EFFECT OF HYPERTHERMIA AND HYPOTHERMIA ON DEVELOPMENT AND COURSE OF ACUTE PULMONARY EDEMA IN ANIMALS ANESTHETIZED WITH ETHER

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Intraperitoneal injection of 6% ammonium chloride solution (40 mg/100 g body weight) into rats led to the development of acute pulmonary edema and death of the animal. Hyperthermia of the rats (rectal temperature 39-41°) intensified, while ether anesthesia and hypothermia (rectal temperature 20-23°) prevented development of pulmonary edema.

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Although considerable attention has been paid to the study of pulmonary edema [1-5], many aspects of this highly important problem have not yet been finally solved.

We have studied the effect of high and low temperatures in animals anesthetized with ether on the development of toxic pulmonary edema.

EXPERIMENTAL METHOD AND RESULTS

Pulmonary edema was produced by intraperitoneal injection of 6% ammonium chloride solution in a dose of 40 mg/100 g body weight.

TABLE 1. Effect of Ether Anesthesia of Development of Pulmonary Edema during Hyper- and Hypothermia

Series of experiments	Experimental conditions	Total number of ani-	Bodytem- perature (in deg)	Animals dying			Animals surviving (sacri - ficed)		
				No. of ani- mals	Pulmonary coefficient	Duration of survival (in min)	No. of ani- mals	Pulmonary coefficient	Duration of survival
-	Normal external en- vironment	46	36-38	1	1.30 ± 0.048	20.5 ± 0.852	•		
	Hyperthermia Hypothermia Light ether	60 60*	39-41 23-20		1.99 ± 1.012	12.3 ± 1.048	45	0.81 ± 0.008	2 h 30 min
	anesthesia Ether anesthesia for	24	36-3 8		1.03 ± 0.025	40.5 ± 2.8	6	0.72	1 h 30 min
	1 h Hyperthermia against background of ether	24	36-3 8	2	1.20	26	22	0.76 ± 0.01	2 h
	anesthesia Hypothermia against	24	39.8-41	15	1.003 ± 0.028	58.06 ± 3.5	9	0.89	2 h
	background of ether anesthesia	24†	23-19	2	0.71	1 .	12	0.75 ± 0.03	2 h

^{*15} animals which remained for observation were indistinguishable from healthy rats after 5-6 h.

^{†10} animals remaining for observation were indistinguishable from healthy rats after 24 h.

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Experiments were performed on 262 albino rats in which the body temperature was measured by an electrothermometer. The degree of pulmonary edema was determined by microscopic investigation of the lungs and calculation of the pulmonary coefficient (PC). The experimental results are given in Table 1. The development of pulmonary edema under normal external environmental conditions (experiments of series I) was studied in 46 rats. Injection of ammonium chloride into intact rats led to the development of pulmonary edema and to death of all the animals. In the 60 rats in series II pulmonary edema was produced after hyperthermia had been produced in the animals in an incubator at 40-50° for 15 min. Hyperthermia intensified the pulmonary edema in all the animals. In 60 animals pulmonary edemas was produced in a state of hypothermia (cooling of the body in an external temperature of -17°) with a rectal temperature of 20-23° (series III). Cooling prevented the development of pulmonary edema in all the animals. In series IV, pulmonary edema was produced in a state of light ether anesthesia. Pulmonary edema developed in 18 of the 24 animals. In series V pulmonary edema was produced in a state of deep ether anesthesia, maintained for 1 h. Pulmonary edema developed in only two of the 24 animals after waking. In series VI, animals deeply anesthetized with ether were placed for 15 min in an incubator at 40-50°, after which pulmonary edema was produced. Of the 24 animals, 15 died, but pulmonary edema could not be found in any of them microscopically, and the increase in pulmonary coefficient was evidently associated with severe congestion of the lung tissue with blood. In series VII pulmonary edema was produced after deep ether anesthesia had fallen to 23-19°. Hypothermia against a background of ether anesthesia prevented the development of edema in 22 of the 24 animals.

Hence, hypothermia against a background of light ether anesthesia and deep ether anesthesia lasting 1 h prevented the development of pulmonary edema. Hyperthermia, both when acting alone and in conjunction with ether anesthesia, had an unfavorable effect on the development of the acute pulmonary edema.

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