How should we best determine the need for inflight oxygen in patients with pulmonary arterial hypertension?

The British Thoracic Society has recently updated the air travel recommendations suggesting that patients with precapillary pulmonary hypertension (PCPH) in functional classes (FCs) 3 and 4 should have inflight oxygen.¹ This replaces the 2004 recommendations which relied upon baseline oxygen saturation (SpO₂) to determine the need for oxygen or to undergo a hypoxic challenge test (HCT). We compared the relative impact of the 2004 and 2011 guidelines on the proportion of PCPH patients who would be recommended inflight oxygen.

We have recently published the effect a HCT would have on arterial oxygen levels in 36 patients with PCPH and baseline SpO₂>90%.² In this study arterial partial pressure of oxygen in PCPH patients fell by 2.36 kPa, (95% CI 2.06 to 2.66 kPa) which is similar to that reported in patients with other chronic respiratory diseases.^{3–4} Twenty-five per cent of this cohort failed the HCT and would according to the 2004 guidance have been recommended inflight oxygen. This is much lower than the 53% who would require inflight oxygen if we applied the FC criteria in the 2011 recommendations. In our cohort FC was not associated with failure of the HCT.

We then extrapolated these data to predict the impact of the new recommendations on the entire population of 221 PCPH patients attending the Scottish Pulmonary Vascular Unit. We estimate that the 2011 recommendations increase the proportion of patients who would be advised to fly with supplementary oxygen from 47% to 68% (based on the cohort derived HCT failure rate of 25%).

The theoretical concern with PCPH patients on airline flights is of worsening hypoxic pulmonary vasoconstriction, elevation of mean pulmonary artery pressures and acute failure of the right ventricle. The HCT has been set aside in the 2011 recommendations because acute alveolar hypoxia may not predict this chain of events but neither is there evidence that we can achieve this with FC. Indeed studies supporting this mechanism of respiratory distress in PCPH patients

during airline flight have yet to be performed. By contrast it has already been shown that during exercise and also sleep (both daily events) the mean pulmonary artery pressures can rise dramatically in PCPH patients and yet they do not suffer catastrophic right ventricle failure.⁵

In conclusion further studies need to be undertaken to assess the effect of hypobaric hypoxia on right ventricular function in these patients which will be important to identify accurately those who need inflight oxygen and avoid the unnecessary inconvenience and expense for those who do not.

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