EDITORIAL

Blood gas estimations from *arterialized* capillary blood *versus* arterial puncture: are they different?

J.M.B. Hughes

Blood gas estimations from arterialized capillary blood versus arterial puncture: are they different? The answer to this question is that the oxygen tension (PO₂) of arterial blood must be higher than the PO₂ of so-called arterialized blood flowing freely from the ear lobe after it has been pierced by a scalpel. This is because there is a gradient of PO₂ from around 13 kPa (98 mmHg) at the arterial end of the capillary bed to 5 kPa (38 mmHg) at the venous end. Fluid collected from the cut ear lobe is a mixture of blood from capillaries and venules. Nevertheless, it is well-known that under certain circumstances, the differences are so small that the arterial and arterialized estimations are, for practical purposes, identical. How is this possible?

As already mentioned, the normal arteriovenous difference for PO₂ at rest is 8 kPa (60 mmHg), increasing to 10 kPa (75 mmHg) on light exercise and to 70.7 kPa (530 mmHg) at least when breathing 100% oxygen. This difference can be reduced by increasing ear lobe blood flow relative to oxygen consumption by vasodilatation, either by heat or by application of a vasoactive cream. No one knows the magnitude of the changes induced in the human ear lobe by such manoeuvres, but it would be interesting to find out! For example, increasing the ratio of blood flow to oxygen consumption fivefold would reduce the arteriovenous oxygen content difference from 5 mL per 100 mL to 1 mL per 100 mL, and the arteriovenous PO₂ difference to 4.0 kPa (30 mmHg), assuming a normal PO2 of 13 kPa (98 mmHg). Provided sufficient vasodilatation could be achieved, arterial and venous Po2 in the ear lobe would tend to converge, and the arterialized PO2 would come to resemble the arterial PO_2 .

As indicated by Sauty *et al.* [1] in this issue, the arteriovenous PO_2 difference depends on the shape of the oxygen dissociation curve (ODC). As the arterial PO_2 falls, the arteriovenous PO_2 difference falls also, to 3.5 kPa (26 mmHg) at PO_2 8 kPa (60 mmHg) and to 2.3 kPa (17 mmHg) at PO_2 6 kPa (45 mmHg). With vasodilatation, these differences might reduce to 1.2 kPa (9 mmHg) and 0.67 kPa (5 mmHg), respectively. Thus,we might expect convergence of arterial and *arterialized* PO_2 values at PO_2 <8 kPa (60 mmHg) and some divergence when the arteriovenous PO_2 difference is higher, especially in hyperoxia. In fact, this is what is found.

In two recent studies [1, 2], where a Bland and Altman analysis of differences was used, there was a definite trend for the arterial-arterialized PO2 difference, plotted against the mean of the two estimates, to increase as the mean PO2 (breathing air) increased; excluding one point in the study by PITKIN et al. [2], there seemed to be a threshold at 9.3 kPa (70 mmHg) above which a significant divergence first appeared. The overall differences were small, but arterial PO2 was systematically higher than arterialized PO₂ by 0.61 kPa (4.6±4.6 (sD) mmHg) in the study by Sauty et al. [1] (n=115) and by 0.17 kPa (1.28 mmHg) (n=40) in the other series [2]. An earlier study [3] found a much bigger difference under hyperoxic conditions; the arterial-arterialized PO₂ difference averaged 6 kPa (45 mmHg) at a mean arterial Po2 of 68.8 kPa (516 mmHg) (n=18).

A spurious elevation of *arterialized PO*₂ (breathing air) may occur if the collection is not fully anaerobic, and the blood is partially exposed to room air during the sampling period [3]. This artefact acts in the opposite sense to the arteriovenous *PO*₂ gradient or venous admixture effect, and the two errors cancel out. This may explain the excellent agreement, independent of the level of arterial *PO*₂, between arterial and *arterialized PO*₂ reported by so many investigators (see [1–3] for bibliography). The "aerobic" effect may explain in part why *arterialized PO*₂ exceeded arterial *PO*₂ by up to 0.6 kPa (4.5 mmHg) in 17 out of 115 [1] and 18 out of 40 [2] of the comparisons in the latest series.

In the study by Sauty *et al.* [1] the arterial-*arterialized P*O₂ difference ranged from -0.5 kPa (-3.8 mmHg) to +2.4 kPa (+18 mmHg) with 95% confidence intervals of +1.76 kPa (13 mmHg) and -0.6 kPa (4.4 mmHg). The variance in the study by PITKIN *et al.* [2] was similar. Is this sufficiently accurate for clinical work? The practical answer is that *arterialized P*O₂ will detect the presence of arterial hypoxaemia with adequate sensitivity and accuracy, but that there will be some false positives, where *arterialized P*O₂ suggests a greater degree of arterial hypoxaemia than is actually present. Thus, it is a "fail safe" technique. *Arterialized P*O₂ values greater than 10.7 kPa (80 mmHg) should be treated with a modicum of caution.

Once the drawbacks of the method are recognized, there are good arguments for the use of *arterialized* PO_2 measurements. Firstly, ear lobe sampling can be carried out by nonmedically qualified persons; this is not, in general, true for arterial puncture. Secondly, PO_2 can be

EDITORIAL 185

measured on exercise without the need to insert an arterial cannula. The most obvious pitfall is a failure to use or learn good technique. Vasodilatation, a free flow of blood and "anaerobic" sampling are all vital. Manual massaging of the ear lobe, to encourage better flow of blood, is not recommended. Each laboratory should check for quality control against simultaneous sampling of arterial blood.

Samples of arterialized PO_2 have definite advantages over measurements of arterial oxygen saturation (S_a,O_2) using a pulse oximeter. S_a,O_2 is rather insensitive, because of the shape of the ODC, at $PO_2 > 10$ kPa (75 mmHg). Unfortunately, this is the PO_2 range at which the arterialized measurement performs least well! From arterialized blood carbon dioxide tension (PCO_2) and pH can be estimated, and important additional information is gained. As Sauty et al. [1] point out, the arterial-arterialized PCO_2 difference is negligible $(0.067 \text{ kPa} (0.5\pm 1.5 \text{ mmHg}))$ because of the small arteriovenous PCO_2 difference at rest (0.8 kPa (6.0 mmHg)).

To conclude, *arterialized* ear lobe sampling can be a valuable measurement in clinical practice, but it is only

an approximation of the arterial PO_2 . The lower the arterial PO_2 , the more accurate the estimate becomes. Saury *et al.* [1] have performed a valuable service in reminding us that "ear lobe sampling is not a reliable mirror of arterial PO_2 in adult patients". But, provided that one is aware of the pitfalls, ear lobe sampling is a useful and noninvasive monitor of arterial PO_2 , superior to more indirect methods, such as transcutaneous PO_2 and calculations of PO_2 from S_{a,O_2} with pulse oximetry.

References

- Sauty A, Uldry C, Debetaz L-F, Leuenberger P, Fitting J-W. Differences in Po₂ and Pco₂ between arterial and arterilized ear lobe samples. Eur Respir J 1996; 9: 186–189.
- Pitkin AD, Roberts CM, Wedzicha JA. Arterialised ear lobe blood gas analysis: an underused technique. *Thorax* 1995; 49: 364–366.
- Christoforides C, Miller JM. Clinical use and limitations of arterialized capillary blood for Po₂ determination. Am Rev Respir Dis 1968; 98: 653–657.