2 ml. of physiologic saline solution containing 0.4–0.6 mc. P^{32} was instilled as rapid as possible through a small canule inserted down the right main bronchus of a dog under various experimental conditions. In some animals only blood samples were obtained through femoral artery, whereas both blood and lymph samples were drawn simultaneously from femoral artery and right lymphatic trunk in other animals. Absorptive capacity of pulmonary blood vessel or lymph vessel was obtained by measuring specific activity of blood or lymph samples.

We chose the ventilatory insufficiency as experimental conditions or loads out of the various pathogenetic factors of acute pulmonary edema above stated. They consisted of hypoxia, hypercapnea, hypoxia combined with hypercapnea, hypoxia with open thorax and increased airway resistance.

Both experimental groups on hypoxia alone with inhalation of about 10% O_2 and on hypercapnea alone showed a decreased absorptive capacity which turned back to control level on discontinuing the loads. Both groups on hypoxia (10% O_2) combined with hypercapnea and on hypoxia combined with open thorax showed marked decrease in absorptive capacity which failed to turn back on discontinuing the load in some animals.

The absorptive process through lymphatic system was so slow and limited that the amount was negligible compared with that of blood vessels. There is an intimate relation between absorption and transudation in and out of pulmonary vascular bed and it is conceivable that the decrease in absorption indicates the increased transudation. Therefore, it can be stated that the absorptive function of pulmonary blood and lymph vessel has only minor significance for the development of acute pulmonary edema.

Pulmonary Edema as a Cause of Surfactant Deficiency

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The induction of pulmonary edema in anesthetized dogs led to a patchy impairment of pulmonary surface activity, as measured by altered force-area relations of saline extracts. Surface activity was decreased whether pulmonary edema was induced by rapid infusion of dextran, or by injection of alloxan or ANTU. The altered surface properties were of sufficient magnitude and extent to result in abnormal pressure-volume relations of degassed lobes. Morphologically, areas of decreased surface activity were usually hemorrhagic but not foamy and had smaller or occluded alveoli.

Loss of surface-active lining in foam was an important factor in causing the deficiency of surface activity with pulmonary edema. This was shown by a less marked impairment of surface activity when foaming was prevented (by degassing a lobe before, and occluding its airway during induction of edema). The persistence of some degree of surface tension alteration in the absence of foaming suggested that the mere transudation of plasma into the alveolar spaces somehow interfered with surfactant.

Biochemical studies have thrown more light on deranged surfactant metabolism during pulmonary edema. When radiolabelled palmitate was injected intravenously in anesthetized dogs, it was rapidly incorporated by the lung, chiefly into phospholipids, primarily lecithin. With the induction of pulmonary edema, the count in lung tissue fell precipitously. Foam had a relatively high count distributed between phospholipids (part derived from alveolar cells) and neutral lipids (part derived from blood). Portions of edematous lung exhibiting hemorrhage and alveolar closure had a lower total phospholipid content than normal lung.

These findings have significant implications in terms of pathogenesis and therapy. The alterations in pulmonary function—arterial hypoxemia and, in advanced cases, CO₂ retention—are thus attributable not so much to accumulation of fluid in alveolar spaces as to premature alveolar closure. Mechanical respiratory assistance could reverse these functional changes to a large measure by forcing open the closed alveoli. The demonstration of impaired pulmonary surface activity due to pulmonary edema raises the possibility that a similar relationship exists in other experimental or clinical situations associated with both pulmonary edema and surfactant deficiency, e.g., oxygen toxicity, vagotomy, and the “pulmonary hypoperfusion syndrome.”

Action of Certain Diuretics in Pulmonary Edema ; An Experimental Study

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Experiments were conducted in 115 rabbits in order to ascertain whether the diuretics influence the course and severity of pulmonary edema. Pulmonary edema was induced by a standard dose of epinephrine P. and D. according to two different plans. In the first, the dose varied between 0.45 and 0.70 mg. according to weight. In the second, one-half of the above dose was given (Table I). Two diuretics were studied: 4-chloro-N (2-furymethyl)-5-sulfamoylanthranilic acid (*Lasix* or *Furosemide* Hoechst-Lloyd), and meralluride sodium (*Mercurhydrin* Lakeside). In a first series of experiments, all surviving animals were sacrificed after 60 min. in order to measure their lung/body index. It was noted that the diuretic employed (*Furosemide*) did not affect the lung/body index, while it modified the length of survival as follows: (a) moderate results was obtained when 80 mg. was given simultaneously with the epinephrine; (b) better results were obtained when 80 mg. i.v. and 80 mg. i.p. were given 30 min. before epinephrine; and the longest average survival was obtained when 1 hour interval separated the administration of the diuretic from that of epinephrine (Table II): instead of an average survival of 16.5 min. (control), there was an average survival of 31.3 min. (treated). In a second series of experiments, the surviving animals were not sacrificed, and the number of survivors was noted at various time intervals. There was a significant difference between the animals treated with *Furosemide* and those which only received epinephrine (controls). The results were proportionally greater when a longer interval elapsed between administration of *Furosemide* and administration of epinephrine (Table