Published in final edited form as:

Crit Care Med. 2013 April ; 41(4): 1133–1134. doi:10.1097/CCM.0b013e31827c02cb.

Pleural Effusion in Acute Respiratory Distress Syndrome: Water, Water, Everywhere, Nor Any Drop to Drain*

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Keywords

acute lung injury; acute respiratory distress syndrome; computed tomography; lung imaging;
pleural effusion; respiratory physiology

On March 30, 1981, President Reagan received lifesaving chest tubes to drain blood collecting in his left pleural space caused by a gunshot wound. This historic public example of the threat posed by accumulation of pleural fluid seems to make the benefits of removing it obvious, but does the same apply to the pleural effusions frequently encountered in patients undergoing mechanical ventilation as a result of pneumonia, sepsis, and other acute illnesses? In some cases, effusion volumes can be large enough to create respiratory and hemodynamic compromise and warrant thoracentesis. To date, however, there is little information on the physiological effects of pleural effusion during acute respiratory distress syndrome (ARDS), so whether such effusions should be drained is currently unclear. In this issue of *Critical Care Medicine*, Ciumello et al (1). present evidence suggesting that the physiological benefits of draining a pleural effusion in ARDS are often minimal, concluding that “Pleural effusion in acute lung injury or ARDS patients is of modest entity ... without affecting gas exchange or respiratory mechanics”

Drawing on clinical and CT data from a sizeable cohort of 129 patients, the authors show that approximately 80% of ARDS patients present with fluid around the lungs (pleural effusion) in addition to fluid in the airspaces (alveolar edema) and within the lung parenchyma (interstitial edema). Elimination of fluid from all three locations would seem to be an obvious treatment goal and in the case of an effusion can be readily achieved via drainage through a chest tube. Nevertheless, placement of a chest tube is not without risk, so its use must be commensurate with the magnitude of the physiological impairment produced by the effusion.

*See also p. 935.

The physiological effects of pleural fluid are interesting, complex, and at times counterintuitive (2, 3). It might seem inevitable, for example, that an effusion would compress the lungs in proportion to the volume of accumulated fluid. In fact, fluid in the pleural space serves to mechanically decouple the lungs and the chest wall, allowing their respective volumes to move in opposite directions. From the perspective of lung function in the ventilated patient, however, it is only the degree of lung recoil that matters, and this may be substantially less than the outward recoil of the chest wall because of the relatively smaller elastance of the latter. This has been demonstrated in experimental studies in both head-up and supine normal dogs in which lung volume was reported to decrease a third of the effusion volume, the other two-thirds of the volume being accounted for by an increase in chest wall volume (4, 5). A similar proportion was found in ARDS patients in the present study by Chiumello et al (1); the chest wall accommodated 2.4 times more effusion volume than the lungs, in line with the measured chest wall elastance of approximately 35% of lung elastance.

Nevertheless, whether to drain a pleural effusion in the ARDS patient still requires careful consideration. For example, the relative roles of lung elastic recoil, hydrostatic pressure, and chest wall recoil on overall respiratory function may vary substantially at different degrees of lung inflation (2), so the physiological significance of a pleural effusion is likely to depend markedly on positive end-expiratory pressure and tidal volume. Also, pleural effusions will become more important in situations where chest wall elastance is increased, such as in the presence of increased abdominal pressures (6) or in obesity. The patient population studied by Chiumello et al had compliant chest walls and poorly recruitable lungs, so there was little improvement in oxygenation and respiratory mechanics with increased positive end-expiratory pressure irrespective of the volume of the effusion fluid. By contrast, a previous study of ARDS of predominantly extra-pulmonary origin showed thoracentesis to produce significant improvements in oxygenation (7). The reasons for this discrepancy are not clear but may relate to the heterogeneity of the ARDS population. Even in the present study, the patients in the higher effusion group were older, with stiffer lungs, and had 6% more consolidated lung tissue at baseline than the patients in the low effusion group, which may explain why respiratory mechanics parameters were similar between the two groups even though minute ventilation in the bilateral effusion group was 20% smaller for equivalent P_{aCO_2} . In any case, drawing general conclusions about how to manage ARDS from a single study may be difficult.

Another important potential consequence of pleural effusion is that loss of air volume, at least in normal lungs, occurs predominately in dependent regions due to alveolar collapse (2, 5). Ventilating such lungs would be expected to cause repeated recruitment of these regions, which could predispose them to ventilator-induced lung injury unless sufficient positive endexpiratory pressure is used (2). This may not have applied to the nonrecruitable consolidated lungs of the ARDS patients studied by Chiumello et al, but there may be other groups of ARDS patients who would be at risk for atelectrauma.

Finally, even though the purely pulmonary effects of pleural effusion in ARDS might be minimal, important consequences could still arise from increases in chest wall volume. De Troyer et al (8) showed that pleural effusions drain to the dependent subdiaphragmatic

regions in the supine position. This changes the shape of the diaphragm in a way that can lower its mechanical efficiency by placing the muscle at an unfavorable location on its force–length relationship. This could adversely affect, for example, the ability of the patient to wean from mechanical ventilation.

Despite these caveats, however, Ciumello et al teach us that the majority of ARDS patients have pleural effusion volumes lower than those found to cause significant physiologic effects in experimental studies and that only about 30% of the effusion volume acts to compress the lungs with the remainder being accommodated via expansion of the chest wall. Draining a pleural effusion in ARDS is thus not automatically indicated.

Acknowledgments

Supported, in part, by a grant from the National Institutes of Health (R01 research grant).

REFERENCES

1. Chiumello D, Marino A, Cressoni M, et al.: Pleural Effusion in Patients With Acute Lung Injury: A CT Scan Study. *Crit Care Med* 2013; 41:935–944 [PubMed: 23385101]
2. Graf J, Formenti P, Santos A, et al.: Pleural effusion complicates monitoring of respiratory mechanics. *Crit Care Med* 2011; 39:2294–2299 [PubMed: 21666452]
3. Mitrouska I, Klimathianaki M, Siafakas NM: Effects of pleural effusion on respiratory function. *Can Respir J* 2004; 11:499–503 [PubMed: 15505703]
4. Krell WS, Rodarte JR: Effects of acute pleural effusion on respiratory system mechanics in dogs. *J Appl Physiol* 1985; 59:1458–1463 [PubMed: 4066576]
5. Dechman G, Mishima M, Bates JH: Assessment of acute pleural effusion in dogs by computed tomography. *J Appl Physiol* 1994; 76:1993–1998 [PubMed: 8063661]
6. Formenti P, Graf J, Cortes GA, et al.: Experimental intra-abdominal hypertension attenuates the benefit of positive end-expiratory pressure in ventilating effusion-compressed lungs. *Crit Care Med* 2012; 40:2176–2181 [PubMed: 22710205]
7. Talmor M, Hydo L, Gershenwald JG, et al.: Beneficial effects of chest tube drainage of pleural effusion in acute respiratory failure refractory to positive end-expiratory pressure ventilation. *Surgery* 1998; 123:137–143 [PubMed: 9481398]
8. De Troyer A, Leduc D, Cappello M, et al.: Mechanics of the canine diaphragm in pleural effusion. *J Appl Physiol* 2012; 113:785–790 [PubMed: 22797306]