in each group: analgesic pathway 39 (26%) *versus* 32 (22%) in placebo.[2] In a robust double-blind randomized trial (n = 299), there is no reason to expect substantive differences between treatment and placebo groups.

Meier *et al.* write that "measuring the Quality of Recovery score 3 days after surgery, as well as other postsurgical pain measures, is confounded when both the experimental and control groups received multimodal analgesics during and after surgery." Confounding—by definition—is restricted to factors that influence *both* exposure and outcome. Randomization usually prevents confounding; but in any case, an intervention *after* a blinded exposure cannot be a confounder. What Meier *et al.* presumably mean is that postrandomization treatments might influence outcomes. We agree, but the fact that patients in each group consumed nearly the same amounts of various analgesics during the initial postoperative days is not a limitation; instead, it confirms that the four combined treatments we tested are ineffective.

A reasonable question is whether Quality of Recovery 3 days after surgery is a suitable primary outcome. Granted, 3 days is distal to the interventions which were largely intraoperative. But Quality of Recovery is a well-validated, patient-centered outcome[3] focused on assessing patient pain and comfort level. Proponents of enhanced recovery or multimodal analgesic pathways presumably believe that their interventions noticeably improve outcomes that patients can detect. Clearly, the four drugs we tested did not. In any case, our predefined secondary outcomes were proximal, namely pain scores and opioid consumption, and neither improved.

Postoperative analgesia certainly matters, and presumably some approaches are better than others. We look forward to trials comparing various approaches, but evaluating postoperative analgesic technique was not our goal. We can, though, conclude that immediate perioperative use of acetaminophen, gabapentin, lidocaine, and ketamine—all of which act *via* separate pathways—contributes little. Future trials might therefore concentrate more on postoperative treatments or different analgesic regimens for perioperative analgesia.

Our results, while robustly equivocal, are nonetheless valuable since all drugs impose risk and cost and should therefore only be used if they are actually effective in a given context. We stand by our conclusion that "an analgesic pathway based on preoperative acetaminophen and gabapentin, combined with intraoperative infusions of lidocaine and ketamine, does not improve recovery in patients recovering from multilevel spine surgery."

Competing Interests

The authors declare no competing interests.

Kamal Maheshwari, M.D., M.P.H., Daniel I. Sessler, M.D.
Cleveland Clinic, Cleveland, Ohio (K.M.). maheshk@ccf.org

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Lung Ultrasound Training in the Critically Ill: Comment

To the Editor:

We read with interest the excellent study by Arbelot *et al.*,[1] evaluating the learning curve for focused, diagnostic lung ultrasound. The authors should be commended for their heroic effort to conduct a multicenter educational study at 10 intensive care units spanning three continents to address an important question that will inform future training guidelines. But although the study's results are broadly consistent with those of other related publications on this topic,[2,3] we take issue with one aspect of the study's methodology: the authors' unique classification of lung ultrasound pathology. Specifically, the authors asked learners to assign each lung ultrasound exam a score on a five-point scale "according to the worst parenchymal pattern" visible in the exam. The scores ranged from 1 for "normal aeration" to 5 for "lung consolidation." Although the authors' definitions for scores 1 and 5 conform to widely accepted norms,[4] their definitions for scores 2, 3, and 4 contain some irregularities.

The authors define these intermediate points as follows: 2 = "interstitial-alveolar syndrome"; 3 = "interstitial syndrome"; 4 = "pulmonary edema." For these three states, both the authors' numerical ordering and their proposed definitions are problematic for several reasons. First, in the lung ultrasound literature, the terms "interstitial-alveolar syndrome" and "interstitial syndrome" are often used

