Title

Reply: Intrapulmonary shunt and alveolar dead space in a cohort of patients with acute COVID-19 pneumonitis and early recovery

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Reply to M. Ackermann and co-workers:

We thank M. Ackermann and co-workers for their interest in our recent publication [1]. These authors highlight our report of persistent increased alveolar dead space in 30% of 17 patients studied within ~2 months after an acute episode of mild–moderate COVID-19. After outlining their own work, demonstrating secondary pulmonary lobule pathologies found in the lungs of deceased COVID-19 patients, they then hypothesise that secondary lobular micro-ischaemia may be responsible for the elevated alveolar dead space found in our study.

Our study findings support persistent pulmonary vascular injury in recovered COVID-19 patients, for which M. Ackermann and co-workers provide pathological evidence. However, while we hypothesised that persistent microvascular obstruction was a likely explanation for the elevated alveolar dead space, M. Ackermann and co-workers provide an alternative and possibly complementary hypothesis linking functional and structural abnormalities. Both hypotheses, however, remain unproven, and the question of how the pathological findings from patients with fatal COVID-19 (provided by M. Ackermann and co-workers) relate to the functional physiological findings from patients recovered from an episode of mild–moderate COVID-19 (as in our study) remains unanswered. Resolving these issues will likely require a multidisciplinary approach combining physiological, pathological, radiological and interventional methodologies.

Shareable abstract (@ERSpublications)
Increased dead space following COVID-19 may be due to microvascular injury or secondary micro-ischaemia https://bit.ly/3Fypdwz

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Reference

1 Harbut P, Prisk GK, Lindwall R, *et al.* Intrapulmonary shunt and alveolar dead space in a cohort of patients with acute COVID-19 pneumonitis and early recovery. *Eur Respir J* 2023; 61: 2201117.