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Can VBG analysis replace ABG analysis in emergency care?

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ABSTRACT

Blood gas analysis is an integral part of the assessment of emergency department (ED) patients with acute respiratory or metabolic disease. Traditionally ABG analyses have been used, but increasingly, emergency clinicians are using venous blood gas (VBG) analyses. This has been challenged, especially by respiratory physicians, as being too inaccurate. This clinical review, using case examples, summarises the evidence supporting use of VBG to guide management decisions. Arteriovenous agreement for pH is such that values are clinically interchangeable and agreement for bicarbonate is also close. Agreement for pCO_2 is poor with 95% limits of agreement of the order of 20 mm Hg (2.67 kPa); however, there is solid evidence that a venous pCO_2 ≤45 mm Hg (6 kPa) reliably excludes clinically significant hypercarbia. Evidence regarding arteriovenous agreement for base excess is unclear. Given knowledge of the performance characteristics of VBG analyses, integration of the clinical findings with VBG results is often sufficient to safely guide treatment decision making.

INTRODUCTION

Blood gas analysis has long been an integral part of the assessment of emergency department (ED) patients with acute respiratory or metabolic disease. Traditionally ABG analyses have been used, but these are painful for patients, are more technically difficult to collect and have a small incidence of serious adverse events such as vascular occlusion or infection.[1] Increasingly, emergency clinicians are using venous blood gas (VBG) analyses to guide management decision making, but this has been challenged, especially by respiratory physicians, as being too inaccurate.[2]

Rather than concentrating on the absolute numerical agreement between ABG and VBG of the various blood gas parameters, it is important to remember that blood gas analysis is one piece of information that is integrated with other data, in particular clinical data, to guide treatment decisions. It is also helpful to define the questions that you are wanting blood gas analysis to answer. I have summarised the key questions as I see them in table 1.

The aim of this article is, using case examples, to describe the evidence supporting use of VBG to guide management decisions and in particular to describe relevant evidence gaps.

A word on terms and statistics

When comparing the accuracy of a new test (in this case, VBG analysis) to a gold standard (in this case, ABG analysis), two parameters are of key importance. The first is the average (or mean) difference, which is the bias or fixed difference between the tests. Weighted mean difference combines the results of a number of studies by weighting the reported mean differences by sample size.

The other key concept is the 95% limits of agreement. This is a measure looking to identify outliers and is calculated as the average difference ±1.96 SDs of the difference. It tells us how far apart measurements by the two methods were likely to be for most individuals. If the width of the 95% limits of agreement is not clinically important, the two methods can be used interchangeably. For example, the reported weighted mean difference for pH is −0.033 with 95% limits of agreement generally ±0.1. So a venous pH of 7.1 would be estimated to reflect an arterial pH of about 7.13 and have a 95% probability of reflecting an arterial pH of 7.0–7.2.

Clinical decision making

The decision to use a VBG or an ABG in a particular patient will hinge on several factors. These might include the level of experience of the clinician, the clarity of the clinical presentation, the potential risks of VBG imprecision and the relative benefits of VBG for both patients and staff. In my experience, the vast majority of patients can be managed using VBG. That said, as with any test, if the result is discordant with the clinical situation, do an ABG analysis to check.

CASE 1: DIABETIC KETOACIDOSIS

Jane is a 26-year-old insulin-dependent diabetic. She attended ED with a 2-day history of nausea, vomiting and diarrhoea. On clinical examination, pulse was 120/min, BP 100/mm Hg, RR 30/min, and there were no specific abnormalities on cardiorespiratory or abdominal exam. Bedside glucose measurement simply read 'Hi'.

Jane's VBG result was pH 7.26, pCO_2 16 mm Hg (2.13 kPa), HCO_3 7.1 mmol/L, K 3.8 mmol/L, base excess (BE) −14 mEq/L and lactate 7.2 mmol/L.

The evidence

There are 13 studies exploring arteriovenous agreement of pH.[2] They have a total of 2009 patients, but individual studies range in size from 44 to 346 patients. Weighted mean difference is −0.033 with 95% limits of agreement generally ±0.1. Three studies, totally 265 patients, have specifically explored agreement in patients with diabetic ketoacidosis (DKA). Weighted mean difference is 0.02. Only one study reports 95% limits of agreement, which were −0.009 to 0.02.

