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MEMORANDUM REPORT

U. S. AIR FORCE
AIR MATERIEL COMMAND
WRIGHT-PATTERSON AIR FORCE BASE
DAYTON, OHIO

MCREXD-696-104I

EXPLOSIVE DECOMPRESSION TO 30 mm. Hg.

18 June 1948

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UNITED STATES AIR FORCE
AIR MATERIEL COMMAND
ENGINEERING DIVISION
MEMORANDUM REPORT ON

No. of pages - 76

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Date: 18 June 1948

SUBJECT: Explosive Decompression to 30 mm. Hg.

OFFICE: Aero Medical Laboratory

SERIAL NO: MCREXD-696-104I

Expenditure Order No. 696-61

A. PURPOSE:

1. To present a report entitled, "Explosive Decompression to 30 mm. Hg." prepared by Drs. A. Adelman and Fred A. Hitchcock for the Department of Physiology, Ohio State University, Columbus, Ohio.

B. FACTUAL DATA:

2. This report attached as Exhibit 1 was submitted in fulfillment of Contract No. W33-038 ac-14478.

C. CONCLUSIONS:

None

D. RECOMMENDATIONS:

None

Prepared by J. W. WILSON, Ph.D.
Chief, Respiration Unit

Approved by J. D. HEIM, Ph.D.
Chief, Physiology Branch

Approved by A. P. GAGGE, Lt. Col. MSC
Aero Medical Operations

Approved by EDWARD J. KENDRICKS, Col. MC
Chief, Aero Medical Laboratory

DISTRIBUTION:

Dr. Fred A. Hitchcock (25)
Department of Physiology
Ohio State University
Columbus, Ohio

School of Aviation Medicine (88)
Randolph Air Force Base
San Antonio, Texas
Distribution G

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University of Illinois
Urbana, Illinois

Engineering Division
Memorandum Report No. MCREXD-696-1041
18 June 1948

Report No. 16 Proj. 248

REPORT

By

THE OHIO STATE UNIVERSITY
RESEARCH FOUNDATION

COLUMBUS 10, OHIO

Cooperator AERO MEDICAL LABORATORY
WRIGHT-PATTERSON AIR FORCE BASE
DAYTON, OHIO
Contract W33-038ac-14478

Investigation of Explosive Decompression to 30 mm. Hg.

Subject of Report Interim Report for Period up to
November 1, 1947.

Submitted by A. Adelman and Fred A. Hitchcock

Date April 30, 1948

Engineering Division
Memorandum Report No. MCREXD-696-104I
18 June 1948

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INTRODUCTION

Until quite recently the question of exposure of men or animals to total ambient pressures less than that of the vapor pressure of body fluids was purely of academic interest. However, with the advent of aircraft capable of reaching altitudes of 100,000 feet, and the possibility of the realization of the space ship, the problem assumes aspects of urgency and practicality.

The experiments about to be described were undertaken to determine the tolerance and physiological responses of animals exposed to pressures of 30 mm. Hg., the pathology resulting from exposure to such pressures and the efficacy of protective measures against the harmful effect of such extremely low pressures.

REVIEW OF THE LITERATURE

The papers covered in this review are those which are concerned with the subject of exposures to pressures below 47 mm. Hg. Other reports, pertinent to, but not directly concerned with the subject will be discussed at the appropriate places later in this dissertation. All available civilian and military literature will be reviewed.

Paul Bert (1878) believed that the important effects of sudden decreases in pressure were the result of the anoxia and that any effects of the reduction in barometric pressure per se were combined with and subordinated to the decreased oxygen tension. Bert observed the expansion of the gases contained in the bodies of fish, birds and mammals. The initial distress of these animals upon being rapidly decompressed was attributed to the expansion of these gases. This occurs throughout the entire body of the bird, the swim bladder of the fish and the gastrointestinal tract of the mammal. He mentions the increased distension of the herbivores as compared with the carnivores. The air of the swim bladder of the carp may escape through the opening of this organ into the mouth and thus alleviate the distress. Those fish, such as the stickleback, which have a closed swim bladder die as a result of the rupture of the overdistended bladder.

