

Avoiding unnecessary arterial blood sampling in COPD exacerbations: a stab in the right direction

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Hospital admissions for exacerbations are major events in the lives of people with COPD. The prognosis for such patients is grim. Around one in seven will die within 3 months of admission, and fewer than half will still be alive at 5 years.^{1 2} The symptoms they experience are frightening and unpleasant,³ their quality of life is reduced⁴ and the restriction in their physical activity, which may persist for weeks after the onset of symptoms, increases the risk that they will become housebound.⁵ In comparison with other medical emergencies, such as myocardial infarction and stroke, progress in improving the management of COPD exacerbations has been depressingly slow. Arguably, the last major advance was the introduction of non-invasive ventilation, which took place sometime in the latter part of the last century. Against this backdrop, it is vital that, as a clinical and research community, we should tenaciously pursue all opportunities to improve the experience for these patients. In doing this, we must reflect critically on our existing practice and the pain and burdens it may present for those who endure it. In this issue of *Thorax*, McKeever *et al*⁶ do just this, by questioning the need for an investigation that many of us would consider almost sacrilegious to omit: the arterial blood gas.

Learning and refining the technique of arterial blood sampling is a rite of passage for medical students, junior doctors and increasingly other healthcare professionals. Evidence of their efforts is perhaps most conspicuous among the inpatient COPD population, where masses of haphazardly taped cotton-wool balls adorn the increasingly purpuric wrists of these unfortunate individuals. As McKeever *et al* have observed, the pain associated with arterial blood sampling is significantly greater than that for venous sampling, and failed attempts are more

numerous. Moreover, the risks of the procedure, including haematoma formation and distal ischaemia, are rare but potentially serious.^{7 8} Pain may be reduced by the prior administration of local anaesthesia⁹ but, contrary to the recommendations of guideline writers,¹⁰ those on the shop floor stubbornly fail to embrace this. McKeever *et al* took a step back and asked the important question of whether, in fact, an arterial blood sample is always needed in the initial assessment of patients with COPD exacerbations. The validity of their question receives circumstantial support from the fact that, despite recommendations,¹¹ audits of real-life practice consistently demonstrate that arterial blood sampling in this population is not universally applied.¹² In some cases, this will simply have been an omission, but in others it may reflect a clinical judgement that the procedure, and its associated discomfort, is unjustifiable in light of the clinical information already available. Such information will invariably include non-invasive measurement of oxygen saturations (SpO₂) and, often, the results of a venous blood analysis. These are the substitute tests that McKeever *et al* set out to evaluate against the gold-standard arterial blood analysis.

The investigators have strengthened the evidence base informing the use of venous blood gas analysis in the context of COPD exacerbations by performing a study specifically in this population, using peripherally sampled venous blood. Their study benefits from having recruited a realistic cohort of patients with COPD exacerbations, in sufficient numbers to produce quite precise estimates of the strength of agreement between the variables of interest. They compared values obtained for pH, PCO₂ and HCO₃⁻ from venous and arterial blood, and compared oxygen saturations from arterial blood analysis (SaO₂) with those obtained non-invasively by pulse oximetry (SpO₂). As recommended for this type of analysis,¹³ they described agreement using Bland–Altman plots and by calculating the mean difference (ie, bias) and the 95% limits of agreement. The latter describe the range within which 95% of differences are

expected to lie. If these limits (and their confidence intervals) do not stray into differences that we would consider clinically important, then we may, for practical purposes, infer that venous and arterial measurements could be used interchangeably. The potential benefits for patients of avoiding arterial puncture, simply by analysing the venous blood that is likely to be collected anyway, are readily appreciable.

The study found that venous pH tends to underestimate arterial pH very slightly, with a mean difference of 0.03 units (ie, venous pH would slightly overstate the severity of arterial acidosis). The limits of agreement for pH measurement suggest that, in general, the arterial pH is expected to lie within –0.05 to +0.11 of the venous pH (we are not given CIs for the limits of agreement, but from those provided for the mean difference we may infer that these estimates are quite precise). Whether differences of this magnitude are clinically meaningful is, to a large extent, dependent on the observed pH value itself. In the initial assessment of a patient with a COPD exacerbation, the finding most likely to alter management is probably an arterial pH <7.35. This would generally mandate hospital admission, modification of supplemental oxygen therapy and prompt consideration of ventilatory support.^{11 14} Thus, the level of agreement required for a venous pH close to this critical threshold is arguably greater than that required if the pH is well above this level.

The role of bicarbonate is less well defined in the context of COPD exacerbations. It serves to inform the assessment of chronic hypercapnic respiratory failure and the identification of metabolic, rather than respiratory, acidosis. The finding in this study that the arterial HCO₃⁻ generally lies within about ±3 mmol/L of venous HCO₃⁻ is informative. Again, however, the clinical implications of this will depend on the observed value and its clinical context. As the venous HCO₃⁻ approaches the lower limit of normal, one should be increasingly concerned that an alternative process may be active, and driving a metabolic acidosis, in which case analysis of arterial blood (including for lactate concentration) would be judicious. Error in this part of the measurement range will probably be of greater significance than it would for a high bicarbonate concentration.

In contrast to the findings for pH and HCO₃⁻, agreement in respect of PCO₂ levels and oxygen saturations was less reassuring. With the potential for venous PCO₂ (ie, P_vCO₂) to misestimate arterial

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PCO₂ (P_aCO₂) by almost 3 kPa, it is clear from this study that if accurate knowledge of P_aCO₂ is necessary for clinical decision making, an arterial sample is required. Likewise, with SpO₂ potentially diverging from SaO₂ by more than 10%, these are clearly not interchangeable measures of oxygen saturation. Interpreting the latter finding is further complicated by the fact that the reference variable, SaO₂, may either be derived or measured, depending on whether the blood gas analyser has co-oximetry capabilities. Derived SaO₂ values inevitably suffer from greater inaccuracy and variability due to the assumptions inherent in their calculation.

So, in light of these results, can we now omit arterial blood sampling in appropriately selected patients with a normal venous pH, and alter the current oxygen guidelines¹⁰ accordingly, as McKeever *et al* suggest? The study has made an important contribution and will undoubtedly inform the development of guidelines in this area. However, we would argue that the evidence supporting a wholesale practice change is not yet sufficient. This study must be interpreted in the context of its limitations, notably stemming from its single-centre design. There are several factors that could be particular to this centre, which may limit the generalisability of the study's findings. These include the procedural proficiency of the individuals who obtained the samples; the method used; the time taken to sample blood, decant it into a heparinised syringe and to perform blood gas analysis; and the particular analyser used. More importantly, however, the study did not seek to evaluate the effects of using the results of venous blood analysis to guide practice.

What will affect patients is not the degree to which a measured variable diverges from the gold-standard reference value, but the way in which the available clinical information—or the lack of information—affects treatment decisions and outcomes. A UK-wide randomised trial comparing the effects of treatment directed by the proposed algorithm⁶ against that directed by universal arterial blood gas analysis on length of stay, intensive care admissions and acceptability of care for patients with COPD hospitalised with exacerbations is now required before this approach can be universally applied.

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