of the lack of randomized clinical trials demonstrating any improvement of clinical outcomes including mortality from perioperative inotropic therapy,3–5 we are restraint to ignore or explain away the underlying signal our data raise.

Competing Interests
The authors declare no competing interests.

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Lung Ultrasonography for the Detection of Anesthesia-induced Lung Atelectasis

To the Editor:
We have read with great interest the article by Acosta et al.1 exploring the use of lung ultrasound as a mean to detect intraoperative atelectasis. However, despite their statement to the contrary, the occurrence of B lines in the setting of atelectasis has already been described by others. Although B lines were initially thought to originate from the interaction of the ultrasound beam with thickened subpleural interlobular septa found in alveolar-interstitial pathologies,2 recent work has challenged this hypothesis. In an elegant series of experiments, Soldati et al.3–5 have shown that B lines are observed when the ultrasound beam interacts at the pleural surface with lung tissue of a specific density. Although this can occur with the replacement of subpleural air by an ultrasound-conductive substance (e.g., water, pus, blood, and fibrous tissue), the withdrawal of air (e.g., resorption atelectasis) will also lead to the genesis of B lines. Demonstrating this last point, in an ex vivo animal model of graded atelectasis, B lines were observed in increasing numbers with increasing atelectasis. Pathologic examination of the excised lungs showed diffusely compressed alveoli mixed with sporadic areas of normally expanded distal air spaces.5

The study by Acosta et al. comes at an interesting moment. The recent publication of two randomized controlled trials6,7 exploring the impact of intraoperative mechanical ventilation parameters on postoperative pulmonary complications has generated much interest.8 Although some have linked the negative results of the PROVHILO (PROtective Ventilation using High versus Low positive end-expiratory pressure) trial to a lack of regular recruitment maneuvers and a positive end-expiratory pressure set too high,9 others blame the use of inappropriately high tidal volumes in the control group for the positive results in the IMPROVE (Intraoperative PROtective VEntilation) trial.10 In the absence of imagery supporting claims of atelectasis or overdistention, the culprits usually blamed for the development of postoperative pulmonary complications, it is unlikely this question will be resolved before more data becomes available. Interestingly, recent anesthesiology literature has demonstrated the advantage of hemodynamic optimization11 championing the concept that individualization is preferable to a “one size fits all” approach. Likewise, individualization of mechanical ventilation parameters might be an interesting avenue to explore if we wish to decrease the occurrence of postoperative respiratory complications. This hypothesis is supported by spiral computed tomography studies reporting significant interpatient variability in the amount of atelectasis induced by general anesthesia.12,13 Therefore, we believe that bedside monitoring to detect lung atelectasis or overdistention is needed. Although magnetic resonance imaging and computed tomography fulfill this requirement, they cannot be used in an intraoperative setting except in specially designed operating rooms and could not realistically be repeated throughout a procedure. Because lung ultrasonography can be performed at the bedside and is devoid of any ionizing radiation, the present study by Acosta et al., although interesting in and of itself, is an important milestone toward establishing lung ultrasonography as a tool to optimize intraoperative mechanical ventilation parameters. Other investigators have described loss of aeration scales that have allowed the study of the therapeutic effects of antibiotics in ventilator-associated pneumonia,14 the effect of different levels of positive end-expiratory pressure in patients with acute respiratory distress syndrome on lung reexpansion15 and the detection of patients likely to fail extubation after a successful spontaneous breathing trial.16 Although not developed specifically for the diagnosis and monitoring of anesthesia-induced atelectasis, the use of these scales would have been an interesting addition to the study by Acosta et al. Whether to optimize intraoperative mechanical ventilation parameters or to assist anesthesiologists in the care of hypoxemic patients,
lungen sonography is likely to have a bright future in our operating rooms.

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Competing Interests
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9. In Reply: Thank you very much for giving us the opportunity to reply the letter by Dr. Girard et al. about our recent paper in Anesthesiology.1 In their interesting letter, Dr. Girard et al. describe different theories about the genesis of B lines in the setting of atelectasis and commented that this very lung sonograph (LUS) sign has already been described. Their argument is based on publications related to patients with pre-existing pulmonary diseases and on data derived from *ex vivo* animal and laboratory models.2–5 As practicing anesthesiologists who simply apply LUS as a diagnostic tool, we focused our literature search primarily on clinical studies employing LUS and thereby have missed important evidence coming from primary ultrasound research, however, we do not only agree with the criticism but are grateful to the authors for having raised our awareness for the complexity of LUS.

To our knowledge, the occurrence of anesthesia-induced atelectasis in children has never before been studied by LUS in detail. For this reason, we cannot infer with certainty that the LUS signs—including B lines—found in adults and in atelectasis of different origins are similar to or even identical with the ones we saw in anesthesia-induced atelectasis by compressive mechanism in our children. This lack of reliable information made us define anesthesia-induced atelectasis a *posteriori* and analyze the prevalence of LUS signs associated with such atelectasis (please see table 1). This is the reason why we presented our results—including those related to B lines—as novel contributions to the clinical understanding and diagnosis of atelectasis in children undergoing general anesthesia.

In the second part of their letter, Dr. Girard et al. highlight the role anesthesia-induced atelectasis may play in creating local inflammatory responses within the lungs and in causing postoperative pulmonary complications.6 Such lung inflammation appears any time cyclic ventilation is applied to a partially collapsed lung, the root cause being *tidal recruitment* (the opening and closing of an atelectatic area during the breathing cycle) and *tidal overdistension* (the excess volume or pressure that normally aerated areas


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