Bert's experiments on rapid decompression of mammals from pressures of one atmosphere are limited to one dog, two cats, and

three rats. These animals were decompressed and maintained at the final pressure until death. The pathology seen at autopsy was ascribed to anoxia. All but the rats were exposed to terminal pressures ranging from 120-160 mm. Hg. The rats were decompressed to 45 mm. Hg. These are the first exposures to pressures less than 47 mm. Hg.

Nothing further concerning the subject was found in the literature until Armstrong (1939) reported the results of experiments performed earlier (1936) on bubble formation in the blood of animals exposed to pressures below 47 mm. Hg.

The recent war lent impetus to research in this field. It was the German Air Force which earliest in the war, conducted experiments at these low pressures. Little attempt was made to study the physiological effects, but rather the investigations were conducted with a more practical view,- the effects of bailing out of a plane at such altitudes. The experiments were carried out not only on animals (Lutz, 1946) but also on inmates of the Dachan concentration camp (Rascher, 1946). A detailed account of their findings are out of place here and will be presented in the discussion. The important finding of these workers as far as this report is concerned is the production of atelectasis as a result of exposure to this low pressure.

Schubert and Gruner (1939) in experiments on young rats, guinea pigs, dogs and cats reported atelectasis on exposure to less

than 70 mm. Hg., but claimed that this effect was overshadowed by mechanical changes in the gastrointestinal tract due to gas expansion. At pressures less than 50 mm. Hg. water vapor and the setting free of physically dissolved and chemically bound gases played an important role in bubble formation. In exposures where this occurred, these workers found that death always resulted because of atelectasis and the incompetance of the circulation due to bubbles. The same investigators in an earlier paper (Schubert and Gruner, 1936) concluded that the "boiling of tissues" did not cause death. These experiments were performed on younger animals because of their increased resistance to anoxia.

Measurements of the intra-abdominal pressure and its effect on the abdominal and thoracic organs of animals taken to 30 mm. Hg. as evidenced by X-ray records were made by von Diringshofen (1935). He found that intra-abdominal pressure increased by 50 to 100 mm. Hg., and this forced the diaphragm up, into the thorax so that the lungs were compressed while the circumference of the heart increased. Sections of the lungs of the animals which survived revealed only mild damage, while the lungs of those that died showed complete atelectasis and hyperemia.

METHODS

All the experiments described in this manuscript have been performed in pressure chambers and not in actual flight. Therefore, the term "altitude" refers to a simulated altitude, or the barometric pressure equivalent of this altitude. For the same reason the only characteristic of the altitude acting upon the animals was the reduced pressure. The temperatures, motion, vibration, sound and any other effects of actual flight were not reproduced.

The decompressions were effected as described by Whitehorn, Lein and Edelmann (1946). Essentially this method consists of the sudden completion of a connection between a large, previously evacuated tank and a smaller chamber containing the animal. Two such systems were used. The first method consisted of a glass bell jar having a volume of 22.7 liters (0.8 cu. ft.) connected to the large decompression chamber, the volume of which was 12.74 cu. meters (450 cu. ft.). The connection consisted of a thirteen inch length of one inch pipe and an one inch knife valve. At the desired moment, the valve was opened manually, establishing the connection between the two parts of the system and the pressures came into equilibrium. The small volume of the bell jar precluded its use for animals other than rats or hamsters. Therefore, a larger chamber was employed for dogs. In this system, the animal chamber was a steel tank having a volume of 0.212 cu. meters (7.5 cu. ft.)

connected to the large tank by means of an opening, fifteen inches in diameter, which was provided with a machined flange. This was bolted to a similar flange welded to a fifteen inch opening cut into the side of the decompression chamber. A sheet of cellulose acetate, 0.02 inches thick, was placed between the two flanges which were then bolted tightly together to form an airtight connection. When the desired pressure gradient had been established the membrane was sheared out by burning a slit in it by means of an electrically heated nichrome wire.

