Absorption of inhaled water in experimental pulmonary edema and embolism

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The rate of absorption of small quantities of intratracheally injected fluids in the normal rat has been reported previously (1). Since inhaled water is known to be absorbed via the pulmonary capillaries (2) it appeared to be of some interest to study this function in conditions affecting pulmonary capillary permeability or blood flow.

METHODS

One hundred rats weighing 190–310 g were used in these experiments. They were divided into ten groups (A–J), each consisting of ten animals.

Olive oil, 0.1 ml/100 g, was given intraperitoneally to group A. The animals were sacrificed 5 hr later and used as controls. Groups B–G were given 10 mg/100 g α-naphthyl-thiourea (ANTU) in a 1% suspension in olive oil intraperitoneally. Two and a half hours later groups C–G were anesthetized intraperitoneally with 40 mg/kg thiopentone, tracheotomized, and intubated. Subsequently 0.1 ml/100 g heparinized blood, fresh water, physiological saline, or sea water was injected intratracheally, as described previously (1). Three hours following the administration of ANTU the animals were sacrificed. The time relationships of these interventions are shown in the protocol.

The jugular vein was exposed in three further groups of anesthetized, tracheotomized, and intubated animals (H, I, J); 0.1 ml/100 g of a starch suspension (2.5 g starch in 10 ml physiological saline) was then injected intravenously in 10–15 sec.

Group H was sacrificed 10 min later. Five minutes following embolization, 0.1 ml/100 g fresh water (group I) or sea water (group J) was administered intratracheally to the others. Group I was sacrificed 5 min, group J 20 min subsequently.

The lungs of the sacrificed animal were removed and weighed as described previously (1). Lung weight was expressed in per cent of body weight ("relative lung weight").

RESULTS

Mean relative lung weight of the control group was 0.53 (group A, Fig. 1); that of the ANTU-treated but otherwise intact rats amounted to 0.92 (group B, Fig. 1). The difference between these two groups was significant (P < .001).

Mean relative lung weight 5 min following the intra-
FIG. 1. Relative lung weight in rats subjected to various procedures. Each point represents 1 animal; numbers indicate mean values for each group. Group A: 1 ml/100 g olive oil i.p. Group B: 10 mg/100 g ANTU i.p. Groups C-G: 10 mg/100 g ANTU i.p., followed by intratracheal administration of 1 ml/100 g of different fluids. Group C: heparinized blood; Group D: fresh water; Group E: physiological saline, sacrificed 5 min later; Group F: physiological saline, sacrificed 15 min later; Group G: sea water, sacrificed 20 min later. Where not stated, animals were sacrificed 5 min following fluid inhalation.

FIG. 2. Relative lung weight in rats subjected to pulmonary embolism. Each point represents 1 animal; numbers indicate mean values for each group. Group H: pulmonary embolism. Group I: pulmonary embolism followed by intratracheal administration of 1 ml/100 g fresh water. Group J: pulmonary embolism followed by intratracheal administration of 1 ml/100 g sea water.

DISCUSSION

The administration of ANTU causes lung edema by increasing the permeability of the alveolo-capillary membrane (3). The extent of the edema is a function of dose and time: it reaches its maximum within 2-4 hr and is still present 10-12 hr later (4, 5). It was this predictable behavior and long steady state which induced us to choose this particular type of pulmonary edema for our studies.

The ANTU-treated animals were sacrificed 3 hr following the administration of the drug. At this stage lung edema had either reached its height or was at the end of its formational phase. There appeared to be a fair chance at this stage to observe any interference with the absorption of inhaled fluid.

Changes in mean relative lung weight were taken as an index of the fluid content of the lung. Lung weight in ANTU-treated rats was elevated. Because of its high protein content the absorption of inhaled blood takes many hours, even in the normal lung. Blood was ad-
ministered intratracheally in order to decide the accuracy of relative lung weight in revealing the presence of a small amount of added fluid in the edematous lung. The increase in mean lung weight after blood inhalation corresponded to the amount of blood administered. On the other hand, lung weight remained unchanged following the inhalation of fresh water, indicating a complete absorption of the added water. By the same criteria, the absorption of saline was complete only after 15 min, suggesting a slower rate of disappearance. Sea water inhalation caused an increase in mean relative lung weight equivalent to approximately three times the amount actually inhaled. This is consistent with the fact that sea water is about three and a half times more concentrated than plasma. All these changes are virtually identical with those seen in normal rats (1) and appear to suggest that ANTU-induced pulmonary edema does not materially interfere with the absorption of inhaled water.

The composition of the extracellular fluid in the lung of ANTU-treated animals is identical to that of the plasma. Since pulmonary capillary blood flow is not grossly altered at this stage (6) any decrease in the total osmotic pressure (crystalloids + proteins) of the edema fluid can be rapidly corrected by the absorption of the extra water via the pulmonary capillaries.

In contrast to these findings are those in the embolized rats. The rise in mean relative lung weight following the inhalation of fresh water is suggestive of an interference with the absorption of fluid from the lung. It is not clear why mean lung weight increased more than the actual amount of fluid inhaled.

Lung weight 90 min following sea water inhalation in embolized rats was increased. This implies that at this stage the flux of filtered fluid toward the alveoli was no longer impeded, and is consistent with the observation that lung circulation gradually improves with time, following pulmonary embolism.

It was a privilege and support.

REFERENCES