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Authors' Reply

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We thank Dr. Medford [1] for his comments regarding our recent study assessing the predictive value of exertional desaturation for lung function decline in patients with chronic obstructive pulmonary disease (COPD) using the Korean Obstructive Lung Disease (KOLD) cohort [2]. He queried whether pulmonary embolic disease or secondary pulmonary hypertension due to COPD might affect the results as confounding factors, and also asked about the availability of the electrocardiography, echocardiography and computed tomography (CT) angiography data.

Less than half of the participants underwent echocardiographic examination, which was not mandatorily performed in the KOLD cohort. Therefore, measurable data of tricuspid regurgitant (TR) flow using Doppler echocardiography were available in 17 out of 37 desaturators and in 57 out of 152 nondesaturators. The mean TR flow was numerically higher in the desaturator group (2.58 ± 0.80 vs. 2.32 ± 0.34 m/s), but this did not reach statistical significance ($p = 0.21$). However, the number of patients with TR flow equal to or greater than 2.8 m/s revealed a significant difference between the desaturator group (8 out of 17 patients, 47.1%) and the nondesaturator group (5 out of 57 patients, 8.8%; $p = 0.001$). Only 3 patients had a TR flow equal to or greater than 3.4 m/s.

Meanwhile, it is hard to review the baseline electrocardiography of the patients to assess P-wave dispersion or atrial arrhythmias because electrocardiographic examination was also optional and the results were not kept as images in the current electronic case

report form. Moreover, volumetric CT scans were performed at baseline in all of the participants, but done without contrast enhancement. Thus, pulmonary vascular abnormalities could not be evaluated.

We partly agree with Dr. Medford that pulmonary hypertension can be a potential confounding factor associated with exertional desaturation in predicting lung function decline. According to a study of the natural history of pulmonary hypertension in COPD, patients with exertional desaturation showed exercise-induced pulmonary hypertension and finally developed resting pulmonary hypertension [3]. In our study, the number of patients with measurable data of TR flow was less than half of the participants and was unevenly distributed in the desaturator and nondesaturator groups, so we cannot fully accept the significance of the difference in the number of patients with pulmonary hypertension between the groups. Actually, we think that exercise-induced pulmonary hypertension might have a greater effect on the results of our study rather than overt resting pulmonary hypertension.

In conclusion, without considering the effects of resting or exercise-induced pulmonary hypertension, we want to highlight that exertional desaturation can be a unique phenotype of COPD and a predictive factor of rapid decline in lung function in patients with COPD. The 6-min walk test can be easily done to assess exertional desaturation with low cost and without any harm. We hope that our response will help to clarify the interpretation of the data.

References

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