Venous and arterial blood gases in respiratory failure

How to choose the most appropriate test for patients undergoing respiratory failure: a case-based critical reflection

Abstract
Arterial and venous blood gases are commonly performed operations in emergency departments. This case-based critical reflection examines the contemporary literature relating to the topic. An evidence-based approach to selecting the most appropriate test for each patient is discussed, aiming to minimise the need for unnecessary arterial sampling.

Keywords
arterial blood gas, venous blood gas, arterial or venous blood gas, respiratory failure, critical reflection, critical analysis

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ARTERIAL BLOOD gas (ABG) and venous blood gas (VBG) analysis are frequently used in emergency departments (ED) to assist in patient evaluation. This article uses a case-based critical reflection, the Driscoll reflective model 'what, so what, now what' (2007), to provide structure for learning and documentation. It also reviews contemporary literature to demonstrate an evidence-based approach to selecting the most appropriate investigation for each patient, depending on the information required, and argues that this could prevent unnecessary discomfort for patients and exposure to the potentially deleterious effects of ABG sampling.

What?
An 80-year-old woman presented to an ED with a two-day history of worsening shortness of breath, and pitting oedema to her lower limbs, sacrum and around her eyes. She had recently finished a course of antibiotics prescribed for a chest infection, and reported that her fever and productive cough had settled. Her past medical history included chronic obstructive pulmonary disease and congestive cardiac failure. Clinical examination also revealed bi-basal crackles, and the patient was diagnosed with, and treated for, pulmonary oedema. The clinical examination findings were:

General
- Alert.
- Looks unwell. 1L nasal oxygen (O₂) in situ.
- Speaking in short sentences.
- No jaundice, anaemia, clubbing, cyanosis, splinter haemorrhage or lymphadenopathy.
- Moist oral mucosa.
- Pitting oedema to lower limbs and around eyes.

Respiratory system
- Respiratory rate 24 with mild accessory muscle use.
- Bilateral, symmetrical chest expansion on looking and feeling.
- No wounds or bruising seen.
- No chest wall tenderness, surgical emphysema or tracheal deviation.
- Resonant percussion noted throughout.
- Bi-basal crackles with scattered expiratory wheeze.
- Peripheral oxygen saturations 83% on air, 92% on 1l nasal O₂.

Cardiovascular system
- No pallor.
- Skin warm to touch peripherally and centrally.
- No clamminess.
- Moist oral mucosa.
- Jugular venous pressure 5cm.
- Heart rate 80 (regular), blood pressure 146/46, temperature 36.6°C.
- Good volume radial and carotid pulses bilaterally.
- No radio-radial delay.
- No palpable heaves or thrills.
- Apex beat not displaced.
- No carotid bruit.
- Heart sounds 1 + 2. No added sounds or murmurs.
- No radial-femoral delay.
- Good volume dorsalis pedis pulses bilaterally.
- Pitting oedema on lower limbs to knee and sacrum.
Abdomen

- Suboptimal examination as the patient was unable to lie flat.
- No gross distension or scarring.
- Soft and non-tender.
- Bowel sounds present.

Central nervous system

- Glasgow coma score 15/15.
- Pupils equal and reactive 3mm.
- Blood glucose 4.8mmol/l.
- Moving all four limbs independently.

A VBG was performed because of the reliable oxygen saturation (SpO₂) trace, however following a review by a senior advanced clinical practitioner (ACP) it was advised that an ABG should be performed to assess for hypoxemia. This request prompted a review of the evidence in relation to this case, to determine whether an ABG had been required. The two blood tests were taken approximately 30 minutes apart and results are shown in Table 1.

So what?

Blood gas analysis is used in EDs for two main purposes, to assess acid-base state, and to establish respiratory function (Kelly 2010). Traditionally, blood gas analysis was performed on arterial blood, although this is painful for patients and has potentially serious complications, including infection and vascular injury or occlusion (Kelly 2010). Furthermore, ABG sampling is more technically difficult than VBG sampling (McKeever et al 2015).

Data from a recent meta-analysis demonstrates agreement between arterial and venous measurements of pH, HCO₃⁻ and base excess (BE), which means that it is possible to use either value interchangeably (McKeever et al 2015).

Unfortunately, the relationship between arterial and venous measurements of pH, HCO₃⁻ and pCO₂ is less strong (McKeever et al 2015). The evidence demonstrates an unacceptably high variability for pO₂ measurements, without a meaningful or consistent relationship (Byrne et al 2014). These data are shown in Table 3.