Regarding pCO_2, eight studies (965 patients) have compared ABG and VBG pCO_2.[2] Although

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Table 1 Key questions for blood gas analysis to address

Respiratory conditions	Metabolic conditions
Is my patient hypoxic?	Is this patient acidotic/alkalotic?
Does my patient have respiratory failure?	What sort of acid–base disturbance do they have?
Is this patient a CO_2 retainer?	Is my treatment working?
Do I need to provide additional ventilatory support?	
Is my treatment working?	

the weighted mean difference is small (6.2 mm Hg; 0.83 kPa), 95% limits of agreement are up to −17.4 to +23.9 mm Hg (−2.32 to +3.19 kPa).

Eight studies (total 1211 patients) have investigated arteriovenous agreement for bicarbonate.[3] In those studies, the weighted mean difference is −1.3 mmol/L with 95% limits of agreement up to ±5 mmol/L.

Arteriovenous agreement regarding BE is less clear. There are only two studies, one reporting close agreement (mean difference 0.089; 95% limits of agreement −0.974 to +0.552)[4] and one in trauma patients reporting mean difference −0.3 BE units with 95% limits of agreement −4.4 to +3.9 BE units.[5]

Regarding lactate, three studies have reported a pooled mean difference of 0.25 mmol/L with 95% limits of agreement from −2 to +2.3 mmol/L.[6]

The agreement for various blood gas parameters is shown in table 2.

Clinical bottom line

The clinical picture is one of moderately severe DKA. Agreement between ABG and VBG pH is close enough for clinical interchangeability. Even allowing for the width of the 95% limits of agreement, pCO_2 and bicarbonate are low and lactate is high consistent with a metabolic acidosis with a significant lactic acidosis component. The bedside glucose is 'Hi'. Taken together, in my opinion, these are sufficient to confirm the diagnosis of DKA and guide initial treatment. Given the accuracy of VBG pH, resolution of acidosis can be reliably tracked using VBG pH alone.

A note of caution. Two studies have explored agreement between serum and blood gas potassium concentrations.[7 8] In both, the serum concentration was usually higher than the blood gas concentration, but 95% limits of agreement were wide with up to 66% being outside ±0.5 mmmol/L. Blood gas potassium concentration might provide useful information, especially if very high or very low, but cannot be relied upon to be an accurate reflection of serum potassium.

CASE 2: ACUTE RESPIRATORY DISEASE

Tran is a 74-year-old man with known chronic obstructive airways disease (COAD). He presented to ED with a 1-day history of worsening dyspnoea following a 'cold'. On examination, he was short of breath at rest, only able to speak in short phrases or words. Pulse was 125/min, BP 140/mm Hg, RR 35, oxygen saturation on air 86%, and on chest examination there was generally reduced breath sounds with scattered rhonchi but nothing focal.

VBG analysis showed pH 7.16, pCO_2 82.6 mm Hg (11.01 kPa) and HCO_3 28.8 mmol/L.

The evidence

As discussed previously, arteriovenous agreement for pH is close and clinically interchangeable.[3] This is also true for patients with COAD. Five studies (643 patients) have shown a weighted mean difference of 0.034 with 95% limits of agreement generally ±0.1.[3]

We have seen already that while weighted mean difference between arterial and venous pCO_2 is small, 95% limits of agreement are very wide precluding clinical interchangeability. The same holds for the subset of studies specifically exploring arteriovenous pCO_2 agreement in COAD. There are four studies (total patients 452 patients) and the weighted man difference is 7.26 mm Hg (0.97 kPa) with 95% limits of agreement: up to −14 to +26 mm Hg (−1.87 to +3.47 kPa). All three studies that report 95% limits of agreement have at least one end of that band >20 mm Hg (2.67 kPa).[3]

There seems to be reasonable arteriovenous agreement for bicarbonate.[3]

The clinical bottom line

On clinical grounds alone it is clear that Tran is hypoxic with significant work of breathing. The evidence is that the venous pH will be an accurate reflection of arterial pH. Even allowing for the wide limits of agreement, pCO_2 is high and coupled