interchangeably to refer to the same condition: a state of pathologically increased lung density short of complete lung consolidation.[4,5] Second, the authors' own definitions for these states seem to overlap. For instance, the authors define "interstitial-alveolar syndrome" as "multiple B lines either spaced or coalescent" and "interstitial syndrome" as "more than two spaced B lines or coalescent B lines, detected in a limited portion of the intercostal space and issued from the pleural line or subpleural consolidations of at least 5 mm." The only way for these definitions to not overlap is if the authors intended learners to think of "interstitial-alveolar syndrome" as having two or fewer B lines per interspace. However, such a definition would be inconsistent with the widely accepted definition of this term: the presence of three or more B lines per intercostal space.[5] Third, if one did wish to use "interstitial-alveolar syndrome" (state 2) and "interstitial syndrome" (state 3) to distinguish between two conceptual conditions, then the terms would need to be flipped in the author's spectrum of severity because interstitial edema precedes alveolar edema clinically.[6] Fourth, in their definition of states 3 and 4, the authors present a description of B lines that contradicts accepted norms: Whereas the authors imply that a B line could emanate from a subpleural consolidation, the literature defines B lines as ring-down artifacts that originate only from the pleural line.[4] In contrast, ring-down artifacts that originate from subpleural consolidations are termed "shred sign" and identify lung that has higher density than edema: consolidated lung.[6] Fifth, the term "pulmonary edema" (state 4) is classically a part of the differential diagnosis of interstitial/interstitial-alveolar syndrome, rather than its own standalone category of lung ultrasound pathology severity.[4] Notably, in addition to the manuscript, we have also reviewed the authors' supplemental digital content: Although the supplemental content is generally excellent, it does not clarify any of the peculiarities described above.

The authors should be praised for conducting a study that will inform multiple specialties' training guidelines. But precisely because the study is likely to be so influential, we think it is important to identify any seeming methodologic flaws.

Competing Interests

Dr. Bronshteyn has performed paid consulting for Teleflex/Arrow (Wayne, Pennsylvania) in 2020 unrelated to diagnostic ultrasound. The remaining authors declare no competing interests.

Yuriy S. Bronshteyn, M.D., F.A.S.E., William C. Fox, M.D., Nazish Hashmi, M.D., Vijay Krishnamoorthy, M.D., Ph.D. Duke University School of Medicine, Duke University Medical Center, Durham VA Health Care System, Durham, North Carolina (Y.S.B.). Yuriy.bronshteyn@gmail.com

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Lung Ultrasound Training in the Critically Ill: Reply

In Reply:

In their letter, Bronshteyn *et al.*[1] pinpoint a critical issue concerning lung ultrasound training[2]: the identification and interpretation of vertical artifacts, the so-called B lines.

