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LETTER

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Raising positive end-expiratory pressures in ARDS to achieve a positive transpulmonary pressure does not cause hemodynamic compromise

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Dear Editor,

High positive end-expiratory pressure (PEEP) is associated with improved survival in patients with moderate to severe acute respiratory distress syndrome (ARDS) [1], but high PEEP has also been reported to cause right heart failure and hemodynamic compromise [2]. In our previous trial of ventilator management in ARDS [3], setting PEEP to achieve a positive transpulmonary pressure estimated using esophageal manometry usually increased PEEP, often significantly, but also led to better blood oxygenation and respiratory compliance than the control PEEP. To determine whether such manipulations of PEEP degrade hemodynamic function, we performed a retrospective analysis of the 61 patients in the EPVent Trial, who were all ventilated to achieve a target range of arterial oxygenation after hemodynamic resuscitation by protocol [3]. Subjects in the intervention group had PEEP set to maintain oxygenation at a positive estimated transpulmonary pressure (P_L = airway pressure − esophageal pressure), whereas those in the control group had PEEP set according to a standard table based on oxygenation as in the ARDSNet low tidal volume trial [4]. Mean arterial pressure (MAP), heart rate, central venous pressure (CVP), vasopressor requirements, fluid balance, and simplified organ failure assessment (SOFA) scores were analyzed for the 3 days following enrollment. The primary between-group comparison was MAP, and secondary comparisons were cardiovascular SOFA score, urine output, creatinine level, and length-of-stay fluid balance.

Baseline characteristics and severity of illness were similar between groups. PEEP and plateau pressures were markedly higher in the intervention group (initial PEEP averaged 18.7 vs 11.0 cmH_2O and plateau pressure 31.4 vs 25.1 cmH_2O in intervention and control groups, respectively) [3].

Nonetheless, hemodynamic variables including MAP and cardiovascular SOFA score were similar between groups (Fig. 1). MAP improved slightly over the first 72 h in both groups (between-group $P = 0.576$), fluid balance was reduced toward zero in both groups (between-group $P = 0.245$), and urine output improved in both groups (between-group $P = 0.701$). The cardiovascular component of the SOFA score, fluid balance, creatinine levels, urine output, and MAP were compared between groups and tested by generalized estimating equations with adjustment for covariates. None was significantly affected by group assignment.

Although limited by the small sample size, these results indicate that raising PEEP as part of a strategy to optimize transpulmonary pressure in adequately resuscitated patients does not result in detectable impairment in hemodynamics, organ function measured by SOFA scores, fluid balance, or vasopressor requirement. With normal lungs, high alveolar pressures in the absence of adequate volume expansion may compress pulmonary vasculature and increase pulmonary vascular resistance (PVR), reducing cardiac output and impairing right heart function [5]. However, in ARDS, low lung volume and atelectasis may also increase PVR. Under these circumstances, raising PEEP could recruit collapsed lung and lower PVR. In this way, raising PEEP to prevent both lung collapse and overdistension may improve the hemodynamic function.

We conclude that in patients with ARDS, individualizing PEEP to optimize transpulmonary pressures using esophageal manometry does not compromise hemodynamic function. We are currently studying the

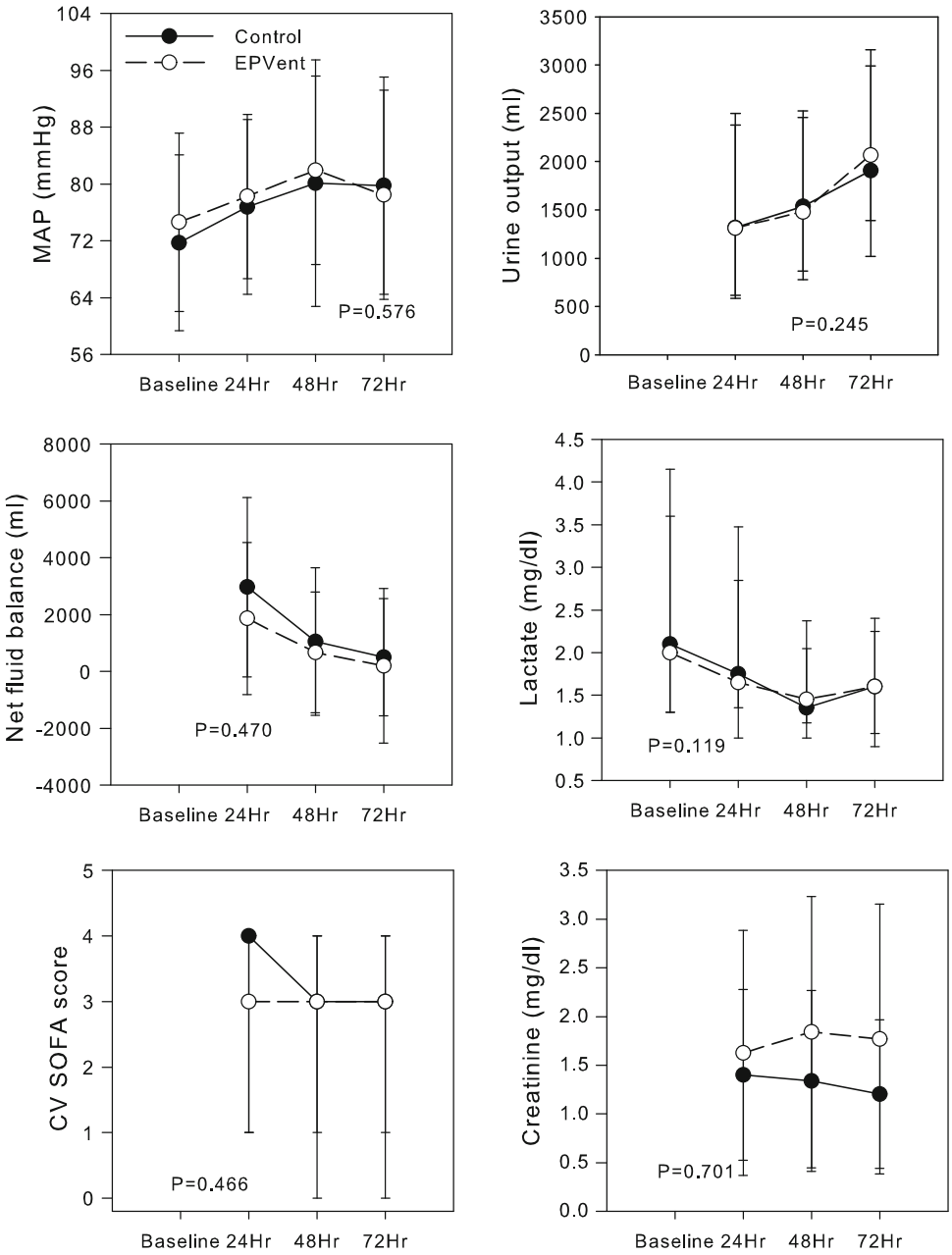

Fig. 1 Indicators of cardiovascular function in EPVent and control groups. Mean ± SD for mean arterial pressure (MAP), net fluid balance, and creatinine; median and IQR for 24-h urine output, serum lactate, and cardiovascular (CV) SOFA score. *P* values by repeated measures analysis of variance at 24, 48, and 72 h

hemodynamic effects of this strategy using echocardiography.

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Conflicts of interest On behalf of all authors, the corresponding author states that none of the authors has any conflict of interest.

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