expiratory esophageal pressure). Regarding electrical impedance tomography results, we are surprised that the authors reported regional compliance only for 10 patients, whereas electrical impedance tomography measurements were made on 18 patients.

Furthermore, the authors report good hemodynamic tolerance of high levels of PEEP in class III obese patients. We are then surprised that the authors did not report $\dot{Q}$ variations, easily measured during transthoracic echocardiography, which was performed on 17 patients. Lack of variation of vasoactive-inotropic score or mean arterial pressure does not exclude that high PEEP did not decrease $\dot{Q}$. To support this comment, in the ARDS swine study, the authors report a trend to lower $\dot{Q}$ (−13%, $P = 0.053$) and higher venous O_2 saturation (Sv_{O_2}) with higher PEEP, despite similar mean arterial pressure and vasoactive-inotropic score. The authors conclude that higher Sv_{O_2} reflects adequate systemic perfusion. However, because $\dot{Q}$ tends to be lower and V_{O_2} stable, it is more likely that higher Sv_{O_2} is explained by the rise of Sa_{O_2} due to higher PEEP.

In patients with class III obesity, we advocate that the strategy of high pleural pressures should be compared with *1*) complete evaluation of respiratory mechanics, which include checking for possible airway closure and expiratory airflow limitation, and *2*) complete evaluation of hemodynamics, including $\dot{Q}$, to set a sufficient level of PEEP. ■

Author disclosures are available with the text of this letter at www.atsjournals.org.

Mehdi Mezidi, M.D., M.Sc.
Lyon 1 University
Lyon, France

Pierre Esnault, M.D., M.Sc.
Sainte Anne Military Hospital
Toulon, France

Sami Hraiech, M.D., Ph.D.
Christophe Guervilly, M.D., M.Sc.*
Assistance Publique–Hôpitaux de Marseille
Marseille, France
and
Aix-Marseille University
Marseille, France

ORCID IDs: 0000-0002-8715-8323 (M.M.); 0000-0001-9066-6591 (C.G.).

*Corresponding author (e-mail: christophe.guervilly@ap-hm.fr).

References

1. De Santis Santiago R, Teggia Droghi M, Fumagalli J, Marrazzo F, Florio G, Grassi LG, *et al*.; Lung Rescue Team Investigators. High pleural pressure prevents alveolar overdistension and hemodynamic collapse in ARDS with class III obesity. *Am J Respir Crit Care Med* [online ahead of print] 02 September 2020; DOI: 10.1164/rccm.201909-1687OC.
2. Coudroy R, Vimpere D, Aissaoui N, Younan R, Bailleul C, Couteau-Chardon A, *et al*. Prevalence of complete airway closure according to body mass index in acute respiratory distress syndrome. *Anesthesiology* 2020;133:867–878.
3. Pankow W, Podszus T, Gutheil T, Penzel T, Peter J, Von Wichert P. Expiratory flow limitation and intrinsic positive end-expiratory pressure in obesity. *J Appl Physiol (1985)* 1998;85: 1236–1243.

Reply to Mezidi *et al.*

From the Authors:

We appreciate the thoughtful letter of Dr. Mehdi Mezdi and colleagues addressing our recent manuscript (1). The authors have raised several key pulmonary and hemodynamic questions on the ventilation of patients with obesity that should be investigated further. Our discussion here is limited and is focused on the apparent paradoxical hemodynamic response to lung recruitment in patients with severe obesity with an average body mass index (BMI) of 57 ± 12 kg/m^2 and an average esophageal pressure range of 17–20 cm H_2O. To address this point, we present novel data, documenting the hemodynamic effects of a lung-recruitment maneuver in two patients who were hemodynamically unstable, had severe obesity, and were treated by the Massachusetts General Hospital Lung Rescue Team (2).

