LETTER TO THE EDITOR

Authors' response

We would like to thank Trinkmann *et al* for their comments[1] on our paper, 'Use of non-invasive haemodynamic measurements to detect treatment response in precapillary pulmonary hypertension',[2] and address the point raised regarding shunt correction. We are of the opinion that the in-built shunt correction algorithm in the inert gas rebreathing device may introduce measurement bias, as the assumptions made to correct for shunt flow may not be applicable to patients with pulmonary vascular disease. In the algorithm,[3] cardiac output (CO) is derived from pulmonary blood flow (PBF), oxygen content in arterial blood (CaO_2), oxygen content in pulmonary end-capillary blood (CcO_2) and oxygen uptake (VO_2) according to the formula $CO=1/(1/PBF+(CaO_2-CcO_2)/VO_2)$. The oxygen content of arterial blood and pulmonary end-capillary blood is calculated from the formulae $CaO_2=0.000139\times$haemoglobin concentration (Hb in g/dl)$\times SaO_2$ and $CcO_2=0.000139\times Hb\times ScO_2$ respectively, where SaO_2 denotes arterial oxygen saturation measured by pulse oximetry and pulmonary end-capillary oxygen saturation (ScO_2) is assumed to be 98%. However, ScO_2 may not reach 98% in patients with pulmonary hypertension due to failure of oxygen equilibration in the alveoli combined with a low mixed venous saturation. As a result of the destruction of pulmonary capillary beds and consequently reduced pulmonary capillary blood volume, red cell transit through pulmonary capillaries is more rapid.[4] This shortens the time available for oxygen diffusion to complete across the alveolar–capillary membranes, especially as PBF increases in response to exercise. This is compounded by systemic venous blood being more deoxygenated at the start of the equilibration process due to increased peripheral oxygen extraction in a low CO state associated with pulmonary hypertension. These two mechanisms contribute to resting arterial hypoxaemia and exercise desaturation commonly seen in pulmonary hypertension patients. Applying the shunt correction algorithm would overestimate CO, especially for exercise measurements. Therefore, we advocate the use of inert gas rebreathing PBF instead of derived CO in this patient group. As Trinkmann *et al* pointed out, other non-invasive techniques for measuring CO such as impedance cardiography and continuous-wave Doppler have the advantage of not requiring patient collaboration and may be more suitable for patients with advanced disease. However, they are not readily applicable during exercise and there are little clinical data on their use in patients with pulmonary hypertension.

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