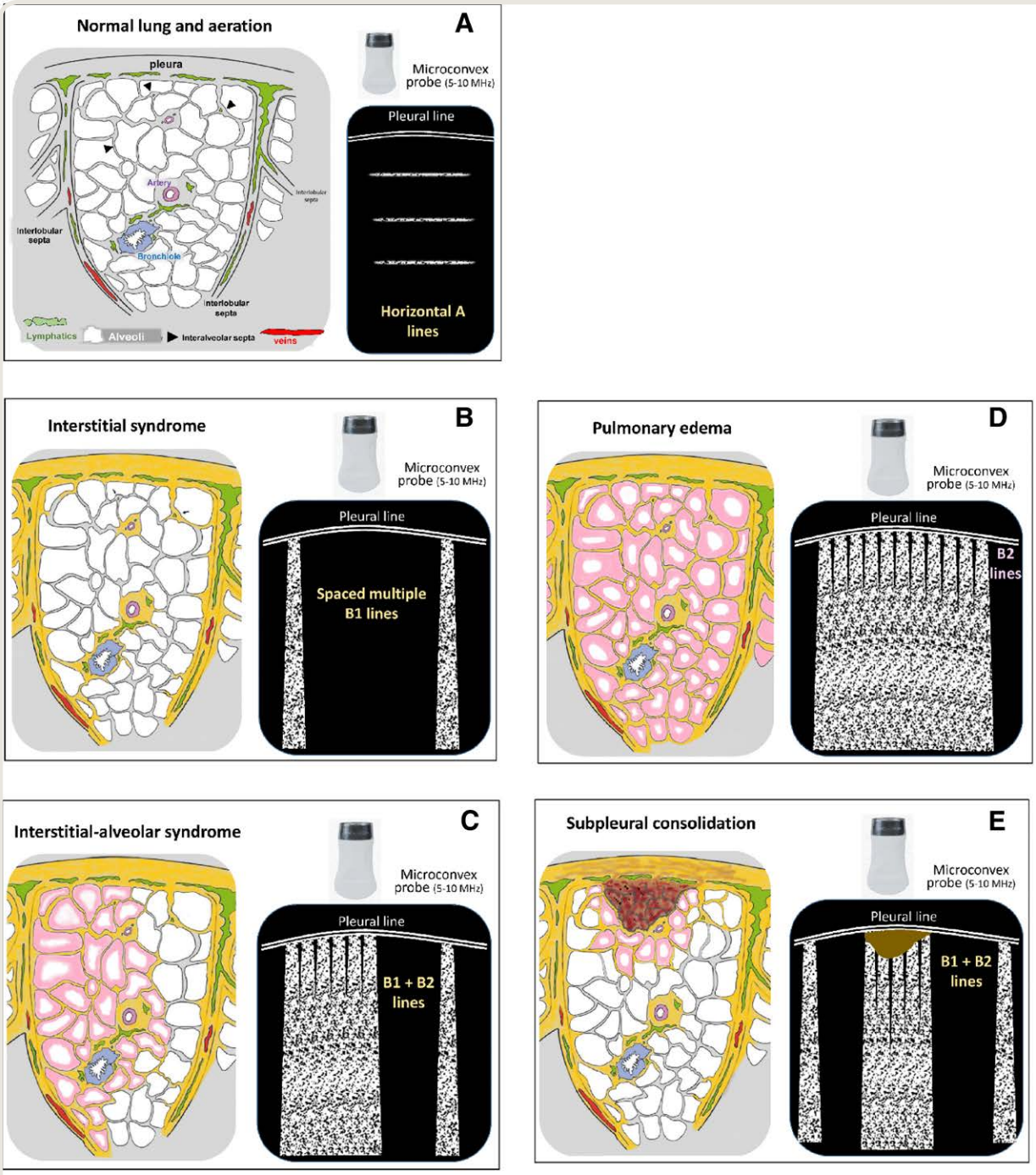

Fig. 1. Simplified representation of ultrasound patterns involving B lines. (*A*) Diagram of a normal secondary pulmonary lobule delineated by interlobular septa and centered by the pulmonary vascular axis. A lines, which are artifactual repetition of the pleural line, characterize normal aeration. (*B*) Interstitial syndrome is characterized by the accumulation of edema within interlobular septa, pleura, and bronchovascular axis (*yellow color*). The abnormal interface between alveolar gas and septal edema results in multiple spaced B1 lines. (*C*) In interstitial-alveolar edema, edema breaks into the alveolar space in some regions (*pink color*). The abnormal interface between alveolar edema and gas result in limited coalescent B2 lines. In other regions with interstitial edema, multiple spaced B1 lines are observed. B1 and B2 lines coexist. (*D*) In acute pulmonary edema, edema is extensively present in alveolar spaces (*pink color*). The abnormal interface between alveolar edema and gas, result in extended coalescent B2 lines. (*E*) Subpleural consolidation (foci of interstitial pneumonia) are surrounded by partially aerated alveoli. Ultrasounds are transmitted through the consolidation (tissue structure), and the abnormal interface between alveolar edema and gas in surrounding alveoli result in limited coalescent B2 lines. In other regions with interstitial edema, multiple spaced B1 lines are observed. B1 and B2 lines coexist.