Case 1

A 59-year-old woman with a BMI of 59 kg/m^2 (predominantly gynoid in distribution) was intubated because of aspiration pneumonia. The Lung Rescue Team was consulted in the setting of worsening hypoxemia (Pa_{O_2}/FI_{O_2} of 55 mm Hg with positive end-expiratory pressure [PEEP] of 15 cm H_2O) in septic shock requiring infusion of four inotropic-vasopressor medications. Before the recruitment maneuver was performed, the patient had a mean arterial pressure (MAP) of 70 mm Hg and a heart rate of 126 beats/min (bpm). After the recruitment maneuver and PEEP titration were performed and a best PEEP of 23 cm H_2O was found, the patient's MAP increased to 84, and her heart rate was 132 bpm. During the ventilatory procedure, inotropic-vasopressor agents were not adjusted. Figure 1 (case 1) shows that increased positive airway pressures translated into a progressive increase in arterial pressure. In the following hours at a PEEP of 23 cm H_2O, inotropic-vasopressor requirements were reduced by more than 50% (no fluid challenges were administered).

Case 2

A 58-year-old man with a BMI of 40 kg/m^2 (predominantly android in distribution) was intubated in the setting of pneumothorax and pulmonary contusions after a high-velocity motor-vehicle accident. The Lung Rescue Team was requested because of worsening oxygenation (Pa_{O_2}/FI_{O_2} of 99 mm Hg with

Originally Published in Press as DOI: 10.1164/rccm.202009-3641LE on October 20, 2020

