from mechanical ventilation showed that 13% of their patients had a pleural effusion at the time of ventilator weaning, but that there was no relationship to the successful discontinuation of ventilation. Indeed, in their discussion on the potential reasons why pleural effusions might influence ventilator weaning failure, they identified three principal mechanisms. The first of these was the impact on respiratory mechanics, suggesting that large pleural effusions could reduce the end-expiratory lung volumes. The second mechanism was linked with the potential for pleural effusions to have an impact on the impairment of gas exchange, stating that associated lung collapse caused by the effusion could increase hypoxemia due to ventilation perfusion mismatch or intrapulmonary shunting. The third mechanism that they outlined related to the potential impact on cardiac filling pressures, with pleural effusions potentially increasing these filling pressures, and as a result, weaning-induced pulmonary edema. We contend that this last mechanism seems far less likely given that it would have to be the result of a significant leftward shift of the interventricular septum or some another cause of left-sided systolic or diastolic dysfunction. What is far more common is that pleural effusions often are the consequence of high left atrial pressure (*i.e.*, forming due to hydrostatic forces), and therefore represent the same underlying pathophysiologic state, that is heart failure or fluid overload. The elevated left atrial pressure, when coupled with increased venous return that occurs with reduced intrathoracic pressures during weaning, may result in pulmonary edema.

Furthermore, the authors failed to elaborate on a potentially even more important and mechanically simple mechanism; namely, mechanical compression of the lung by the effusion that could subsequently increase the pulmonary vascular resistance. Indeed, it is well known that pulmonary vascular resistance is optimal at functional residual capacity.[2] Anything that reduced this functional residual capacity (such as a pleural effusion) could increase pulmonary vascular resistance, and subsequently cause right ventricular dysfunction,[3] leading to an impairment in the ability to successfully wean from ventilation. However, because they did not measure pulmonary artery pressures, nor have an identified subset of patients with preexisting right ventricular dysfunction, they were not able to determine whether pleural effusions would have a negative impact on this patient subset. Indeed, this subset of patients is increasingly common and as a result, their negative study (*i.e.,* not being able to demonstrate that pleural effusions had any impact on weaning), should likely be specifically applied to the nonpulmonary hypertensive patient, with no preexisting right ventricular dysfunction.

Competing Interests

The authors declare no competing interests.

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Accurate Quantification of Pleural Effusion and Cofactors Affecting Weaning Failure

To the Editor:

In a recent issue of Anesthesiology, we read with great interest the article by Dres *et al.*,[1] who prospectively studied the prevalence and risk factors of pleural effusion in patients in the intensive care unit. They showed that the prevalence of pleural effusion had no significant impact on weaning failure, the duration of mechanical ventilation, or the intensive care unit length of stay. We appreciate this research for providing insight into the presence of pleural effusion at the time of liberation from mechanical ventilation among patients in the intensive care unit.

However, several factors that could potentially affect the study results should be discussed. First, the procedure for quantification of pleural effusion is still controversial. The authors adopted the procedure recommended by Balik *et al.*,[2] who quantified the pleural effusion volume using the following formula: pleural effusion volume (ml) = 20 × Sep (mm), where Sep was defined by Balik *et al.* as the maximal end-expiratory distance between the parietal and visceral pleura on ultrasound. However, Balik *et al.*[2] suggested several potential limitations associated with this procedure. They excluded patients with a small volume of pleural effusion (Sep less than 10 mm), Sep and pleural effusion were not linearly correlated in patients with a Sep of less than 17 mm (*i.e.*, pleural effusion of less than 340 ml), and the Sep value was affected by patient height (size of the thoracic cavity). However, Dres *et al.*[1] included patients with a small volume of pleural effusion, and information regarding the patients' height is lacking. An additional analysis with consideration of these factors would be helpful. Furthermore, whether the pleural effusions were detected unilaterally or bilaterally and whether the total volumes were calculated as a sum remains unclear. Because the effect of pleural effusion on the respiratory condition and gas exchange might differ, unilateral and bilateral effusions should be analyzed separately.

Second, information regarding interventional and supportive therapy after extubation is lacking. Noninvasive ventilation and high-flow nasal cannula deliver positive pressure to the lungs without intubation, thus improving the lung volume and unloading the respiratory muscles. Previous studies demonstrated that the prophylactic use of noninvasive ventilation and high-flow nasal cannula reduced the risks of postextubation respiratory failure and reintubation.[3,4] Considering the effects of these supportive therapies is important to ensuring accurate evaluation of the effect of pleural effusion.

Third, a failed spontaneous breathing trial and an extubation requiring reintubation should be analyzed separately. Extubation failure is commonly defined as the inability to sustain spontaneous breathing after removal of the tracheal tube. Although the most common cause of extubation failure is respiratory failure, which can be evaluated by a spontaneous breathing trial, other frequent causes include airway edema, excessive secretions, inadequate muscle strength, and glottic incompetence.[5] The presence of pleural effusion does not appear to affect these causes equally. Provision of the etiologies of extubation failure, and separate analysis of a failed spontaneous breathing trial and extubation requiring reintubation would be helpful to ensure a better understanding of the impact of pleural effusion.

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Competing Interests

The authors declare no competing interests.

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In Reply:

We thank Dr. Vetrugno *et al.*, Drs. Jacobsohn and Grocott, and Dr. Iwasaki *et al.* for their interest and positive appreciations of our study, "Prevalence and Impact on Weaning of Pleural Effusion at the Time of Liberation from Mechanical Ventilation: A Multicenter Prospective Observational Study," recently published in Anesthesiology.[1]

As pointed out by Dr. Vetrugno *et al.*, we used a slightly different method of estimating pleural effusion volume than the method used by Balik *et al.*[2] In the study by Balik *et al.*,[2] patients were investigated supine with a mild torso elevation of 15°, whereas in our study pleura ultrasound was performed while patients were semirecumbent. We choose this approach because pleura ultrasound was performed at the end of the spontaneous breathing trial, which requires the patients to be semiseated. Accordingly, Dr. Vetrugno *et al.,* as well as Dr. Iwasaki *et al.,* suggested that our method could misclassify some patients and potentially bias our findings. We wish to point out, however, that in our study, patients were classified as "no or small pleural effusion" or "moderate to large pleural effusion" based on the British Thoracic Society (BTS) classification[3] rather than on the Balik formula.[2] Dr. Vetrugno *et al.* also challenged the sample size of our study given that the majority of patients with pleural effusion had "no or small" pleural effusion. This comment is legitimate, and we agree that further studies are required to investigate specifically the impact of large pleural effusion on weaning outcome.

Drs. Jacobsohn and Grocott suggested that pleural effusion may influence weaning outcome through a mechanism that we did not consider, the increase in pulmonary vascular resistance. Although we are ready to believe that this mechanism may be of relevance, we were not able to find any study dealing with this interesting topic.

Dr. Iwasaki *et al.* commented on the lack of information regarding laterality, calculation of total pleural effusion volume, and height of the patient. We would like to point out that most of these data are shown in the Results section of our article as well as in figures. In fact, it is noted in the Methods section that "On average, the mean fluid volume was (mean ± SD) 509 ± 408 ml on the left side and 411 ± 329 ml on the right side." Table 1 displays the sum of volume of pleural effusion (left + right), which is (median [interquartile range]) 80 (0 to 150) ml for "no or small pleural effusion" and 900 (600 to 1,200)