The Answer May Lie in Avoiding the Underwater Seal Rather Than the Chest Drainage Tube

To the Editor:

We read with interest the thought-provoking perspective article by Walker and colleagues and the critical editorial by Lee and Singh (1, 2). Walker and colleagues argued that the pressure gradients generated by the presence of an intercostal drainage tube (ICD) disallow the rapid healing of the underlying visceral pleural defect in patients with pneumothoraces. Furthermore, they proposed that the absence of an ICD lets the lung be in a collapsed state, which helps keep the defect in the visceral pleura small, thus allowing natural healing to occur. Consequently, conservative management without an ICD might lessen the incidence of a persistent air leak. Contrastingly, Lee and Singh expressed the practical concern that a life-threatening tension pneumothorax might develop in the absence of an ICD. Continuing this discussion, we present another perspective on the basis of physiological concepts, limiting our proposal currently to primary spontaneous pneumothorax (PSP), which is defined as the presence of air in the pleural space that occurs spontaneously (without any antecedent intervention or trauma, etc.) in patients without clinically apparent underlying lung disease.

When a patient presents to a health facility with symptoms suggesting a PSP, a finite amount of time has already passed since the index event of the creation of a visceral pleural defect (T0). When the PSP is diagnosed using radiological tests, an ICD may be inserted, according to the existing guidelines, at time point T1 (3). Two different events could have occurred between T0 and T1: the pleural defect causing the pneumothorax is either closed because of natural healing or still open. When the ICD is connected to an underwater seal, the underwater placement acts as a barrier to air entry into the pleural space through the drainage tube had it been left open to the air. The negative pleural pressure during each inspiration results in lung expansion, while it also pulls the water column up the drainage bag to a level numerically equal to the negative pleural pressure. During expiration, the positive pleural pressure drives the accumulated air out through the drainage tube. If healing of the visceral pleural defect has occurred between T0 and T1, there might be a quick expulsion of air from the pleural cavity through the ICD with successive respiratory cycles over a few minutes to hours. At the end of this variable period, bubbling in the ICD bag would cease. In the other scenario, in which the visceral pleural defect is persistent, air will repeatedly accumulate in the pleural cavity with each inspiration, only to be expelled through the ICD either with each expiration (when the leak is grade 2 according to Cerfolio classification) or during an inadvertent forced expiratory maneuver such as coughing, after every few respiratory cycles (if the leak is grade 1 by Cerfolio classification) (4). A grade 1 leak might cease in a short period with

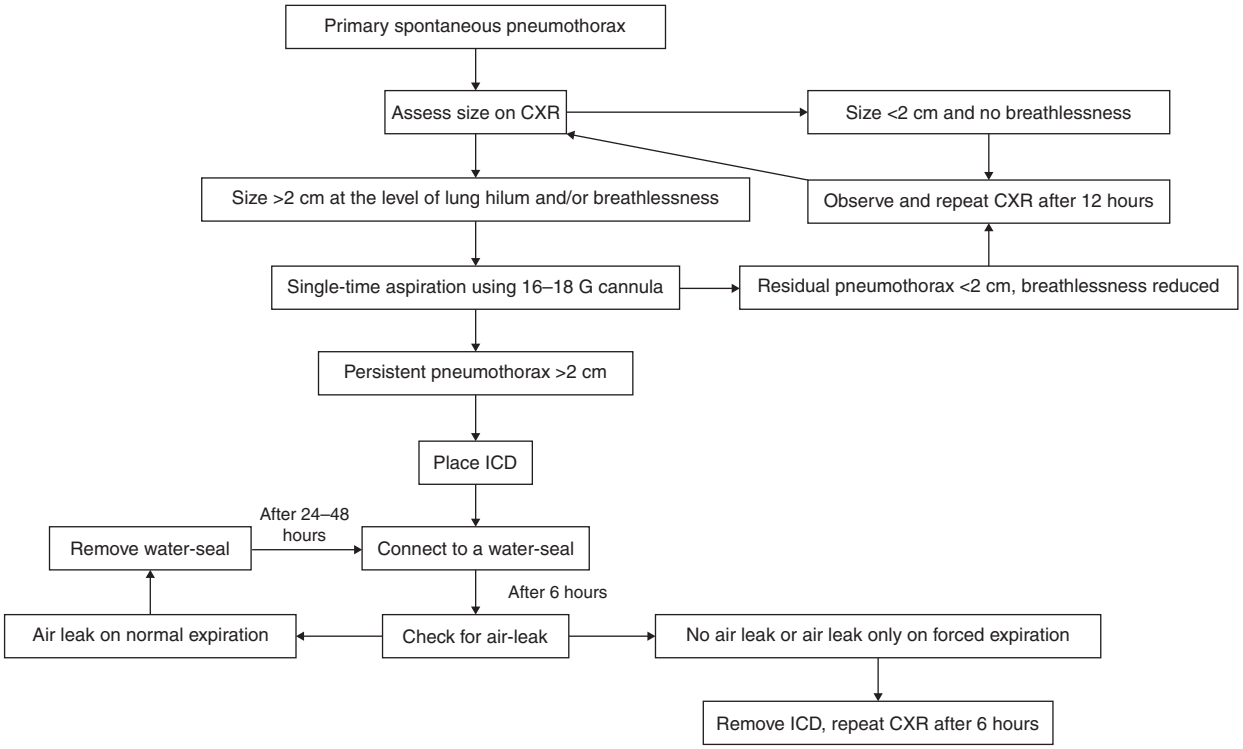

Figure 1. An algorithmic approach to primary spontaneous pneumothorax. CXR = chest radiography; ICD = intercostal drainage tube.

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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. ■

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