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Recommended Citation

Keasling, H. H. and Hanna, Calvin (1951) "Factors Influencing the Extent of Pulmonary Hemorrhage Following Explosive Decompression," *Proceedings of the Iowa Academy of Science*, 58(1), 481-485. Available at: <https://scholarworks.uni.edu/pias/vol58/iss1/61>

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Factors Influencing the Extent of Pulmonary Hemorrhage Following Explosive Decompression

By H. H. KEASLING and CALVIN HANNA

In recent years there has been a great increase in interest in the effects of explosive decompression, primarily because this phenomenon represents a major hazard of high altitude flight. Majovski *et. al.* (1944) in their studies concerning the effect of vitamin-P-like materials on the incidence of lung hemorrhage, noted that the presence or absence of food in the stomach seemed to alter the nature of the response. Later Kibrick and Goldfarb (1944) reported that 20-27 hour fasting markedly reduced the incidence of lung hemorrhage compared with fed controls. In a previous report from this laboratory, Bass *et. al.* (1950), it was indicated that both starvation and dehydration tend to decrease the incidence of lung hemorrhage resulting from explosive decompression of mice. These studies were initiated, therefore, to investigate further the effects of starvation and dehydration with respect to their ability to reduce the incidence of lung hemorrhage in mice.

EXPERIMENTAL

As in our previous study we have used a method similar to that of Majovski *et. al.* (1944). The minimum airflow pathway of our apparatus was 3 mm. in diameter rendering both decompression and recompression virtually instantaneous (less than one second). Our criteria for the effects of the various agents has been the percentage of lungs which sink when placed in water (per cent sinks) according to the procedure we have previously described (Bass *et. al.* [1950]).

In the course of these studies, 520 white mice of both sexes weighing between 15 and 25 Grams have been decompressed. Except in those experiments designed to test the effects of starvation and dehydration, the mice received Purina dog chow and water *ad libitum*.

RESULTS AND DISCUSSION

It was of interest initially to investigate the effect of the duration of starvation upon the incidence of lung hemorrhage. The results of this study are shown in Figure 1 in which each point represents the per cent sinks for a group of at least 20 mice. With regard to the number of mice per group, we have noted that the effect of a given treatment cannot be evaluated on the basis of groups of less