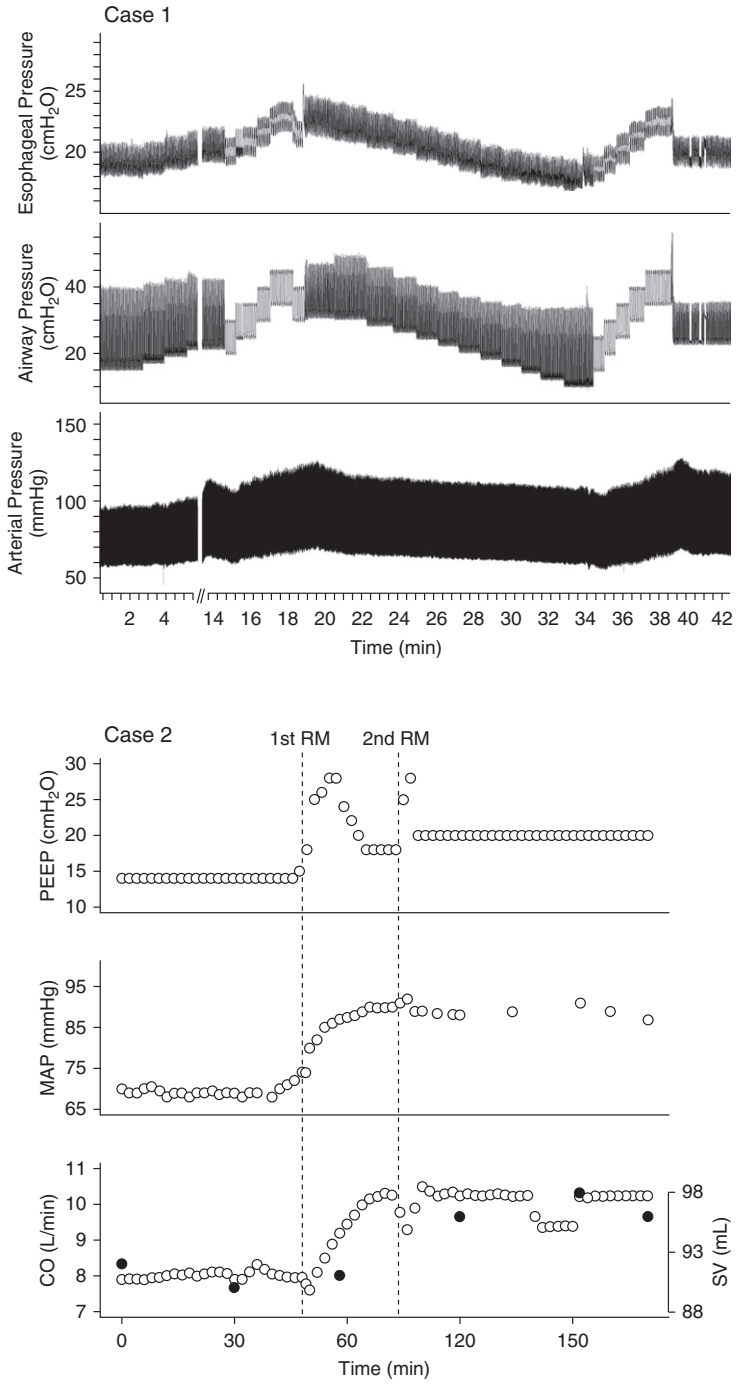

Figure 1. Case 1: Continuous recording of esophageal pressure (centimeters of water), airway pressures (centimeters of water), and invasive arterial pressure (millimeters of mercury) during the recruitment maneuver (RM) and positive end-expiratory pressure (PEEP) titration by the Lung Rescue Team is shown. Note that the driving pressure decreased from 17 cm H_2O (baseline) to 10 cm H_2O after the RM, suggesting lung recruitment. Doses of intravenous inotropic-vasopressor medications were unchanged before, during, and after RM and PEEP titration. The doses of inotropic-vasopressor medications were as follows: epinephrine, 5 μg/min; norepinephrine, 33 μg/min; phenylephrine, 25 μg/min; and vasopressin, 0.04 U/min. After 2 hours at 23 cm H_2O of PEEP, epinephrine and phenylephrine were stopped, norepinephrine was decreased to 25 μg/min, and vasopressin was unchanged. After 4 hours, norepinephrine was decreased further to 21 μg/min, and vasopressin was unchanged. Case 2: CO (L/min), stroke volume (SV; ml/min) (both measured by transpulmonary thermodilution), MAP (millimeters of mercury), and PEEP (centimeters of water) before, during, and after the Lung Rescue Team intervention are shown. Open circles indicate CO values. Solid circles indicate SV values. Intravenous inotropic-vasopressor medication requirements before the RM were as follows: epinephrine, 3 μg/min; norepinephrine, 50 μg/min; and vasopressin, 0.08 U/min. Epinephrine was reduced to 2 μg/min during alveolar recruitment because of systemic hypertension. After 2 and 4 hours at 20 cm H_2O of PEEP, epinephrine was decreased to 1 μg/min, and norepinephrine and vasopressin were unchanged (50 μg/min and 0.08 U/min, respectively). CO = cardiac output; MAP = mean arterial pressure.

PEEP of 14 cm H_2O) in the setting of requiring three inotropic-vasopressor medications (MAP of 72 mm Hg and heart rate of 94 bpm). During the recruitment maneuver, $\dot{Q}$ increased by 2.7 L/min, stroke volume increased by 8 ml/beat (both measured by transpulmonary thermodilution), and MAP increased by 20 mm Hg (Figure 1 [case 2]). We also observed a decreased stroke volume variation (from 16% to 6%) and no changes in central venous pressure (13–14 cm H_2O). His heart rate was 103 bpm. PEEP was set at 20 cm H_2O. The improved blood pressure allowed the epinephrine dose to be decreased rapidly during and in the hours after lung recruitment (no fluid challenges were administered).

Taken together, both the data presented in our manuscript and these two cases make us hypothesize that *high pleural pressure acts like a shield for the cardiovascular system against high ventilator pressures*. Prior investigations of the effects of positive-pressure ventilation on heart–lung interactions did not examine the role of high baseline pleural pressure (3–5).

As shown in the patients represented in Figure 1, the counterbalance of high pleural pressure due to obesity permitted the use of high airway pressures that not only improved respiratory system compliance (reduced driving pressure) and lung volumes but also raised $\dot{Q}$ and systemic blood pressure. The increased pleural pressure prevents high-airway-pressure lung injury (6) and hemodynamic collapse (7) that might otherwise occur during high-pressure ventilation in patients with low baseline pleural pressures. Future studies are needed to determine the validity of our hypothesis. ■

Author disclosures are available with the text of this letter at www.atsjournals.org.

