Normalising renal tissue oxygen tension with higher inspired oxygen concentration may be falsely reassuring. Comment on *Br J Anaesth* 2020;125:192–200

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Keywords: hyperoxia; inspired oxygen concentration; renal perfusion; tissue hypoxia; tissue oxygenation

Editor—Iguchi and co-workers[1] reported an interesting and well-conducted piece of experimental work elucidating the effects of anaesthetic conditions on renal perfusion and oxygenation. They showed reductions in renal tissue oxygen tension (PtO_2) in response to reduced renal perfusion during general anaesthesia. However, the finding that PtO_2 can be restored to un-anaesthetised levels by increasing inspired oxygen fraction (FiO_2) merits further discussion. As the authors note, PtO_2 is determined by a balance between oxygen delivery and consumption and is therefore sensitive to reductions in oxygen delivery.[2] However, oxygen diffusion is also an important factor that complicates interpretation of this parameter.

Implantable probes to measure local oxygen tension have been used clinically in the management of conditions such as traumatic brain injury.[3] Although often thought of as localised probes, in reality their design inherently senses a macroscopic region of tissue (many capillary units). Such regions are not homogeneous. Tissue autofluorescence microscopy experiments have elegantly demonstrated substantial variation in redox state with periarterial tissue emission intensity consistent with higher levels of oxidative metabolism.[4] The PtO_2 measured with invasive probes reflects a spatial average over both periarterial and non-arterial tissue.

Although increasing renal blood flow could increase oxygen delivery, this seems unlikely as no such increase was demonstrated in a haemorrhage model when FiO_2 was increased.[5] Unlike the effect of increasing tissue blood flow, high arterial blood oxygen tensions (Pao_2, e.g. achieved by increasing FiO_2 as in the work of Iguchi and colleagues[1]) do not substantially increase oxygen delivery because of the shape of the oxy-haemoglobin dissociation curve.

In silico simulations[6] suggest that although PtO_2 may be increased with supraphysiological Pao_2, this effect is predominantly attributable to peri-arterial hyperoxia with little or no change occurring in non-peri-arterial regions. Thus, substantial volumes of tissue remain unaffected by this manoeuvre and may be hypoxic even when PtO_2 is reassuring. This may be particularly undesirable given the potentially harmful effects of supranormal Pao_2.[7]

In summary, caution should be exercised before concluding that improvements in PtO_2 owing to increasing FiO_2 necessarily represent a true state of metabolic rescue as this may be falsely reassuring. Clinicians should exercise caution before translating the apparently beneficial effects of supraphysiological FiO_2 on renal PtO_2 demonstrated in this work into clinical practice.

Declarations of interest

The authors declare that they have no conflicts of interest.

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doi: 10.1016/j.bja.2020.10.007
Advance Access Publication Date: 10 November 2020