in our view, near equivalence would need to be shown for both components of acid-base status.

Although the explanation for these discrepant conclusions (apparent reliable substitution in medical patients vs insufficient clinical precision in trauma patients) is unclear, we postulate that the clinical and hemodynamic state in trauma would change more rapidly than in medical patients, due to ongoing hemorrhage. Hence, a longer interval between blood samples may reflect a greater change in values than for medical patients. However, we did not find that the discrepancy between ABG and VBG values varied significantly by time.

Furthermore, we had short transport times in an urban emergency medical services system and assessed patients essentially in the first hour after injury, when hemodynamic state would be most rapidly changing. This is contrary to the situation with acutely ill medical patients, whose illness more commonly develops over several hours to days. We believe that our study results will be applicable to most urban, ACS-verified centers that also have short emergency medical services transport times.

Our venous blood samples were drawn from a peripheral vein, whereas our arterial samples were drawn from a central source (femoral). This would have an unknown effect on the acid-base values and may account for our observed discrepancy. To address this, the study would have to be repeated with both samples drawn centrally, which is not feasible during the acute resuscitation.

Our study advanced the issue of ABG vs VBG results by studying BE. Schmelzer et al [14] previously reported on 100 critically ill trauma patients (24 died) and found that central venous base deficit was associated with survival past 24 hours, whereas arterial base deficit was not. Traditional teaching has advocated acid-base determination via ABG, but high Fi_{O_2} can artificially drive P_{O_2}, yet tissues may extract inadequate oxygen. With improvements in pulse oximetry making arterial P_{O_2} less relevant, we believe, in concert with Schmelzer et al, that venous BE may be a more clinically important determinant of global perfusion and, hence, patient outcome. Hence, there may be additional reasons, beyond pain and risk of arterial injury to deemphasize ABG in favor of VBG.

Our objective to determine if ABG and VBG are clinically interchangable in trauma patients begs the question of whether VBG alone or serial VBG values are sufficient to guide resuscitation. As we drew only 1 pair of samples and did not follow up patients to clinical outcome, our study design does not answer this question of clinical relevance.

Future studies would improve on the current offering by enrolling a consecutive sample, shortening the time between blood draws, and following up patients clinically for outcome. Arterial blood gas on all trauma patients would be required to fully answer the clinical question of equivalence but would be unethical in patients with minor injuries. Lastly, assessment of central rather than peripheral venous pH and BE would likely be more physiologically relevant in early resuscitation of trauma patients but is inherently more difficult to study.

5. Summary

Although VBG results do correlate well with ABG results, only 72% to 80% of paired samples are clinically equivalent, and the 95% LOAs are unacceptably wide. Therefore, ABG samples should be obtained for the management of acutely ill trauma patients if accurate acid-base status is required. Reliance on VBG samples to predict arterial pH and BE cannot be justified.