Vertical artifacts have been indifferently termed comet tail artifacts, ultrasound lung comets, ring-down artifacts, and B lines.[3] Multiple internal reverberations issued from subpleural airspaces surrounded by edematous tissue is the main biophysic mechanism producing B lines.[4] In experimental models,[4,5] the generation of B lines requires two conditions: (1) The existence of acoustic traps combining transonic access channels and air bubbles. The multiple reflections between bubbles reradiate the incident wave to the probe, with a wavelength that depends on transonic access channels' shape and size. (2) The acoustic trap should have a minimal size under which it cannot emit a B line. The ratio between the wavelength emitted by the acoustic trap and the probe, determines B lines spatial characteristics: When the ratio is greater than 1, B1 spaced lines are generated; when the ratio is less than 1, B2 coalescent B lines are emitted.[4] Basically, the detection of B lines is indicative of an abnormal interface between alveolar gas and pulmonary tissue extending to the lung periphery.

The applicability of experimental models to clinical situations is illustrated in figure 1. When the lung is normally aerated (fig. 1A), there is no available acoustic trap because the normal interlobular septa is not thick enough to transmit the incident wave and induce reflections between airspaces. Only horizontal A lines are present (slide 3, http://links.lww.com/ALN/C148). Edematous or fibrotic interlobular septa characterizing interstitial syndrome open transonic access channels and generate multiple spaced B1 lines (fig. 1B). Because the size of the secondary pulmonary lobule varies from 10 to 30 mm,[1] multiple B1 lines may be regularly or irregularly spaced (slides 9 and 10, http://links.lww.com/ALN/C148). Ultrasound interstitial syndrome is defined as the presence of more than two spaced B1 lines, and not "as the presence of more than two spaced B lines or coalescent B lines, detected in a limited portion of the intercostal space and issued from the pleural line or subpleural consolidations of at least 5 mm," as falsely stated in the Method section (typographical error attributable to an automatic copy and paste).[2] We thank Dr. Bronshteyn *et al.* for their careful reading of our article and agree that our ultrasound definitions were confusing because of this typographical error. Interstitial-alveolar syndrome (fig. 1C) is characterized by the coexistence of acini with interstitial edema and acini with pulmonary edema. Alveolar flooding increases the number and the size of transonic access channels and creates multiple interconnected small air bubbles (fig. 1D), two conditions resulting in coalescent B2 lines. Ultrasound interstitial-alveolar syndrome is defined as the simultaneous presence of B1 and B2 lines in adjacent lung regions (slide 4, http://links.lww.com/ALN/C149). An analog situation is created when alveolar flooding is on the border of a subpleural consolidation representing a foci of interstitial pneumonia (fig. 1E). According to the size of the subpleural infectious foci, which determines the size of the transonic access channel, either B1 or B2 lines can be detected. Therefore, B1 and B2 lines characterizing interstitial-alveolar syndrome can be issued either from the pleural line or subpleural consolidations (slide 11, http://links.lww.com/ALN/C148, and slide 3, http://links.lww.com/ALN/C149). Pulmonary edema, which is characterized by alveolar flooding involving all lung regions, creates conditions that generate diffuse coalescent B2 lines (fig. 1D). In hemodynamic pulmonary edema, B2 lines are issued from the pleural line (slide 5, http://links.lww.com/ALN/C149). In high-permeability type pulmonary edema, as observed in severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) severe pneumonia,[6] B2 lines can also be issued from subpleural consolidations (slide 6, http://links.lww.com/ALN/C149). Ultrasound pulmonary edema can be defined as the presence of coalescent B2 lines issued from pleural lines or juxtapleural consolidations, in all examined regions.

The ability to identify and correctly interpret B lines is a major, and difficult, part of the lung ultrasound training. We hope that our answer clarifies this complex issue. We thank Dr. Bronshteyn *et al.* for identifying a mistake related to a typographical error in the ultrasound definition of Interstitial syndrome.

