needed to optimize anticoagulant selection and dosing to prevent VTE severe sepsis and septic shock.

The timing of VTE development in sepsis also intrigues us, and we appreciate this insightful comment. Cook et al[5] performed serial ultrasound examinations on 817 critically ill patients (5.4% of whom had sepsis) and found that the median time to VTE diagnosis was 8 days (interquartile range, 4-14 days). Since only a small minority of patients in this cohort had sepsis, however, additional prospective studies are needed to determine the timing of VTE in septic patients. This information would potentially lead to earlier VTE diagnosis and treatment, and more favorable clinical outcomes. Meanwhile, providers should maintain a low threshold for evaluating the possibility of VTE in sepsis.

In our opinion, whether VTE is an unavoidable consequence is not yet known. Whereas thromboprophylaxis reduces VTE in hospitalized medically ill and surgical patients, current guideline-supported VTE prevention strategies may be less effective among critically ill patients who have sepsis. Higher-intensity anticoagulation (perhaps regardless of BMI) combining thromboprophylaxis with mechanical devices or using recently approved anticoagulants may reduce VTE risk but requires further investigation.

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**DOI:** http://dx.doi.org/10.1016/j.chest.2016.01.016

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# Posterior Consolidation

## An Important Clue for Differentiating ARDS From Cardiogenic Pulmonary Edema

**To the Editor:**

I read with interest the study by Sekiguchi et al[1] in a recent issue of *CHEST* (October 2015) that focused on combined cardiac and lung ultrasound to evaluate the etiology of acute hypoxic respiratory failure. The authors reported good diagnostic utility in differentiating ARDS from cardiogenic pulmonary edema (CPE) by evaluating the amount of left pleural effusion, the severity of left ventricular dysfunction, and the diameter of the inferior vena cava. In the Supplemental Materials section of the online article, the authors describe that both C (consolidative)-pattern and pleural effusion were assessed in examination point 5 (posterolateral zone of the lung), but only data regarding pleural effusion were reported in e-Table 1. Whether posterior consolidation exists may be important in differentiating ARDS from CPE. Copetti et al[2] identified consolidation by using lung ultrasound in 83% of patients with ARDS but in no patients with CPE. In contrast, posterior consolidation and/or atelectasis have been known to be a classic finding in ARDS, documented by using CT scan results.[3] In a study evaluating lung ultrasound for the assessment of lung recruitment in patients with ARDS, Bouhemad et al[4] also reported that consolidation was the predominant ultrasound pattern in the lower part of the posterior thoracic region. Could Sekiguchi et al[1] provide the detailed information about the ultrasound pattern in examination point 5, especially the consolidation pattern?

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**DOI:** http://dx.doi.org/10.1016/j.chest.2015.10.083

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# Response

**To the Editor:**

We appreciate the insightful comments about our article[1] made by Dr Keng. The information on consolidative (C)-pattern in examination point 5 was missing in e-Table 1. The revised e-Table 1 is attached to this response letter. We defined C-pattern as areas of tissue hepatization with or without air bronchogram.[1] C-pattern in zone R5 was seen in 36 patients (61%) in the group with cardiogenic pulmonary edema (CPE), 23 (55%) in the group with ARDS, and 12 (38%) in the miscellaneous group ($P = .093$). C-pattern in zone L5 was seen in 44 patients (75%) in the group with CPE group, 22 (52%) in the group with ARDS, and 13 (41%) in the miscellaneous group ($P = .004$). These variables were not selected as statistically significant in a prediction model for CPE in the multivariate analysis.

C-pattern reflects a variety of causes such as atelectasis (eg, resorptive, compressive, and passive), consolidation, contusion, infection, mass, and pulmonary embolism.[2] It is often difficult to identify a cause of C-pattern with critical care ultrasonography alone.[3] We anticipated that C-pattern in the form of compressive atelectasis was associated with pleural effusion in the posterolateral zone. In an effort to differentiate effusion-induced compressive atelectasis from consolidation or other causes of C-pattern, we created a variable, C-pattern without significant pleural effusion (< 20 mm), in zone 5. C-pattern without significant pleural effusion in zone R5 was seen in 14 patients (24%) in the group with CPE, 16 (38%) in the group with ARDS, and 10 (31%) in the miscellaneous group ($P = .304$). C-pattern without significant pleural effusion in zone L5 was seen in 16 patients (27%) in the group with CPE, 12 (29%) in the group with ARDS, and 10 (31%) in the miscellaneous group ($P = .94$). These two variables were included in the multivariate analysis; however, they were not statistically significant for differentiating CPE from ARDS.

C-pattern was not an important variable for differentiating CPE from ARDS in patients with acute hypoxemic respiratory failure. Although variables on C-pattern were accidentally missing in the original e-Table 1, they were included in the initial multivariate analysis. Therefore, the study conclusions are the same as demonstrated in our original article: left-sided pleural effusion (> 20 mm), moderately or severely decreased left ventricular function, and a large inferior vena cava minimal diameter (> 23 mm) were predictive of CPE. Our results are consistent with previous reports on CT, which demonstrated that atelectasis and consolidation are seen in both patients with CPE and those with ARDS.[4,5]

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**DOI:** http://dx.doi.org/10.1016/j.chest.2016.01.022

## Acknowledgments

**Additional information:** The e-Table can be found in the Supplemental Materials section of the online article.

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# Radiologic Criteria for the Diagnosis of High-Attenuation Mucus in Allergic Bronchopulmonary Aspergillosis

**To the Editor:**

We read with interest the article by Alikhan et al[1] in a recent issue of *CHEST* (December 2015). The