References

1. Rutherford EJ, Morris JA Jr, Reed GW, Hall KA. Base deficit stratifies mortality and determines therapy. J Trauma. 1992; 33:417–23. [PubMed: 1404512]
2. Davis J. Admission base deficit predicts transfusion requirements and risk of complications. Trauma. 1996; 41:769–74.
3. Davis JW, Kaups KL. Base deficit in the elderly: a marker of severe injury and death. J Trauma. 1998; 45(5):873–7. [PubMed: 9820695]
4. American College of Surgeons Committee on Trauma. 8th. Chicago, IL: American College of Surgeons; 2008. Advanced Trauma Life Support for Doctors; p. 10
5. McGillivray D, Ducharme FM, et al. Clinical decisionmaking based on venous versus capillary blood gas values in the well-perfused child. Ann Emerg Med. 1999; 34(1):58–63. [PubMed: 10381995]
6. Brandenburg MA, Dire DJ. Comparison of arterial and venous blood gas values in the initial emergency department evaluation of patients with diabetic ketoacidosis. Ann Emerg Med. 1998; 31(4):459–65. [PubMed: 9546014]
7. Gennis PR, Skovron ML, et al. The usefulness of peripheral venous blood in estimating acid-base status in acutely ill patients. Ann Emerg Med. 1985; 14(9):845–9. [PubMed: 3927796]
8. Malinoski DJ, Todd SR, et al. Correlation of central venous and arterial blood gas measurements in mechanically ventilated trauma patients. Arch Surg. 2005; 140:1122–5. [PubMed: 16342377]
9. Luce EA, Futrell JW, Wilgis EF, et al. Compression neuropathy following brachial arterial puncture in anticoagulated patients. J Trauma. 1976; 16:717. [PubMed: 1011308]
10. Bland JM, Altman DG. Statistical methods for assessing agreement between two methods of clinical measurement. Lancet. 1986; 1(8476):307–10. [PubMed: 2868172]
11. Levene, H. Robust tests for equality of variances. In: Olkin, I.; Ghurye, SG.; Hoeffding, W.; Madow, WG.; Mann, HB., editors. Contributions of probability and statistics: essays in honor of Harold Hotelling. Menlo Park, CA: Stanford University Press; 1960. p. 278-92.
12. Elborn JS, Finch MB, et al. Non-arterial assessment of blood gas status in patients with chronic pulmonary disease. Ulster Med J. 1991; 60(2):164–7. [PubMed: 1785150]
13. Gindi M, Sattler S, et al. Can venous blood gas samples replace arterial blood gas samples for measurement of base excess in severely injured trauma patients? N Y Med J. 2007; 2(2)

14. Schmelzer TM, Perron AD, Thomason MH, Sing RF. A comparison of central venous and arterial base deficit as a predictor of survival in acute trauma. Am J Emerg Med. 2008; 26:119–23. [PubMed: 18272088]

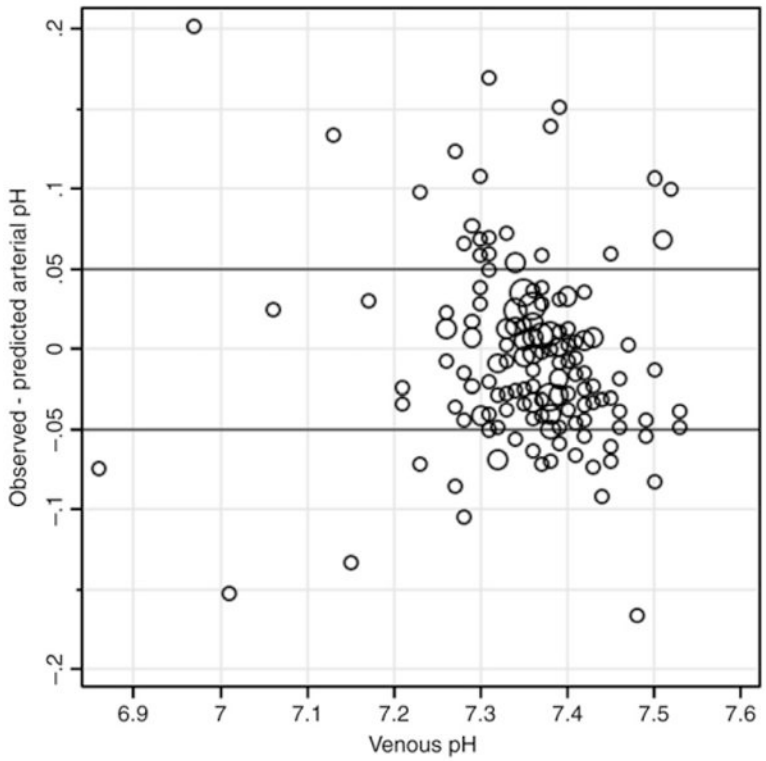

Fig. 1.