Table 2 Summary of the evidence

Parameter	No. studies	No. patients	Weighted mean difference (bias)	95% limits of agreement*	Clinical interpretation
pH	13	2009	−0.033	Approximately ±0.1	Clinically interchangeable
pCO_2	8	965	6.2 mm Hg (0.83 kPa)	−17.4 to 23.9 mm Hg (−2.32 to +3.19 kPa)	Poor, unpredictable agreement
Bicarbonate	8	1211	−1.3 mmol/L	Approximately ±5 mmol/L	Probably close enough agreement for classification as high, normal or low
Base excess	2	429	Divergent results	Up to −4.4 to 3.9 BE units	Agreement unclear
Lactate	3	338	0.25 mmol/L	−2 to +2.3 mmol/L	May be close enough agreement for classification as high or normal

Parameter	No. studies	No. patients	Sensitivity for hypercarbia	NPV for hypercarbia	Clinical interpretation
pCO_2 ≤45 mm Hg (6 kPa) as a screening test for hypercarbia	4	529	100% (95% CI 97% to 100%)	100% (95% CI 97% to 100%)	Reliable screening test; congruence with clinical assessment required

*Interpreted from available data; not reported by all studies.
BE, base excess; NPV, negative pressure ventilation.

with the pH and near normal bicarbonate is sufficient evidence of acute hypercarbia and respiratory failure. In my opinion, this is more than sufficient evidence to confirm a diagnosis of acute respiratory failure requiring careful oxygen management and ventilatory support with non-invasive ventilation.

Given the accuracy of venous pH, it could be used together with clinical parameters (such as reduced work of breathing, improved oxygenation, pulse and RR) to monitor improvement and resolution of acute respiratory acidosis.

CASE 2: A VARIATION

Let us consider the same patient as in case 2 but with different clinical features. On examination, Tran can speak in short sentences, has a pulse of 110/min, BP of 140/mm Hg and RR of 30/min with oxygen saturation on air of 86%. His chest findings are the same.

This time the VBG shows pH 7.45, pCO_2 42 mm Hg (5.60 kPa) and HCO_3 28.7 mmol/L.

The evidence

Evidence already presented has demonstrated the clinical interchangeability of pH and reasonable close agreement of bicarbonate. It has also shown 95% limits of agreement of pCO_2 of ±20 mm Hg. The clinical question there is whether Tran has clinically significant hypercarbia not identified by the VBG analysis.

Four studies have explored whether there is a VBG level of pCO_2 that reliably rules out clinically significant hypercarbia.[3] Those studies have included 529 patients and established that a screening cut-off of VBG pCO_2 of 45 mm Hg (6 kPa) rules out clinically significant hypercarbia. Pooled sensitivity was 100% (95% CI 97% to 100%) and negative predictive value 100% (97% to 100%).

Clinical bottom line

In this variation of the scenario, Tran is hypoxic but not in acute respiratory failure and not significantly hypercarbic at the time of the test. That is not to say that if too high a level of oxygen was given he would not develop hypercarbia but the same would be true of an ABG. Both tests tell us what is happening now and cannot predict the future.

EVIDENCE GAPS

There are some limitations of the evidence that should be considered when interpreting it. Most of the studies are small and in diverse patient groups. There is also significant between-study variation in cohorts. As emergency clinicians, we are most interested in arteriovenous agreement in patients with abnormal values, but it is often hard to tell how many of these were in the studied cohorts.

There is some evidence that arteriovenous agreement regarding pH deteriorates as haemodynamic compromise increases, at least in unresuscitated patients.[9 10] More research is needed to clarify this.

There are no data in mixed acid–base disorders or in specific groups of interest such as severe sepsis or ventilated patients. There are minimal data in toxicological conditions.

CONCLUSION

While arteriovenous agreement for pH is such that values are clinically interchangeable and agreement for bicarbonate is close, agreement for pCO_2 is poor and for BE is unclear. That said, given knowledge of the performance characteristics of VBG analyses, integration of the clinical findings with VBG results is often sufficient to safely guide treatment decision making.

Contributors A-MK designed the review, collected and interpreted the data and drafted and revised the manuscript.

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Notes