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CORRESPONDENCE

Gas exchange in acute COPD exacerbation

To the Editor:

The article "Mechanisms of worsening gas exchange during acute exacerbations of chronic obstructive pulmonary disease" by BARBERÀ *et al.* presents an interesting analysis of the factors influencing gas exchange [1]. We would like to add some additional considerations. The authors assessed the influence of physiological variables using the Wagner West model of ventilation/perfusion (V'_A/Q') distribution. This is a static model and may be mis-leading if it is assumed that the V'_A/Q' distribution remains constant when arterial oxygen tension (P_{a,O_2}) and/or mixed venous oxygen tension ($P_{\bar{v},O_2}$) are varied. To demonstrate the likely quantitative and qualitative errors resulting from their assumptions we used a computer model that combines pulmonary gas exchange and blood flow control [2, 3]. We initiated the computer model with the V'_A/Q' characteristics and haemodynamic data of the chronic obstructive pulmonary disease (COPD) patients provided in the article, together with some assumed values required by the model. The model prediction of changes in P_{a,O_2}/inspired oxygen fraction (F_{I,O_2}) for each of the physiological variables when hypoxic pulmonary vasoconstriction (HPV) is absent (authors' prediction) and when HPV is present (our prediction) are shown in table 1.

These results show that the calculated effects of the combined parameters in the lung model, when HPV is present, equal 6.0 kPa (45 mmHg) which coincides with the actual measured decrease of $P_{a,O_2}/F_{I,O_2}$ in the patient group 6.0 kPa (45 mmHg). These effects are overestimated in the lung model when HPV is absent 11.3 kPa (85 mmHg). Furthermore, the relative contribution of each factor on $P_{a,O_2}/F_{I,O_2}$ in the lung model when HPV is present is less, compared to the absence of HPV, due to the homeostatic effects of HPV on P_{a,O_2}.

The use of this dynamic lung model that incorporates the HPV provides further insights into the effects of the complex interactions between oxygen consumption, cardiac output and V'_A/Q' imbalance in COPD patients with an acute exacerbation of COPD.

Table 1. – Analysis of the relative contributions of the factors that determined the change in ratio of arterial oxygen tension to inspired oxygen fraction calculated by means of the computer model in the presence and absence of hypoxic pulmonary vasoconstriction (HPV)

Factor	HPV	
	Present mmHg	Absent mmHg
V'_E	0	0
$\overline{Q}'$	5	10
V'_{O_2}	-35	-42
Extrapulmonary factors (V'_E, Q', V'_{O_2} combined)	-30	-33
V'_A/Q' inequality	-19	-61
All factors combined	45	85

The changes in minute ventilation (V'_E), cardiac output ($\overline{Q}'$), oxygen consumption (V'_{O_2}) and ventilation/perfusion (V'_A/Q') inequality are the same as in the article.

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REPLY

From the authors:

In their re-evaluation of our data, Noordegraaf and Marshall suggest that in our analysis of the relative contribution of different factors on the change in arterial oxygen tension (P_{a,O_2}) during exacerbations of chronic obstructive pulmonary disease (COPD) we assumed that hypoxic pulmonary vasoconstriction (HPV) was not present. This was not the case. Our analysis was based on the ventilation-perfusion (V'_A/Q') distributions that were actually measured in each patient using the multiple inert gas elimination technique, during the acute exacerbation episode and under stable clinical conditions. We are firmly convinced that in each of these conditions HPV was operating and contributed to V'_A/Q' matching. Indeed, we have previously shown that in COPD patients studied under different clinical conditions, inhibition of HPV with either 100% oxygen breathing or vasodilators results in substantial deterioration of V'_A/Q' distributions and worsening of hypoxaemia [1–4]. We believe that the absence of HPV in our study would have resulted in a further impairment of

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gas exchange during both the acute exacerbation episode and under stable clinical conditions. Therefore, we would like to emphasize that the absence of HPV was not at all an assumption in our study.

Since the analysis we performed was based on the actual measurements of V'_A/Q' distributions, it is likely that in the model proposed by Noordegraaf and Marshall the effect of HPV would have been superimposed on a condition where it was already present, thus overestimating its impact on pulmonary gas exchange. Nonetheless, we share their view that HPV plays a crucial role in modulating gas exchange in COPD patients.

Furthermore, it is interesting to note that in the analysis performed by Noordegraaf and Marshall, extrapulmonary factors (*i.e.* oxygen consumption and cardiac output) play an important role in modulating the P_{a,O_2} value, regardless of the effect of HPV. This reinforces one of the major messages of our study, that both oxygen consumption and cardiac output are crucial, each one on a different direction, in determining the final P_{a,O_2} value during acute exacerbations in COPD patients.

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