However, Koul et al (2011) reported a high degree of correlation between digital pulse oximetry (SpO₂) and oxygen saturation, as determined by ABG analysis (SaO₂), which could be used in conjunction with a pH to reduce the number of ABGs required. This approach could be flawed however, due to the non-linear relationship of SpO₂ and pO₂ caused by the sigmoid shape of the oxygen dissociation curve (Figure 1) (Byrne et al 2014).

In clinical practice, this means that a large change in pO₂ can result in only small changes to the SpO₂ at the upper limits of the curve, making the titration of supplemental O₂ extremely difficult (Byrne et al 2014).

The difference between arterial and venous pCO₂ measurements are less marked than pO₂ measurements, but Byrne et al (2014) noted that it is still large enough to influence patient management. The researchers found that arterial CO₂ is typically 0.5kPa less than venous CO₂ levels, with 95% confidence intervals of -1.4 to +0.3kPa (Byrne et al 2014).

These data were presented in Byrne et al’s (2014) meta-analysis, in which there was a statistically significant degree of heterogeneity between the studies, thus limiting the applicability of the findings and the authors’ conclusions.

While these data rule out a direct replacement of arterial CO₂ sampling with venous CO₂, there is

<table>
<thead>
<tr>
<th>Test</th>
<th>Venous blood gas</th>
<th>ABG on 1L O₂</th>
<th>Reference ranges</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>7.31</td>
<td>7.32</td>
<td>7.35-7.45</td>
</tr>
<tr>
<td>pO₂</td>
<td>7.16</td>
<td>9.0</td>
<td>11.1-14.4 kPa</td>
</tr>
<tr>
<td>pCO₂</td>
<td>7.41</td>
<td>7.39</td>
<td>4.27-6.40 kPa</td>
</tr>
<tr>
<td>Bi carbonate (HCO₃⁻)</td>
<td>25</td>
<td>26</td>
<td>22-26 mmol/l</td>
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<thead>
<tr>
<th>Test</th>
<th>Agreement</th>
<th>Comments</th>
</tr>
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<tbody>
<tr>
<td>pH</td>
<td>+0.035 pH units difference</td>
<td>n=252 good correlation</td>
</tr>
<tr>
<td>HCO₃⁻</td>
<td>-1.41 mmol/L mean difference (95% confidence interval -5.8 to +5.3 mmol/L)</td>
<td>n=905 good correlation</td>
</tr>
<tr>
<td>Base excess</td>
<td>+0.089 mmol/L mean difference (95% confidence interval -0.974 to +0.552 mmol/L)</td>
<td>n=103 Good correlation</td>
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<th>Test</th>
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<tr>
<td>pO₂</td>
<td>ABG is typically 4.9kPa greater than VBG (95% confidence interval +3.6 to +6.2kPa)</td>
<td>n=1151 Very poor correlation and large variability</td>
</tr>
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</table>
considerable evidence to support the use of venous CO₂ as a screening test for arterial hypercapnia (Kelly 2010). Two studies (Kelly et al 2002, 2005) demonstrated that a venous CO₂ of less than 6.0kPa could rule out arterial hypercapnia with a sensitivity of 100% and specificity of 51%. Laboratory-based analysers performed the testing in both trials, therefore the authors advised that further studies using point-of-care analysers should be undertaken (Kelly 2010).

A more recent study (McCanny et al 2012) confirmed the laboratory-based findings using point of care analysers, and reported a sensitivity of 100% and specificity of 34% in ruling out arterial hypercapnia if venous CO₂ is less than 6kPa (positive likelihood ratio of 1.51). A cut off of 6.0kPa was used, as Kelly (2014) determined this was a clinically significant hypercapnia when managing patients with type 2 respiratory failure. An overview of the data for pCO₂ agreement is shown in Table 4.

What?

Having reviewed the evidence for VBG and ABG correlation, it is clear that the patient described here did require the ABG that was advised by the senior ACP who reviewed her. There is poor correlation between pCO₂ when above 6.0kPa, and as the patient’s measurement was 7.41kPa on VBG, an ABG was required to further assess whether hypercapnia was present and, if so, to what degree. Additionally, the ABG result enabled accurate assessment of oxygenation, which would have allowed for titration of supplemental oxygen if required.

The literature demonstrates a good correlation between ABG and VBG levels of pH, HCO₃ and BE (Kelly 2010). The role of VBG in screening for hypercapnia is supported by numerous studies, and could reduce arterial sampling by 33% if used in the appropriate clinical contexts (Kelly 2010). This could provide clinicians with all the diagnostic values required, while reducing patients’ discomfort and exposure to the complications associated with ABG sampling (McKeever et al 2015).

References


