against structural damage to the alveolar endothelial barrier. Elevated sweat sodium and chloride levels, which reflect impairment of systemic epithelial ion transport, were found to be associated with sepsis related pulmonary edema and its severity, as expressed by ventilation index and duration of mechanical ventilation,[4] and should be evaluated as a prognostic marker and risk factor for ARDS.

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REFERENCES

1. Villar J. What is the acute respiratory distress syndrome? Respir Care 2011;56(10): 1539-1545.
2. Aman J, Groeneveld J, Van Nieuw Amerongen GP. Predictors of pulmonary edema formation during fluid loading in the critically ill with presumed hypovolemia. Crit Care Med 2012;40(3):793-799.
3. Ware LB, Matthay MA. Alveolar fluid clearance is impaired in the majority of patients with acute lung injury and the acute respiratory distress syndrome. Am J Respir Crit Care Med 2001;163(6):1376-1383.
4. Eisenhut M, Wallace H, Barton P, Gaillard E, Newland P, Diver M, Southern KW. Pulmonary edema in meningococcal septicemia associated with reduced epithelial chloride transport. Pediatric Crit Care Med 2006;7(2):119-124.
5. Eisenhut M, Wallace H. Ion channels in inflammation. Pflugers Arch 2011;461(4): 401-421.

The author replies:

I thank Michael Eisenhut for his interest and comments about a recent symposium paper published in the Journal.[1] While I could not argue against a reduced pulmonary fluid clearance in the early phases of ARDS, disturbances in the endothelial and epithelial sides of the alveolar barrier, manifested by deterioration of the pulmonary function, usually represent the sum total effect of numerous cellular and molecular processes. The importance of endothelial injury to the formation of pulmonary edema in this disorder has been well established.[2] Simultaneous and/or sequential activation of signaling pathways, caused by different insults and stimuli in the pulmonary endothelium, interstitium, and epithelium, result in increased permeability to plasma solutes and water with flooding of the interstitial and alveolar space.

Since in some patients, pulmonary edema can resolve quickly, it is likely that those patients do not have the same characteristic pathological abnormalities as those who have diffuse alveolar damage, as could be the case in many patients from the series of patients reported by Aman et al.[3] As pointed out by Schuster, almost 20 years ago,[4] those patients should not be classified as ARDS but as some alternative form of non-cardiogenic pulmonary edema.

To document the permeability disorder in early ARDS, we measured simultaneously the total plasma protein concentration and the protein concentration in the bronchial aspirate of 22 ARDS patients within the first 12 hours of ARDS onset.[5] In all patients, the bronchial aspirate/plasma protein ratio was ≥ 0.6 (mean 0.75 ± 0.10, range 0.60-0.98) and there were no significant differences between the mean values in survivors (0.78 ± 0.13, $n = 8$) versus non-survivors (0.73 ± 0.09, $n = 14$). In all cases, pulmonary capillary wedge pressure was lower than 14 mm Hg during the study period (mean 9 ± 2 mm Hg). When the relationship between pulmonary capillary wedge pressure and the bronchial aspirate/plasma protein ratio was evaluated, a statistically significant inverse correlation between both parameters were found ($P = .025$).

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REFERENCES

1. Villar J. What is the acute respiratory distress syndrome? Respir Care 2011;56(10): 1539-1545.
2. Ware LB, Matthay MA. The acute respiratory distress syndrome. N Engl J Med 2000; 342(18):1334-1349.
3. Aman J, Groeneveld AB, van Nieuw Amerongen GP. Predictors of pulmonary edema formation during fluid loading in the critically ill with presumed hypovolemia. Crit Care Med 2012;40(3):793-799.
4. Schuster DP. What is acute lung injury? What is ARDS? Chest 1995;107(6):1721-1726.
5. Villar J, Blazquez M, Manzano JJ, Quintana J, Lubillo S. Value of the measurement of alveolar proteins in the adult respiratory distress syndrome. Med Intensiva 1989;13(6):274-277.

CORRECTION

In the paper "Possible prognostic value of leukotriene B_4 in acute respiratory distress syndrome" by Masclans JR, Sabater J, Sacanell J, Chacon P, Sabin P, Rosa O, Planas M. (Respir Care 2007 52[12]:1695–1700), the *complete* affiliation information for the authors should have stated: Intensive Care Unit, Area General, Hospital Universitari Vall d'Hebron de Barcelona, *Department of Medicine, Universitat Autònoma de Barcelona (UAB)*, Barcelona, Spain.