On behalf of the medXcloud group

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Pneumothorax: Challenging the Paradigm of Persistent Air Leak, but Where Is the Leak?

To the Editor:

Walker and colleagues (1) suggest that chest tube drainage of pneumothoraces may increase air leaks and that chest tube drainage decreases pleural pressure, thereby increasing the pressure gradient between the site of the leak in the parenchyma and the pleural space. Their conclusion is on the basis of the premise that the air is leaking across the visceral pleura. Although they cite one paper that identified transpleural air leaks in eight patients who had multiple bullous lesions and large air leaks (2), and certainly direct transpleural air leaks can occur as a result of stab wounds or rib fractures, Lee and colleagues (3) note in the accompanying editorial that "most patients do not have an obvious site of leak … even when surgeons submerge the lung underwater intraoperatively."

In 1944, Macklin and Macklin proposed that pneumothoraces occur when the base of an overdistended alveolus ruptures, resulting in air escaping into the perivascular interstitium and subsequently dissecting along the interstitium toward the mediastinum, after which it can track into the pleural space (bilaterally on occasion) as well as into the soft tissue of the neck, face, arms, chest wall, pericardium, or into the abdominal cavity (4). If this were the mechanism by which air enters the pleural space (which Macklin and Macklin considered to be far more likely than transpleural leakage), the pressure gradient of interest would be from the alveolus to the interstitium and factors affecting interstitial pressure (e.g., lung volume, surface tension, lung elasticity, and airway and vascular diameters) would need to be included when considering the pathophysiology and treatment of pneumothoraces.

Macklin and Macklin (4) also noted that the pulmonary vessels dilated and elongated in response to lung inflation (predating the description of the effects of inflation on extra-alveolar vessels by Howell and colleagues in 1961 [5] and the concept of parenchymal interdependence described by Mead and colleagues in 1970 [6]). Accordingly, expanding the lung by evacuating pleural air would reduce interstitial pressure and augment the alveolar-to-interstitial gradient, similar to what Walker and colleagues propose for the alveolar–pleural gradient. Expanding the lung will also induce or increase alveolar overdistension (which occurs most commonly in response to local or regional atelectasis or inhomogeneities in airway resistance) that initially resulted in the septal rupture. ■

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

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