the ICD *in situ* and an expanded lung. However, a grade 2 leak with a larger defect in the visceral pleura may benefit from letting the lung collapse to reduce the size of the defect. If such a lung is allowed to remain collapsed for a short period (a period that needs to be defined by further research but might be about 24–48 h), the pleural defect might have a better chance of healing, as proposed by Walker and colleagues (1). So, we propose that the ICD be left in place but disconnected from the underwater seal for this period (Figure 1). A bacterial filter may be placed at the end of the tube to prevent infection. If there is any fluid draining, a bag can be connected to the ICD but without the water seal. Although such a method would allow the lung to remain collapsed, it will ensure that a tension pneumothorax does not develop as the pleural cavity is open to the atmosphere. The ICD can be reconnected to an underwater seal after the potential healing period and checked for an air leak again, and the cycle can be repeated till the air leak ceases (Figure 1). ■

Author disclosures are available with the text of this letter at www.atsjournals.org.

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Reply to Albert and Dhooria *et al.*

From the Authors:

The recent research in spontaneous pneumothorax has stimulated unprecedented interest in the subject, as emphasized in our editorial (1). Healthy discussions (like the review by Walker and colleagues [2] and our editorial [1]) and insightful comments, such as those from Dr. Albert and Dr. Dhooria and colleagues, are welcomed, add to the momentum, and generate novel hypotheses to be explored.

The suggestion by Dr. Albert to consider factors affecting interstitial pressure in the pathophysiology and treatment of pneumothorax is on the basis of the idea that air may leak from the lung into the interstitial space, enter the mediastinum and subsequently appear in the pleural cavity. This idea has also been cited in high-profile reviews (3).

Direct and indirect evidence in animal models suggests that interstitial pressure of healthy lungs (approximately −10 to −12 cmH_2O at functional residual capacity) is considerably lower than pleural pressure because of the powerful lymphatic pump. As such, there certainly appears to be a higher pressure gradient for air to leak from alveoli to interstitial spaces than to the pleural space (4, 5). However, elastance of the extracellular matrix in the interstitium is high because of the mechanical resistance of proteoglycans, so interstitial pressure rises rapidly to above atmospheric pressure with fluid loading or hypoxia (6, 7). This may limit the capacity of the interstitial space to act as a conduit for air between the alveoli and mediastinum. We are unaware of comparable data in humans.

We also note that in patients with spontaneous pneumothorax, residual air in the mediastinum (of any volume) is exceedingly rare, even on computed tomography scans. These observations suggest that air leak from alveoli to interstitial spaces is unlikely to be a common cause of spontaneous pneumothorax.

We also thank Dhooria and colleagues for their proposed algorithm. We support clinical algorithms that minimize unnecessary chest tube insertions. We advocate the findings of the PSP (primary spontaneous pneumothorax) randomized trial (8), which convincingly showed that most (85%) patients with PSP do not require aspiration or drainage, on the proviso that the pneumothorax does not enlarge on a repeat radiograph after 4 hours and vital signs are stable. Insertion of a chest tube significantly increased the risk of prolonged air leak, time in hospital, need for surgery, and serious adverse events compared with the patients managed conservatively. The trial included patients with moderate to large pneumothorax (median 64% of hemithorax), and we apply this regularly in our practice, even to patients with complete pneumothorax (9). It is important to note that most patients with PSP are much more troubled by pain than breathlessness.

An interval chest radiograph is a useful alternative to determine if the air leak is ongoing without interventions. Simple aspiration has been shown to be at least as effective as chest tube insertion (10). Another recent randomized trial (11) showed that an ambulatory device (incorporating an 8F catheter attached to a one-way Heimlich valve and fluid collection chamber) is useful for community management of patients who may have an ongoing air leak without connecting to an underwater seal bottle. Hence conventional chest tube insertion and underwater seal bottle management should only be necessary for a minority of patients. ■

Originally Published in Press as DOI: 10.1164/rccm.202207-1450LE on September 7, 2022

Author disclosures are available with the text of this letter at www.atsjournals.org.

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Reply to Albert and Dhooria *et al.*

From the Authors:

We read with interest letters from Dr. Albert and Dr. Dhooria and colleagues in response to our article (1). We are pleased that our discussion piece has generated such interest, as this was the intention. We hypothesized and described a potential mechanism for chest tube insertion prolonging air leaks.

Albert accurately describes the mechanism for the formation of pneumomediastinum as described by Macklin and Macklin (2). However, we challenge the presumption that this is the mechanism for the generation of spontaneous pneumothoraces in general on two counts.

First, if their presumption of mechanism were true, we would expect evidence of pneumomediastinum in every spontaneous pneumothorax. Numerous randomized trials and case series of spontaneous pneumothorax management that include the review of chest radiographs and (increasingly) computed tomography scans, fail to report any pneumomediastinum (which one presumes would be a significant enough finding to warrant mention). We believe it is unlikely that a significant air leak via the mediastinum, sufficient to cause pneumothorax, would leave no detectable residual air in the mediastinum. Macklin describes the potential of "malignant pneumomediastinum," with intramediastinal pressures high enough to cause significant (and sometimes fatal) sequelae, to cause pneumothorax by mediastinal–pleural rupture rather than as the *de facto* mechanism for all spontaneous pneumothoraces (2).

Second, this theory ignores the evidence of abnormalities at the visceral pleural surface (even in primary spontaneous pneumothorax [PSP]) that are often categorized as occurring in the absence of demonstrable lung disease. There is good evidence that the lungs of patients with PSP are not normal (3). A total of 50–80% of patients with PSP have blebs on computed tomography scanning (4–6), and this is higher than in the nonpneumothorax population. Blebs are an outpouching (or vesicle) of the visceral pleura caused by air in the interstitium, forming between the lamina elastica interna and externa of the pulmonary pleura. Historically, it was postulated that it was the rupture of blebs, causing leakage of air from the alveoli to the pleural space that created a pneumothorax. Indeed, Macklin describes this as a common mechanism distinct from pneumomediastinum (2).
We do agree that visible air leak from blebs is not routinely observed and, in fact, many blebs remain intact when the lung is inspected at the time of surgery. In some cases, no macroscopic lesions are seen at all (7). However, rather than presuming the air is entering the pleural space via the mediastinum, we believe that these findings support the

Supported by the National Institute for Health Research Academic Clinical Lectureship (R.J.H) and National Institute for Health Research Oxford Biomedical Research Centre (N.M.R).

Originally Published in Press as DOI: 10.1164/rccm.202209-1642LE on September 7, 2022