Aditi Balakrishna, M.D.
Raffaele Di Fenza, M.D.
Massachusetts General Hospital
Boston, Massachusetts
and
Harvard Medical School
Boston, Massachusetts

Caio C. A. Morais, Ph.D.
Massachusetts General Hospital
Boston, Massachusetts

David A. Imber, M.D.
Massachusetts General Hospital
Boston, Massachusetts
and
Harvard Medical School
Boston, Massachusetts

Pankaj Arora, M.D.
University of Alabama at Birmingham
Birmingham, Alabama

Robert M. Kacmarek, Ph.D.
Massachusetts General Hospital
Boston, Massachusetts
and
Harvard Medical School
Boston, Massachusetts

Roberta De Santis Santiago, M.D., Ph.D.
Lorenzo Berra, M.D.*
Massachusetts General Hospital
Boston, Massachusetts

ORCID IDs: 0000-0002-5675-7139 (R.D.F.); 0000-0003-2702-2093 (L.B.).

*Corresponding author (e-mail: lberra@mgh.harvard.edu).

References

1. De Santis Santiago R, Teggia Droghi M, Fumagalli J, Marrazzo F, Florio G, Grassi LG, *et al*.; Lung Rescue Team Investigators. High pleural pressure prevents alveolar overdistension and hemodynamic collapse in ARDS with class III obesity. Am J Respir Crit Care Med [online ahead of print] 02 September 2020; DOI: 10.1164/rccm.201909-1687OC.
2. Spina S, Capriles M, De Santis Santiago R, Florio G, Teggia-Droghi M, Grassi L, *et al*.; Lung Rescue Team. Development of a lung rescue team to improve care of subjects with refractory acute respiratory failure. *Respir Care* 2020;65:420–426.
3. Vieillard-Baron A, Matthay M, Teboul JL, Bein T, Schultz M, Magder S, *et al*. Experts' opinion on management of hemodynamics in ARDS patients: focus on the effects of mechanical ventilation. *Intensive Care Med* 2016;42:739–749.
4. Mahmood SS, Pinsky MR. Heart-lung interactions during mechanical ventilation: the basics. *Ann Transl Med* 2018;6:349.
5. Lemaire F, Teboul JL, Cinotti L, Giotto G, Abrouk F, Steg G, *et al*. Acute left ventricular dysfunction during unsuccessful weaning from mechanical ventilation. *Anesthesiology* 1988;69:171–179.
6. Kolobow T, Moretti MP, Fumagalli R, Mascheroni D, Prato P, Chen V, *et al*. Severe impairment in lung function induced by high peak airway pressure during mechanical ventilation: an experimental study. *Am Rev Respir Dis* 1987;135:312–315.
7. Katira BH, Engelberts D, Otulakowski G, Giesinger RE, Yoshida T, Post M, *et al*. Abrupt deflation after sustained inflation causes lung injury. *Am J Respir Crit Care Med* 2018;198:1165–1176.

Bedside Evaluation of Pulmonary Embolism by Saline Contrast–enhanced Electrical Impedance Tomography: Considerations for Future Research

To the Editor:

We read with great interest the article by He and colleagues (1) entitled "Bedside Evaluation of Pulmonary Embolism by Saline Contrast Electrical Impedance Tomography Method: A Prospective Observational Study." The authors found that pulmonary embolism (PE)-invoked regional perfusion defection could be detected with saline-contrasted electrical impedance tomography (EIT) and claimed that the method showed high sensitivity and specificity for diagnosis of PE. However, several factors potentially affecting the reported findings should be discussed.

Supported by the National Natural Science Foundation of China (grant no. 81971807).

Originally Published in Press as DOI: 10.1164/rccm.202009-3640LE on October 22, 2020