Bland-Altman plot of observed minus predicted arterial pH plotted against venous pH in validation data set (n = 173). Horizontal lines represent consensus-derived clinical equivalence of ±0.05 pH units or less. Circle size is proportional to the number of observations for each data point (range, 1-4).

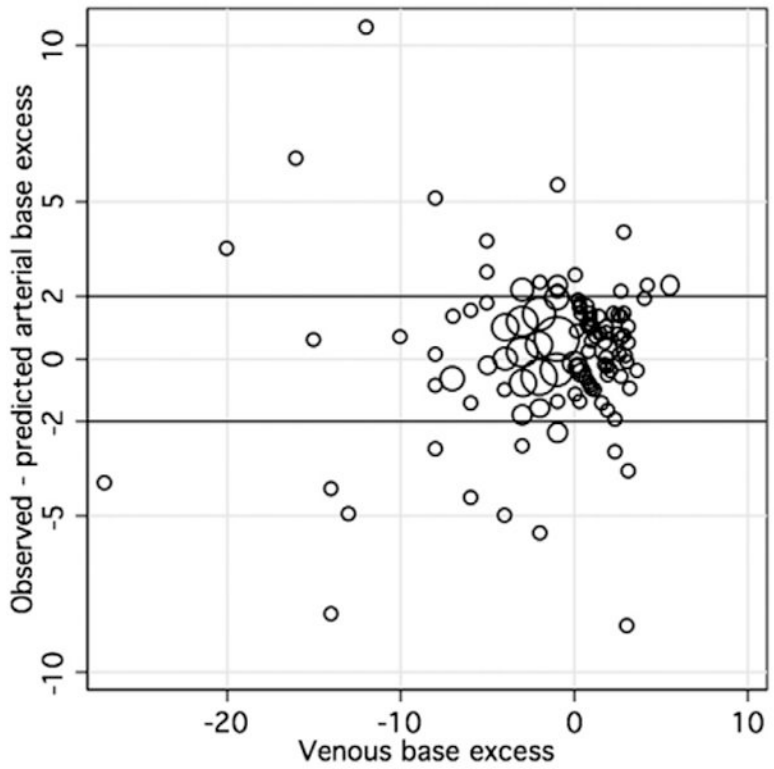

Fig. 2.

Bland-Altman plot of observed minus predicted arterial BE plotted against venous BE in validation data set (n = 173). Horizontal lines represent consensus-derived clinical equivalence of ±2 BE units or less. Circle size is proportional to the number of observations for each data point (range, 1-11).

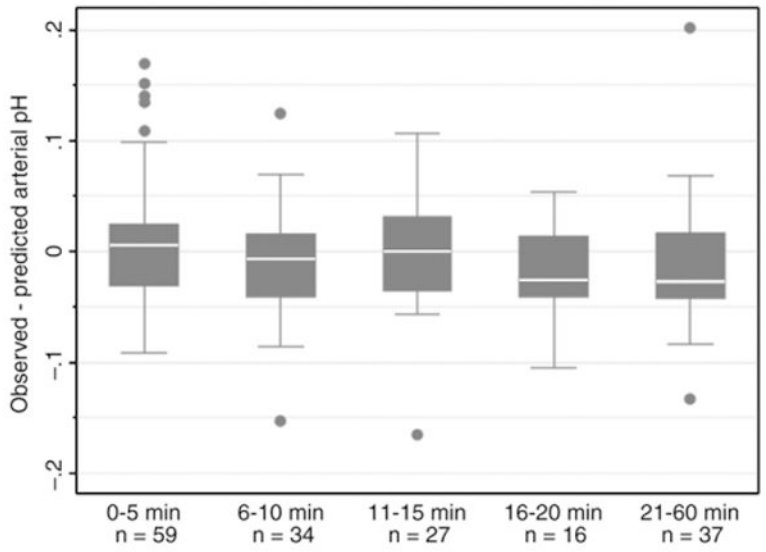

Fig. 3.

