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COMMENTARY

THE MAJOR ROUTE FOR ABSORPTION OF FLUID FROM THE PLEURAL SPACE

K.I. Gourgoulianis, C.H. Hatzoglou, P.A. Molyvdas

Department of Respiratory Medicine (KIG), and Department of Physiology (CHH,PAM), University of Thessaly, Greece

From early in the 20th century, there were conflicting theories about the entry and exit of pleural liquid including that fluid was secreted by pleural mesothelial cells and entered from the parietal (high-pressure) pleura and was absorbed into the bloodstream via the visceral (low-pressure) pleura. During the last several decades, a consensus has developed that pleural liquid arises from blood vessels in both the parietal and visceral pleura, flows across the leaky pleural membranes into the pleural space and exits via the parietal lymphatics (1).

The arrangement of the lymph vascular system differs in the parietal compared with the visceral pleura. On the parietal pleura, the lymphatic plexus parallels the mesothelium and connects to the pleural cavity via stomas opening at the confluence among adjacent mesothelial cells. This lymphatic network is particularly well developed over the diaphragm where the stoma density is higher than on the other pleural surfaces. The diameter of these stomas range from 0.5 μm to 20 μm depending on tissue stress. Thus, during inspiration, descent of the diaphragm promotes a threefold increase of stoma surface without links to the pleural space (2).

Fluid that exits from the pleural space normally depends on parietal lymphatics. The rhythmic myogenic activity of the lymphatic walls (intrinsic mechanism), and the movement of soft tissue that surrounds the lymph channels (extrinsic mechanism), during muscle contraction move fluid out of the pleural cavity both in health and disease (3). Our recent observation that one out of four pregnant women during labor has measurable (at ultrasonography) pleural effusion reflects a relative lymphatic insufficiency (poor inspiration) as pleural fluid influx increases during the third trimester of pregnancy because of elevated capillary hydrostatic pressure and decreased plasma oncotic pressure. During labor, in addition to occlusion of diaphragmatic stomas, no recruitment of other collapsed stomas can occur. A hydrothorax at time of delivery, therefore, is an indirect marker for the important role of lymph propulsion in the absorption of pleural fluid.

A recent study of our group showed that amiloride increases the transepithelial resistance only of parietal pleura, although ouabain, an inhibitor of the Na^+-K^+ pump, and nitroprusside, a nitric oxide (NO) donor, exerts a similar effect on both the parietal and visceral pleura. This amiloride effect is more prominent in the basolateral and diaphragmatic than the apical pleural membrane (5). Amiloride is a drug that impairs smooth muscle contractility (6).

Negrinie et al (3) estimated that ~40% of total pleural lymphatic flow depends on the intrinsic mechanism. Our data are in agreement with that view because the amiloride effect occurs only at sites with stomas (parietal pleura, especially diaphragmatic) and differs from the effects on mesothelial cell channels induced by NO or ouabain. The actual increase of parietal pleura resistance is only from 20Ω/cm^2 to ~22Ω/cm^2, but this small increase, nonetheless, may retard pleural fluid absorption (5).

Despite previous conflicting theories, lymphatic drainage is now recognized as the major route of pleural fluid efflux. Both the clinical observation of benign peripartum pleural effusion and the electrophysiologic experiments in the parietal pleura document the pivotal role of lymphatic stoma activation in pleural dynamics.

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K.I. Gourgoulianis, M.D.
Associate Professor
Medical School, University of Thessaly
22 Papakyriazi
41222 Larissa, Greece
Telephone: +30 41 682439
Fax: +30 41 627853
E-mail: Kgourg@med.uth.gr