A 69-year-old man presented with pneumogenic sepsis. A chest radiograph showed bilateral extensive infiltrates that did not respond to broad-spectrum antibiotic treatment. Progressive respiratory failure necessitated invasive mechanical ventilation (bilevel positive airway pressure, 0.5 inspiratory oxygen saturation; peak inspiratory pressure, 24 cm H₂O; positive end-expiratory pressure, 8 cm H₂O). Chest computed tomography (CT) revealed inflammatory infiltrates with caverns and bilateral pleural effusions. Blood and bronchoalveolar cultures remained sterile. Therefore, computed tomography-guided lung biopsy was performed with an 18-gauge core needle with the patient in prone position (Figure, A). A control computed tomodography scan (Figure, B) revealed massive systemic air embolism. Shortly thereafter, the patient developed cardiorespiratory failure. Cardiopulmonary resuscitation remained unsuccessful.

Percutaneous computed tomography-guided lung biopsy is a frequently performed procedure for histological and microbiological analyses of pulmonary lesions and infiltrates. Moderate complication rates have been described, including pneumothorax (27%), intraparenchymal hemorrhage (11%), or hemoptysis (7%).

Systemic air embolism is an extremely rare and feared complication that is potentially fatal, reported previously to have an incidence of ≈0.02% to 0.7%. However, it can be assumed that there is a much higher incidence of systemic air embolism because of clinically unapparent cases (0.4% to 3.8%).

Systemic arterial air embolism is caused by the entry of gas into the pulmonary veins or directly into the arteries of the systemic circulation. A common mechanism is that a biopsy needle opens a pulmonary vein to the atmosphere. Direct formation of fistulae between pulmonary veins and alveoli or bronchi is another possible mechanism.

Any factor that increases pressure gradient between airway and pulmonary vein may promote the entrance of air into the pulmonary vein and cause air embolism: positive end-expiratory pressure ventilation, coughing during lung biopsy (Valsalva), or obstructive pulmonary disease (chronic obstructive pulmonary disease) with air trapping may increase airway pressure and thus the risk of air embolism. Prone position, which implies a puncture site at a level above the heart, is also considered to increase pressure gradient between airway and pulmonary vein and therefore is a risk factor. Lung diseases with cavities or cystic lesions or larger needle size with the needle tip placed not properly in the lesion may be associated with an increased risk for air embolism. The coaxial technique is often discussed as a risk factor for systemic air embolisms, but there are a number of cases described without the use of the coaxial technique.

If available, hyperbaric oxygen and Trendelenburg positioning is considered to be the first-line therapy for systemic air embolism and to prevent cerebral edema in cases of air embolism in cerebral arteries.

In the presented case, we believe that positive pressure ventilation was the main reason for this fatal air embolism, with prone position being an additional risk factor. Furthermore, the chronic inflammatory process of the lung that had been present in our patient for several weeks is an additional risk factor that interferes with hemostatic mechanisms that promote vessel occlusion and directly inhibits vessel contraction. In contrast to the massive intravascular air, no air was visible in the biopsy area or in the pleura postinterventional. Most likely, the true cut biopsy formed a direct fistula between a pulmonary vein and a small bronchus or alveolus that did not seal because of the chronic inflammatory lung changes. It is less likely that the room air entered the pulmonary vein via the coaxial needle because the coaxial needle was directly closed by the thumb of the interventionalist after removal of the biopsy needle. The large needle size (18 gauge), the long true cut biopsy sample (2 cm), and the number of biopsy samples (3) aggravated tissue damage and enhanced the risk of perforating a pulmonary vein. The puncture path plays a subordinate role in causing systemic air embolisms and is mainly important to prevent a pneumothorax. It is reported that a smaller amount of parenchyma along the needle path decreases the risk of an air embolism. In the reported case, we chose a short needle path to the infiltrates. No pneumothorax occurred.

In summary, lung biopsies with positive end-expiratory pressure and biopsies in prone position should be avoided. It needs to be determined whether a temporary reduction of positive end-expiratory pressure (eg, for 1 hour) is feasible in patients undergoing aggressive mechanical ventilation attributable to pulmonary failure. Several variables could potentially be adjusted as compensatory measures: (1) oxygen delivery; inspiratory oxygen saturation, duration of inspiration, hemoglobin concentration, dose of inotropic agents, optimization of gas exchange by preinterventional bronchoalveolar lavage, or recruitment maneuvers; and (2) oxygen consumption.
treatment of pyrexia, discrete hypothermia, respiratory quotient (nutrition), sedation/relaxation, or β-blockade.

The interventionalist should be aware that chronic inflammatory lung changes are a risk factor for developing systemic air embolism attributable to the inhibition of vessel contraction and interference with hemostatic system. Therefore, the indication for biopsy of chronic infiltrates should not be indicated without diligence.

**Disclosures**

None.

**References**


**Figure.** A, Computed tomography-guided lung biopsy of infiltrates was performed with an 18-gauge needle and the patient in prone position. B, Air embolism after lung biopsy involving intercostal arteries (arrow), coronary arteries (arrowheads), and the aorta (white asterisk).
Massive Air Embolism After Lung Biopsy
Mareike Franke, Hans Christian Reinhardt, Michael von Bergwelt-Bailsdon and Christopher Bangard

_Circulation_. 2014;129:1046-1047
doi: 10.1161/CIRCULATIONAHA.113.004241
_Circulation_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2014 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/129/9/1046

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in _Circulation_ can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to _Circulation_ is online at:
http://circ.ahajournals.org//subscriptions/