Box plots of the difference between observed and predicted arterial pH values by time between the ABG and VBG blood draws. The white line indicates the median; the shaded box, the interquartile range; the bars, any values within 1.5 times the interquartile range; and the dots, more extreme values.

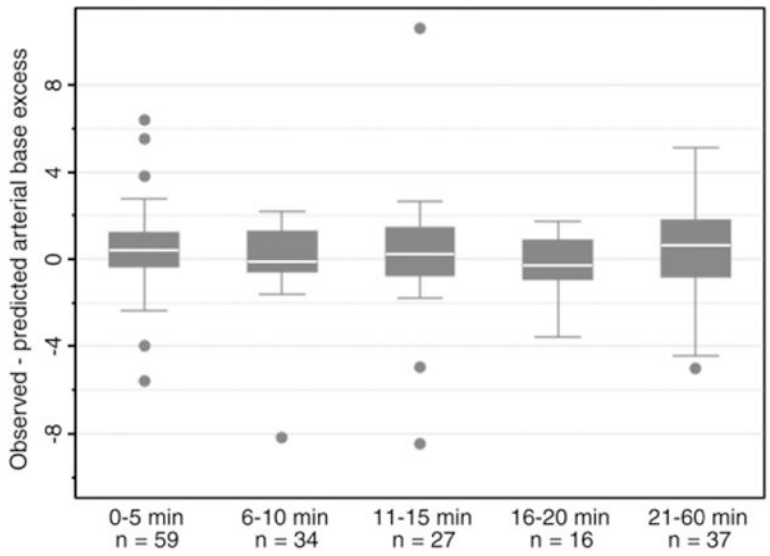

Fig. 4.

Box plots of the difference between observed and predicted arterial BE values by time between the ABG and VBG blood draws. The white line indicates the median; the shaded box, the interquartile range; the bars, any values within 1.5 times the interquartile range; and the dots, more extreme values.

Table 1

Similarity of observed ABG values to those predicted by linear regression from VBG values

				Percentage of paired samples within consensus clinically equivalent range	Percentage of paired samples within double the clinically equivalent range
		n	**95% LOA**	**± ≤0.05 (95% CI)**	**± ≤0.10 (95% CI)**
pH	Validation sample	173	−0.11 to 0.10	72% (65-79%)	93% (88-96%)
	Low BE	31	−0.17 to 0.12	58% (39-75%)	81% (63-93%)
	Normal or high BE	142	−0.09 to 0.09	75% (67-82%)	96% (91-98%)
		n	95% LOA	±≤2	±≤4
BE	Validation sample	173	−3.9 to 4.4	80% (74%-86%)	93% (88%-96%)
	Low BE	31	−7.6 to 4.6	61% (42%-78%)	74% (55%-88%)
	Normal or high BE	142	−2.5 to 3.8	85% (77%-90%)	97% (93%-99%)

Both pH and BE values are stratified by the injury severity marker, low BE (even for pH value reporting). Rightmost column presented to consider possibility that consensus-derived clinically equivalent range is too narrow and that ±0.10 pH units or less and ±4 BE units or less may be more appropriate as clinically equivalent. Low BE was defined as less than −4 for all patients and −2 in the elderly.

Table 2

Difference between observed and calculated arterial pH value derived from venous pH value using regression equation (predicted arterial pH = 1.09 + 0.86 [venous pH]) across a broad range of potential pH values

Observed ABG arterial pH value	Calculated arterial pH derived from observed VBG venous pH value	Difference
7.60	7.63	0.03
7.50	7.54	0.04
7.40	7.45	0.05
7.30	7.37	0.07
7.20	7.28	0.08
7.10	7.20	0.10
7.00	7.11	0.11
6.90	7.02	0.12
6.80	6.94	0.14
6.70	6.85	0.15
6.60	6.77	0.17
6.50	6.68	0.18

Gray cells indicate accurate prediction by regression equation within clinically equivalent limits, whereas clear cells do not.