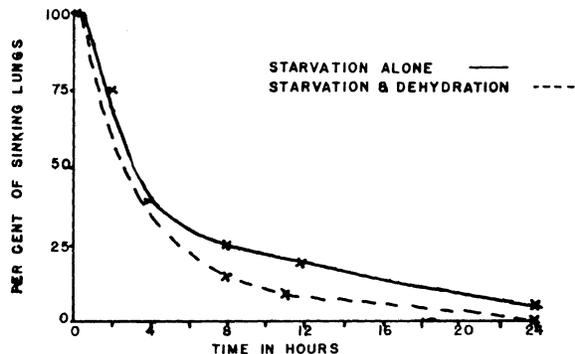


FIGURE 1 - EFFECT OF DURATION OF STARVATION ON INCIDENCE OF LUNG HEMORRHAGE

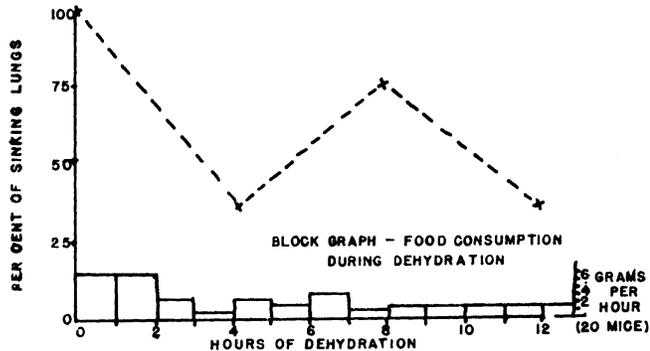


FIGURE 2 - EFFECT OF DEHYDRATION ON INCIDENCE OF LUNG HEMORRHAGE

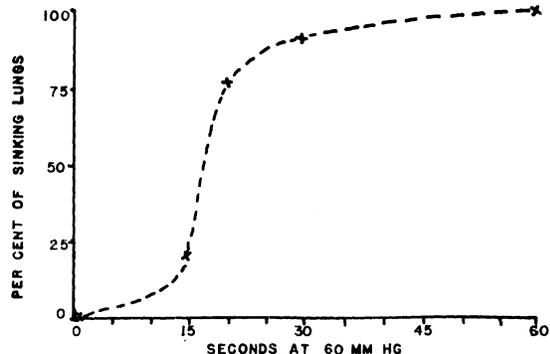


FIGURE 3 - EFFECT OF TIME AT FINAL PRESSURE ON INCIDENCE OF LUNG HEMORRHAGE

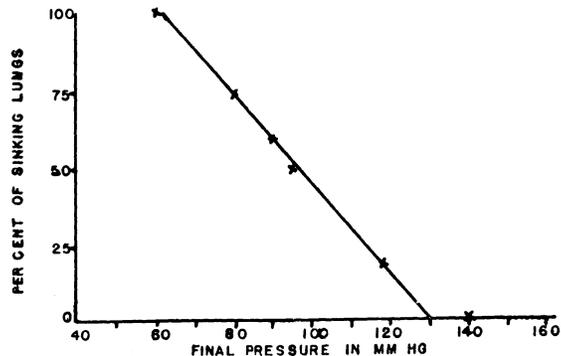


FIGURE 4 - EFFECT OF FINAL PRESSURE ON INCIDENCE OF LUNG HEMORRHAGE

than twenty mice. When smaller groups are used the first few mice may yield a percentage of sinks which is quite different than that noted with a group of 20. Further, in those groups in which we have decompressed as many as 50 mice, we have observed that only minor variation in the percentage of sinks occur when compared with this percentage computed on the basis of the first 20 mice.

The combined effect of both starvation and dehydration is also noted in Figure 1. It appeared that the added stress of dehydration when coupled with starvation merely hastened the onset of the starvation effects, however, in order to test this hypothesis, the effect of dehydration alone was determined. The percentage of sinks following dehydration alone is noted in Figure 2, it is of interest that these data appear biphasic in that the initial response was a decrease in incidence of lung hemorrhage followed by an increase and a secondary fall. This effect is correlated with the food intake of the mice as recorded in the block graph of Figure 2. This graph was constructed from data obtained on the hourly food consumption of a group of twenty mice which were dehydrated for twelve hours. It would appear, therefore, that the effect of dehydration is a reflection of the effect of voluntary starvation and that dehydration *per se* has little influence on the incidence of lung hemorrhage in decompressed mice.

Although it seemed unlikely that this effect of starvation could be a mechanical effect due to the volume in the body cavity of the mice it was necessary to investigate this factor. Accordingly a group of 20 mice starved for eight hours were given an oral injection of Bentonite Magna (1 ml.) before decompression. The percentage of sinks in this group was 50% compared with the expected percentage for 8 hours starvation of 25%. The difference between these groups is not statistically significant ($p = 0.60$).

While these studies were in progress a report by Cory and Lewis (1950) appeared in which these authors concluded that the duration of the post decompression anoxia was the major factor in determining the lethal effects of explosive decompression of rats. According to these workers nitrogen breathing gave effects similar to those noted with explosive decompression. The marked lung hemorrhage which we have seen in our studies was not observed by these workers. It was of interest, therefore, to test the effects of nitrogen breathing on the incidence of lung hemorrhage in mice. Accordingly a group of 20 mice were killed by displacing the air in a flask with nitrogen. The animals survived for one and one-half to two minutes which is in good agreement with the data of Corey and Lewis. Upon exam-

ination of the lungs, however, neither sinking lungs nor the typical lung hemorrhage of explosively decompressed mice was noted, although occasional hemorrhage spots were observed. This result indicated the desirability of investigating the effect of time at low pressure on the incidence of lung hemorrhage. Figure 3 is the plot of the data obtained in testing this factor. It would appear that between 15 and 20 seconds is a critical period during which the incidence of lung hemorrhage changes from 22% to 77% of sinking lungs. In our experiments none of the mice remaining at 60 mm. Hg. for 15 seconds or longer survived the decompression. This is in contrast with the experiments of Corey and Lewis (1950), in which rats survived decompression to 21 mm. Hg. for 20 seconds with only 30% fatalities.

If the differences between nitrogen breathing and explosive decompression in our experiments and those of Corey and Lewis (1950) are not due to species variation, it would seem that pressure change *per se* is a factor influencing the lethal effects of explosive decompression. Support for this notion is derived from the data plotted in Figure 4 which records the effect of changing the final pressure on the incidence of lung hemorrhage. In mice the pressure for 50% sinking lungs was calculated from these data as 95 mm. Hg. It is of interest that this effect appears to be a linear function of pressure and not a logarithmic function as is true of most biological phenomena. This would seem to indicate the possibility that the physical pressure changes and not biological factors are operative at least in this low pressure range.

Experiments are now in progress in an effort to determine the pressure and time relationship which yields 50% of surviving mice, and although these studies are not complete it appears that starvation reduces the percentage fatalities of decompressed mice as well as decreasing the incidence of lung hemorrhage. Fed mice decompressed to 95 mm. Hg. and held at this pressure for 15 seconds have 59% fatalities while mice which have been starved and dehydrated for 6 hours prior to decompression have only 12% fatalities. Mice surviving explosive decompression exhibit what appears to be a marked central stimulation. These mice in response to the slightest stimulus (such as the tap of a pencil on the side of the cage) alternately bounced high (one to two feet) into the air and ran very rapidly around the cage.

SUMMARY

1. The effects of starvation, dehydration, combined starvation and dehydration, final pressure and time at final pressure on the incidence of lung hemorrhage in mice have been determined.

2. The etiology of explosive decompression is discussed in relation to the data presented.

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