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"A Message in the Bottle"

To the Editor:

We read with interest the work by Dres *et al.*[1] We would like to highlight some aspects that deserve particular consideration. First of all, the authors scanned the basal pleural space, estimating the effusion volume according to the British Thoracic Society classification as small, moderate, or large.[2] They also used the equation proposed by Balik *et al.*[3] to estimate the effusion volume at the maximal end-expiratory pleural distance between the parietal and visceral pleura. Specifically, the authors stated that they performed the ultrasound exam in the semirecumbent position, with the patient's torso reclined at about 45°, as free fluids accumulate at the lung bases due to gravity. We would tend to disagree with the authors at this point considering that the patients of Balik *et al.*[3] were investigated supine with a mild torso elevation of 15°; furthermore, the mean prediction error of this equation is quite high (158 ± 160 ml). The patient's position has a high impact on the estimation of pleural effusion volume. Furthermore, a consistent evaluation of the effusion volume is very challenging for many reasons (*e.g.,* tall people have a larger thoracic cavity area, diaphragm position, phrenic nerve palsy, diaphragmatic hernia), which was not considered or commented on by the authors. The authors also failed to report the laterality of the pleural effusions: It is well known that the ultrasound assessment of pleural effusions is overestimated on the left side, because the heart increases the fluid level (like a stone in a water receptacle). To overcome these limitations, other methods of pleural effusion estimation have been proposed using a transthoracic lung ultrasound approach.[3–6] However, as correctly stated in the study limitations acknowledged by the authors, the biggest problem lies in the numbers. The overwhelming majority of patients had a small pleural effusion, which has barely any impact on patient ventilation and hardly affects complex outcomes such as duration of mechanical ventilation, weaning success, and intensive care unit length of stay. The very low number of patients with moderate to large pleural effusions is thus an important limitation to the study. Consequently, a much larger study is needed. The authors calculated the sample size for their study, starting from the random assumption that a proportion of patients with a pleural effusion of 25% would be found in the group of patients with successful weaning. From these assumptions, the calculation of 136 patients for the sample size is correct. However, the authors should have recruited 68 patients per group, and not 51 patients with pleural effusion *versus* 85 patients without. To conclude, we believe the study is up to date and interesting; however, keeping the aforementioned limitations in mind, we are still far from reaching a definite conclusion on the real impact of pleural effusion on weaning.

Competing Interests

The authors declare no competing interests.

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Impact of Weaning from Mechanical Ventilation: The Importance of Pleural Effusions and Their Effect on Pulmonary Vascular Resistance

To the Editor:

The recent multicenter prospective observational study by Dres *et al.*[1] examining the impact of pleural effusions on liberation

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.