Competing Interests

The authors declare no competing interests.

Jean-Jacques Rouby, M.D., Ph.D., Felippe Leopoldo Dexheimer Neto, M.D., Mao Zhang, M.D., Ph.D., Luiz Marcello Sà Malbouisson M.D., Ph.D., Jie Lv, M.D., and Charlotte Arbelot, M.D. Multidisciplinary Intensive Care Unit, Department of Anesthesiology and Critical Care, La Pitié-Salpêtrière Hospital, Sorbonne University of Paris, Sorbonne University, Paris, France (J.-J.R.). jjrouby@invivo.edu

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Getting to a New Normal: Mandating That Patients Wear Masks as Hospitals Fully Reopen during the Coronavirus Pandemic: Comment

To the Editor:

Liu and Fleisher recommend that both patients and healthcare providers wear facemasks to prevent the spread of COVID-19 as a new normal in hospitals.[1] We agree, and recommend adding risk stratification of those involved with airway management to mitigate further any transmission or adverse effects of COVID-19 on vulnerable healthcare providers.

Liu and Fleisher noted that the close proximity between patients and healthcare providers combined with the highly transmittable SARS CoV-2 virus places healthcare providers at a high risk of infection, especially those healthcare providers involved in airway management. El-Boghdadly *et al.* reported that 10% of healthcare providers either tested positive for SARS CoV-2 or were self-isolated due to COVID-19 symptoms within 30 days of performing their first tracheal intubation on a COVID-19 patient.[2] To identify healthcare providers most at risk, we used Centers for Disease Control guidance.[3]

Procedurally, we request SARS CoV-2 testing for patients having elective surgery. For any patients arriving without testing, we determine which are at high risk for being SARS CoV-2–positive. These patients include those with cough, fever, or contact with a COVID-19–positive person, or those coming from nursing homes or prisons. Then we exclude the highest-risk healthcare providers from airway management of these patients or those known to be SARS CoV-2–positive.

We calculate healthcare providers risk using a point scale: 4 points for age >70 yr, immunocompromised, or pregnant; 2 points each for age 60 to 70 yr, diabetes, or medical conditions involving the heart, lungs, and kidneys; and 1 point for age 50 to 60 yr or primary caregiver for a family member at risk or a child under 6 months old. Eleven percent of 149 anesthesia healthcare providers returned surveys of 4 or greater points. Healthcare providers not returning surveys are treated as having scores of 0.

We collected these data through a voluntary survey of all anesthesia healthcare providers. Department members self-identified their risks, with no documentation required. Faculty anesthesiologists returned their surveys to the department chair, residents to the program director and nurse anesthetists to their chief. These department leaders relayed the summary results to the charge anesthesiologists, who use the results to make daily work assignments. We handle data that healthcare providers report as private information, not available outside this small departmental group. Healthcare providers with a summative score of 4 or greater have received no clinical assignments involving airway management of high-risk patients.

This combination of wearing face masks, testing patients, and risk-stratified assignments of healthcare providers has led to no known transmission of COVID-19 within the institution. We plan to study the effects of these mitigation practices over time on worker morale, practice efficiency, and disease prevention, and modify the program as needed. We may also modify our risk calculation scale as more information about COVID-19 accumulates.

Competing Interests

Dr. Johnstone's son-in-law is CEO of Lab Corps, which does COVID-19 testing. He and Lab Corps are not associated with West Virginia University.

Hong Wang, M.D., Ph.D., James G. Cain, M.D., M.B.A., Adam Uraco, B.S., Eric Lindstrom, C.R.N.A., Robert E. Johnstone, M.D. West Virginia University, Morgantown, West Virginia (R.E.J.). JohnstoneR@wvumedicine.org

This letter was sent to the author of the original article referenced above, who declined to respond.—Evan D. Kharasch, M.D., Ph.D., Editor-in-Chief.

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