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Angiotensin II Impedes the ability of the lungs to clear edema

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Background and Aims: Pulmonary edema is a life threatening condition of various etiologies. Active alveolar fluid clearance (AFC) is important in keeping the airspace free of edema. AFC is mediated via the alveolar active sodium transport, a process by which Na+ is extruded out of the alveolar airspace by epithelial transport proteins, including apical Na+ channels and basolateral Na,K-ATPases with water following isosmotically. Angiotensins contribute to the pathogenesis of hypertension, arterial disease, cardiac hypertrophy and heart failure. However, little is known about the direct effects of Angiotensin II on alveolar epithelium and AFC. Thus, we aimed to investigate the physiologic role of Angiotensin II in alveolar fluid clearance in rats and conceivably the molecular basis of the angiotensin effects on AFC.
Results: The rate of AFC in control rats was 0.48±0.02 ml/h (all values are Mean ± SEM) and decreased by 36%, 48% and 60% in rats treated with 10-6 M, 10-7 M and 10-8 M (P = 0.003). The angiotensin receptor blocker (ARB), losartan, prevented the inhibitory effect of angiotensin effects and brought back AFC to baseline levels. The movement of large solutes across the alveolar-capillary barrier was not different among the study groups compared to control rats indicating that the alveolar-capillary barrier was not disrupted.
Conclusions: Angiotensin has a direct adverse effect on lung epthilium manifested by decreasing alveolar fluid clearance. This effect maybe reversed by ARB therapy.