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 \( (P_{O_2}) \) of arterial blood must be higher than the \( P_{O_2} \) 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 \( P_{O_2} \) 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 estimates are, for practical purposes, identical.

As already mentioned, the normal arteriovenous difference for \( P_{O_2} \) at rest is 8 kPa (60 mmHg), increasing to 10 kPa (75 mmHg) on light exercise and to 70.7 kPa (516 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 \( P_{O_2} \) difference to 4.0 kPa (30 mmHg), assuming a normal \( P_{O_2} \) of 13 kPa (98 mmHg). Provided sufficient vasodilatation could be achieved, arterial and venous \( P_{O_2} \) in the ear lobe would tend to converge, and the arterialized \( P_{O_2} \) would come to resemble the arterial \( P_{O_2} \).

As indicated by SAUTY et al. [1] in this issue, the arteriovenous \( P_{O_2} \) difference depends on the shape of the oxygen dissociation curve (ODC). As the arterial \( P_{O_2} \) falls, the arteriovenous \( P_{O_2} \) difference falls also, to 3.5 kPa (26 mmHg) at \( P_{O_2} \) 8 kPa (60 mmHg) and to 2.3 kPa (17 mmHg) at \( P_{O_2} \) 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 \( P_{O_2} \) values at \( P_{O_2} \) <8 kPa (60 mmHg) and some divergence when the arteriovenous \( P_{O_2} \) 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 \( P_{O_2} \) difference, plotted against the mean of the two estimates, to increase as the mean \( P_{O_2} \) (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 \( P_{O_2} \) was systematically higher than arterialized \( P_{O_2} \) 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 \( P_{O_2} \) difference averaged 6 kPa (45 mmHg) at a mean arterial \( P_{O_2} \) of 68.8 kPa (516 mmHg) (n=18).

A spurious elevation of arterialized \( P_{O_2} \) (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 \( P_{O_2} \) gradient or venous admixture effect, and the two errors cancel out. This may explain the excellent agreement, independent of the level of arterial \( P_{O_2} \), between arterial and arterIALIZED \( P_{O_2} \) reported by so many investigators (see [1–3] for bibliography). The "aerobic" effect may explain in part why arterialized \( P_{O_2} \) exceeded arterial \( P_{O_2} \) 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 \( P_{O_2} \) 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 \( P_{O_2} \) will detect the presence of arterial hypoxaemia with adequate sensitivity and accuracy, but that there will be some false positives, where arterialized \( P_{O_2} \) suggests a greater degree of arterial hypoxaemia than is actually present. Thus, it is a "fail safe" technique. Arterialized \( P_{O_2} \) 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 \( P_{O_2} \) measurements. Firstly, ear lobe sampling can be carried out by non-medically qualified persons; this is not, in general, true for arterial puncture. Secondly, \( P_{O_2} \) can be
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 (SaO$_2$) using a pulse oximeter. SaO$_2$ 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 kPa (0.5±1.5 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. Sauty 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 SaO$_2$ with pulse oximetry.

References