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LETTER

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Veno-venous extracorporeal CO_2 removal improves pulmonary hypertension in acute exacerbation of severe COPD

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Dear Editor,

Acute exacerbation of chronic obstructive pulmonary disease (COPD) typically leads to acute hypercapnic respiratory failure with respiratory acidosis, which requires the augmentation of alveolar ventilation by mechanical support. At best, non-invasive ventilation is implemented early in these patients as it reduces intubation rate and intubation-related complications, and, importantly, mortality. Recently, veno-venous extracorporeal CO_2 removal (vv-$ECCO_2R$) has been shown to rapidly correct even severe respiratory acidosis [1], and this technique is currently under investigation as an adjunct to treat severe COPD patients. Here, vv-$ECCO_2R$ is aimed at avoiding intubation or at reducing the time spent on invasive mechanical ventilation [2]. In ARDS patients there is some new evidence that vv-$ECCO_2R$ provides additional benefit by decreasing elevated systolic pulmonary artery pressure (PAPsys) both in animals [3] and in patients, where this effect reportedly depends on the amount of CO_2 removed [4]. Even though a strong correlation between pulmonary artery dilatation and severe COPD exacerbation was shown previously [5], the clinical consequences of an increased PAP occurring during COPD exacerbation are still unclear, and the impact of vv-$ECCO_2R$ on PAP in COPD exacerbation still needs to be elucidated.

We report on five patients (mean age 52 ± 13 years, four female) with severe COPD exacerbation and invasive mechanical ventilation following intubation, in whom vv-$ECCO_2R$ was initiated to reduce intubation time and in whom pulmonary artery catheterization was used for regular hemodynamic monitoring. All patients had failed at least two spontaneous breathing trials within 48 h before vv-$ECCO_2R$ was considered according to the study protocol of the "PALP^{TM}-COPD Trial" (www.clinicaltrials.gov: NCT02107222), which is planned to start in the near future.

During vv-$ECCO_2R$ mean PAP decreased from 36.8 ± 5.6 (baseline) to 27.0 ± 7.0 (15 min), to 26.6 ± 6.1 (30 min), to 24.4 ± 3.4 (45 min), and, eventually, to 22.8 ± 2.8 (60 min) mmHg (Fig. 1), while ventilator settings used for invasive ventilation were adapted accordingly. The mean blood flow used for vv-$ECCO_2R$ was 2.0 ± 0.5 L/min, and the mean sweep gas flow was 7.6 ± 1.7 L/min using a FiO_2 of 1.0. During 60 min of vv-$ECCO_2R$ mean $PaCO_2$ decreased from 95.0 ± 25.8 to 53.2 ± 13.7 mmHg, while mean pH increased from 7.27 ± 0.08 to 7.49 ± 0.09. At baseline mean cardiac index (CI) was 4.0 ± 0.49 L/min/m^2 and mean wedge pressure was 16.4 ± 2.9 mmHg, while further CI measurements during vv-$ECCO_2R$ were not reliable as a result of the artificially induced jet blood flow next to the right atrium. Finally, all patients were extubated successfully; one patient had to be reintubated and required tracheostomy and prolonged weaning.

To our knowledge, this case series shows for the first time that vv-$ECCO_2R$ has not only the potential to rapidly correct respiratory acidosis by lowering high $PaCO_2$ values but also to reduce elevated mean PAP values in severe COPD significantly. Mean PAP values were substantially elevated in our patients with exacerbated COPD and could be normalized within 1 h of vv-$ECCO_2R$. This may be of major clinical importance given the known deleterious effects of acute pulmonary hypertension in patients

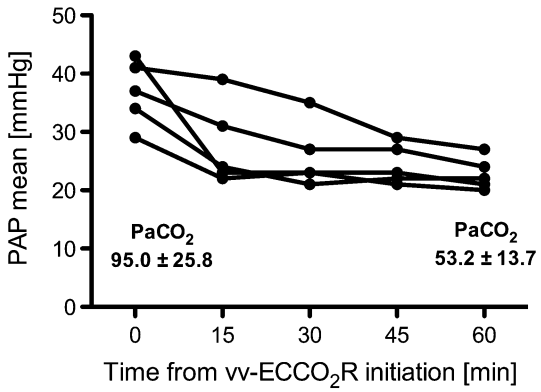

Fig. 1 Time course of mean pulmonary arterial pressure (PAP) after vv-$ECCO_2R$ initiation in severe COPD

with respiratory failure. Consequently, further studies should systematically investigate the impact of vv-ECCO$_2$R on right heart function in patients with pulmonary hypertension during severe exacerbation of COPD that requires mechanical ventilation to treat respiratory acidosis.

Compliance with ethical standards

Conflicts of interest C.K. received travel grants and lecture fees from Maquet, Rastatt, Germany; S.S. and A.P. have no conflicts of interest; T.M. received travel grants from Maquet, Rastatt, Germany; W.W. received fees for advisory board meetings and lectures from Maquet, Rastatt, Germany.

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