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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

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theory of "pleural porosity" (i.e., that pneumothorax occurs when air leaks from an area of thinned visceral pleura, rather than rupture of a bleb) (8, 9). The exact pathophysiological mechanism for the generation of the abnormally thinned area is not clear, but there is evidence that chronic peripheral lung inflammation is present. Histopathological analysis of surgical specimens from patients with pneumothorax demonstrates subpleural fibrosis, accumulation of macrophages, and proliferation of mesothelial cells at the lung surface (10, 11), and data suggest abnormal expression of inflammatory proteases (matrix metalloproteinases) in the lung in PSP (12–15).

Dhooria and colleagues describe the dilemma of managing spontaneous pneumothorax: How does one know at first presentation if the initial air leak has resolved (and one is now managing the residual, nonexpanding pneumothorax) or if there is an ongoing air leak? The holy grail in addressing this issue in PSP would be a noninvasive test to assess for ongoing air leak (without the need for insertion of a tube in the chest). In the absence of such a test, Dhooria and colleagues have devised a pathway of management that includes a trial of the management of patients with a spontaneous pneumothorax with a chest tube *in situ* without underwater seal for a nominal period of 24 to 48 hours. Although the rationale for such a pathway should be applauded, there are practical and physiological flaws with this approach. The initial management steps of PSP seem overly cautious with a reliance on size measurement. The Australian conservative management study has shown that the size of pneumothorax is not a barrier to conservative management. We believe that the severity of symptoms should be the main decision criteria for intervention, and this principle is likely to be reflected in upcoming guidelines. Also, the suggestion of observing all patients for 12 hours, thereby requiring admission to the hospital for this period, does not seem necessary. Tension pneumothorax for PSP is thankfully rare (although admittedly, data are very limited). We do not believe there is justification for leaving the chest drain "open" to allow for the remote possibility of a tension pneumothorax while the patient is in the hospital. A significant proportion of physicians advocate a trial of "clamping" of the chest tube before removal that by the same reasoning risks the development of tension pneumothorax but is easily ameliorated by "unclamping" the chest tube by medical staff in response to physiological change.

Physiologically, the pleural space is at subatmospheric pressure of -0.5 to -0.8 kPa because of the elastic recoil of the lung, but it can be up to -8 kPa on forced maximal inspiration. The purpose of the underwater seal is to avoid the ingress of air by these negative pressures. The proposal of leaving a chest tube "open" could result in two problems. First, equalizing this negative pleural pressure to atmospheric pressure would remove the beneficial effect of the evacuation of air once the air leak has ceased. Second, inadvertent rapid inspiration (e.g., if the patient is in pain) and associated greater negative pressure could result in a significant indrawing of air, further collapsing the lung in the absence of an ongoing air leak because of the elastic recoil of the lung.

We encourage discussion and further research into spontaneous pneumothorax, a common and understudied area of respiratory physiology. ■

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