stated in their first paragraph) are the hydrostatic pressure and colloid osmotic pressure of the interstitial fluid outside the capillaries. The interstitial hydrostatic pressure is not known, but some physiologists think that it may be considerably less than alveolar pressure. The colloid osmotic pressure of the interstitial fluid is not known with certainty, but based on the value of pulmonary lymph in experimental animals, a reasonable value is approximately 20 mm Hg.

Far from there being a "gradient of approximately 17 mm Hg that opposes the egress of fluid into the interstitial space," it seems likely that, at the base of the lung at least, the net pressure is outward, and this causes a small normal flow of lymph. An important implication of this is that any rise in capillary hydrostatic pressure will increase the loss of fluid from the pulmonary capillaries, thus increasing the flow of lymph. Recent experimental evidence supports this. These newer notions on the pathophysiology of pulmonary edema have important implications. The lung does not normally have a large reserve of pressure opposing the movement of fluid out of the capillaries; but, rather, any pathologic state which, for example, increases capillary hydrostatic pressure will increase the amount of interstitial fluid in the lung. Presumably the reason why this was not appreciated years ago is that interstitial pulmonary edema is so difficult to recognize; however, the lung is far more vulnerable to factors promoting pulmonary edema than was previously thought.

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To the Editor:

We appreciate and respect the comments of West and especially those that relate to pulmonary microvascular pressures in the upright human subject. We could cite no greater authority than West himself with regard to the spectrum of hydrostatic pressures from the apex to the base of the lung; however, this is really not an issue, inasmuch as the pulmonary arterial microvascular pressure is estimated from the wedge pulmonary arterial pressure or the pulmonary diastolic pressure, which is measured in the supine subject and with arbitrary zero reference to a horizontal midthoracic plane. The very range of values for microvascular pressure that West cites (namely, 0 to 15 mm Hg) of itself corresponds to our estimate of an average pulmonary microvascular pressure of 8 mm Hg, which obtains both in the upright and the recumbent positions.

In holding that there is a normal "gradient" of approximately 17 mm Hg that opposes the egress of fluid into the interstitial space, we took special care to pinpoint the theoretic limitations of such a computation, which takes into account only two of the six known factors determining the flow of fluid across the capillary. In spite of these unknowns, the computation of this gradient has proven to be a remarkably good measurement of the risk of hemodynamic pulmonary edema. When the measured pulmonary arterial wedge pressure was subtracted from the colloid osmotic pressure in the recumbent subject and the difference was less than 4 mm Hg, pulmonary edema usually appeared. Indeed, the very incentive to our editorial1 was the concurrent publication in Chest of the confirmation by Rackow and his associates2 of this relationship which had previously been reported from our own laboratory.3,4

In our editorial, we stated that "the clearance of fluid from the interstitium by the lymphatic system constitutes an additional dynamic variable that ultimately determines sequestration of fluid into the lung"2,3,4,5 Accordingly, we did not fail to recognize the important implications of what we referred to as "the dynamic flux of fluid provided by lymphatic clearance of extravascular fluid from the lung."3,4,5,6

With regard to West's conclusion, we are in complete accord that the lung is far more vulnerable to factors promoting pulmonary edema than was previously thought; however, for the record, none of the theoretic considerations cited by West controvert the experimental observations commented upon in our editorial, namely, that a critical reduction in the difference between colloid osmotic and pulmonary arterial wedge pressures is associated with radiographic signs of pulmonary edema.

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