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)

ml for "moderate to large." In addition, Table 2 displays information on laterality: Pleural effusion was bilateral in 17/79 (21%) patients with weaning success and in 12/57 (22%) patients with weaning failure. As per request by Dr. Iwasaki *et al.*, we provide here the height of our patients, which was 168 ± 14 cm in patients with "moderate to large pleural effusion" and 168 ± 24 cm in patients with "no or small pleural effusion." Later, Dr. Iwasaki *et al.* suggested that the impact of pleural effusion might differ according to the postextubation ventilation strategy: noninvasive ventilation, high-flow oxygen, or standard oxygen. Although we definitely share the concerns raised, we are unable to address this issue. A comprehensive understanding of the interaction between postextubation ventilation strategy and the impact of pleural effusion would require specific measurements of breathing pattern and lung mechanics. Given that our study was mostly observational, we did not aim at investigating this question. Dr. Iwasaki *et al.* suggested that our findings would have been different if, rather than comparing weaning success *versus* weaning failure, we had compared success *versus* failure of spontaneous breathing trial. In response to this comment, we reassessed our data and found a "moderate to large" pleural effusion in 7/45 (16%) of patients who failed the spontaneous breathing trial and in 11/91 (12%) of patients in whom the spontaneous breathing trial was successful (P = 0.60).

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Competing Interests

Research contracts with Medtronic (Dublin, Ireland; to Dr. Dres), Maquet (Rastatt, Germany; to Dr. Dres), and Philips (Amsterdam, The Netherlands; to Dr. Dres). Dr. Dres also has received personal fees from Medtronic (Dublin, Ireland), Maquet (Rastatt, Germany), and MSD (Courbevoie, France). Dr. Demoule received personal fees from Pulsion Medical System (Feldkirchen, Germany) and Astra Zeneca (Cambridge, United Kingdom).

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One Size Fits All for Stress-dose Steroids

To the Editor:

Liu *et al.* provide a thorough review of perioperative steroid replacement and make evidence-based recommendations to help clear up the "confusing" recommendations about who needs "stress-dose" steroids, what agent to administer, and how much to administer.[1] They report that there is limited evidence that such supplementation is necessary, but continue on to provide an algorithm for how much hydrocortisone to give at-risk patients based on anticipated surgical stress. They also point out that mineralocorticoid deficiency does not occur in secondary adrenal insufficiency (*i.e.*, due to chronic exogenous steroid administration). They also indicate that administration of hydrocortisone can result in excess mineralocorticoid activity with resulting (and undesirable) fluid retention and hypokalemia.

The lack of evidence, clinical confusion, and adverse effects of hydrocortisone seem to beg for a simpler solution. As it happens, there is one: dexamethasone 4 (or 8) mg. The 30+ fold glucocorticoid potency compared with hydrocortisone, absence of mineralocorticoid activity, and longer half life seem to make it a superior agent for perioperative supplementation for any level of stress. Unlike the limited evidence of need for stress-dose steroids, or for an antiemetic effect of hydrocortisone, the evidence of efficacy and safety of dexamethasone for prevention of postoperative nausea/vomiting (PONV) is extensive.[2,3] Since most of our patients have one or more risk factors for PONV, administering dexamethasone is usually indicated even without a question of adrenal insufficiency. Therefore, administering a PONV prophylaxis dose of dexamethasone seems like a simple, one-size-fits-all algorithm for dealing with any concern about secondary adrenal insufficiency.

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