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A Comparison of Survival to Decompression in Air and in Oxygen.

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It is generally assumed that death due to ascension to high altitudes or simulated high altitudes by decompression is due to anoxia entirely. Since human alveolar air is approximately saturated with water vapor the pH_2O at body temperature (37°) is about 47 mm Hg. The alveolar pCO_2 is ordinarily assumed to be about 40 mm Hg. Therefore the minimal pressure of 87 mm would be required before any oxygen would be available for respiration.[1,2]

The amount of water and carbon dioxide dilution of alveolar air would be influenced by the rate of ventilation of the lungs and in small animals such as rats and mice hyperventilation would have a significant effect on pCO_2 and pH_2O. Hailman[3] has shown that hyperventilation raises the altitude at which respiration fails in rats. He suggests that this is due to the reduction of pCO_2 and pH_2O by hyperventilation and from a possible fall in body temperature as a result of anoxia.

Many factors are known to influence survival to anoxic anoxia besides the pO_2, including the age of the animal,[4] rate of ascent (or decompression).[5,6] Armstrong and Heim[5] reported that higher altitudes can be reached before death if a rapid ascent is made. In our experiments (unpublished) we have found a greater tolerance to hypoxia in mice decompressed slowly as compared to those decompressed rapidly unless the decompression is extremely rapid ("explosive decompression".)

In this investigation white mice of approximately the same age and of about 17 g average body weight (males and females) and baby chicks of the same age (all males) were used in a decompression chamber (bell jar) which communicated with a large ballast jar which in turn was connected to a vacuum pump. The pressure inside the animal chamber was carefully controlled by a Hg manometer. The rate of decompression was regulated by an adjustable inlet valve admitting air or oxygen to the animal chamber. After various trials with different rates of decompression both continuous and discontinuous it was decided to standardize the technic to a rate of decompression shown in Table I, A from a starting pressure of 740 mm down to the critical pres-

TABLE I.

A. Rate of decompression in oxygen and in air:

Time in min:	0	3	6	9	12
Barom. press. mm Hg:	740	339	172	98	58

B. Barometric pressures at which animals died (mm Hg.):

Mice		Chicks	
In air	In oxygen	In air	In oxygen
166	83	212	90
160	76	258	90
154	94	220	88
150	87	218	89
170	70	212	87
167	69	190	93
147	67		
136	65		
142	95		
139	94		
161	75		
147	70		
176	74		
170	71		
147	76		
146	80		
171	76		
166	79		
185	74		
172	76		
Avg 158.6	70.7	218.3	89.5

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1 Armstrong, H. G., *Principles and Practice of Aviation Medicine*, 1939, Baltimore, Williams and Wilkins.

2 Horvath, S. M., Dill, D. B., and Corwin, W., *Am. J. Physiol.*, 1943, **138**, 659.

3 Hailman, H. F., PROC. SOC. EXP. BIOL. AND MED., 1943, **53**, 221.

4 Fazekas, J. F., Alexander, F. A. D., and Himwich, H. E., *Am. J. Physiol.*, 1941, **134**, 381.

5 Armstrong, H. G., and Heim, J. W., *J. Aviat. Med.*, 1938, **9**, 45.

6 Emerson, G. A., and Van Liere, E. J., *J. Lab. and Clin. Med.*, 1943, **28**, 689.

sure at which the animal either died or collapsed. Using chicks, the pressure at which the animals fell backward was decided upon as the critical pressure since this pressure is very close to death and represents a definite end-point. The pressure at which the last extensor thrust of the hind legs occurred was chosen as the end-point in death of the mice.

Using these two criteria as end-points chicks singly and mice in pairs were decompressed in atmospheres (1) of air and (2) of oxygen. To make certain that the animal jar contained as nearly pure oxygen as possible for the latter experiment it was washed out with approximately 14 times its own volume with oxygen and then a constant stream of oxygen passed through it during the trial. Samples of gas in the chamber were also analyzed with a Haldane apparatus to make certain of oxygen saturation. Since the rate of decompression was kept the same in all experiments by an inlet valve it can be seen that the same rate of inflow of air or oxygen existed throughout all the trials. Therefore at any given barometric pressure the only difference in the animal jar was the pO_2. All experiments were conducted at room temperature of 26°C. Each experiment was started at 740 mm Hg pressure and time was allowed for the animals to become adapted to the starting pressure. It is doubtful if this precaution was necessary because the natural barometric pressure varied between 741 and 751 and therefore there was no significant pO_2 difference.

An examination of the data (Table I, B) shows a considerable species difference in the anoxic resistance of mice and chicks. Wright[7] computes that 110 mm is the lowest barometric pressure at which a person breathing pure oxygen can remain conscious. He calculates that hyperventilation by washing out CO_2 from the blood could reduce alveolar pCO_2 from 40 to 23 mm. It has been stated[8] that pO_2 of alveolar air at the time of death is probably about 10 mm Hg. The very rapid polypneic breathing of mice undoubtedly accounts partly for their ability to tolerate lower barometric pressures than the chicks. Hailman's[3] data for rats decompressed in oxygen compare favorably with ours for mice, there being only about 5 mm difference at death.

Summary. Mice and chicks were decompressed in atmospheres of (1) air and (2) oxygen and the critical pressures determined below which the animals could not survive. A considerable species difference exists.

7 Wright, S., *Applied Physiology*, 1941, N. Y., Oxford Univ. Press.

8 Selladurai, S., and Wright, S., *Quart. J. Exp. Physiol.*, 1932, **22**, 233.

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Relation of Number of Injections to the Titer of Sperm Iso-agglutinins. in Mice.*

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It is well known that certain animals can form antibodies against their own spermatozoa[1] or against the spermatozoa of other individuals of the same species.[2] In the case of the species heretofore used (guinea pigs, rabbits, rats) relatively few injections have suf-

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1 Metalnikoff, S., *Ann. Inst. Pasteur*, 1900, **14**, 577.

2 Henle, W., *J. Immunol.*, 1938, **34**, 325.