The rate of decompression of the first or smaller system was determined by optically recording the movements of a stiff rubber membrane which was part of a sealed manometer placed inside the bell jar. The average time required for eight decompressions of the bell jar from 749 mm. Hg. to 31.5 mm. Hg. was 0.806 seconds. The rate of decompression therefore was 890 mm. Hg./second or 17.2 pounds/sq. ins./second.

The rate of decompression of the second system was estimated by extrapolation of data obtained earlier (Whitehorn, Lein and Edelmann, 1946) on decompressions to 87 mm. Hg. Calculated in this manner, the time of decompression from 750 mm. Hg. to 30 mm. Hg. is 0.03 seconds. This is equivalent to a rate of 24,000 mm. Hg./second, or 420 pounds/sq. in./second.

Male albino rats and both male and female dogs were used in these experiments. In most of the experiments the animals were

unanesthetized. Nembutal, one grain per five pounds of body weight, was used when anesthesia was desired.

Except for a few preliminary experiments, the dogs were carefully deparisitised and observed by a veterinarian before being used. This precaution was taken because it had been found in the past that lung and intestinal damage resulting from parasite infestations exerted complicating influences upon the results, the lungs of control animals exhibiting as much, and in some cases even more damage than those of the decompressed dogs.

Two series of experiments were performed with dogs. The first group was explosively decompressed from atmospheric pressure to 30 mm. Hg., maintained at this final pressure for varying periods of time and then recompressed to atmospheric pressure. Oxygen was not administered at any time during this procedure. In the second series, the dogs were placed in the animal chamber which was then closed and ventilated with 100 per cent oxygen, this ventilation continuing until the termination of the experiment. Analysis of the gas flowing from the chamber revealed that after ten minutes it contained better than 90 per cent oxygen. The explosive decompressions did not occur until thirty minutes after the start of the experiment so that the dogs preoxygenated for a period of at least twenty minutes before the actual decompression. After the explosive decompression all the dogs were maintained at the final altitude for periods of from fifteen

seconds to four minutes, recompressed to 160 mm. Hg. in one minute, then brought to atmospheric pressure as rapidly as possible. The second recompression required about one minute. The animals were observed until recovery or death occurred. This procedure was followed in order to more closely simulate actual flying conditions.

The rats were used in several types of experiments. The first series was performed to determine the length of time at the final pressure which was just compatible with life. Groups of ten rats each were explosively decompressed to 30 mm. Hg. from atmospheric pressure with a subsequent stay at the final altitude which varied by ten seconds increments from a period of fifty to one of 100 seconds. When the survival time for unanesthetized rats was determined, a second series of three groups of ten rats each was subjected to the same procedure except that these animals were preoxygenated for varying periods of time before the decompression. The survival time was determined for anesthetized rats in a third series. Following this, a group of anesthetized rats was decompressed with their abdomens slit open in such a way as to allow free expansion of the abdominal contents. A fifth set of experiments was performed upon anesthetized rats with increasing amounts of air injected intraperitoneally in order to increase abdominal pressure. In all of these groups, the survival of the animals was compared with the normal controls in order to determine

whether the modified procedure increased or decreased the chances of survival following a stay at final altitude for a constant length of time.

In order to determine the effect of the explosive decompression upon the production of atelectasis it was evident that a quantitative method for the determination of the lung collapse was necessary. Previous investigators (Bert, 1878; Zuntz et al, 1906; c.f. Fegler and Banister, 1946; Schubert, 1930; Greely et al, 1943; Lutz, 1946; Edelmann et al, 1946; Fegler and Banister, 1946.) have estimated the degree of collapse by observation of the lung surface or by determining the increase in specific gravity of the lung tissue. It is obvious that inspection of the lung surface alone can give at best only a rough approximation of the condition of the entire lung and therefore would be open to severe criticism. The use of increased specific gravity as an indication of atelectasis is also open to question. The specific gravity of the lung, as of other tissues, depends upon two factors, weight and volume. If the volume of air contained in the lungs remains constant then any factor which would tend to increase the weight would cause an increase in specific gravity. Thus, a hemorrhage, edema, or even an hyperemia would exert this effect. Although the increased fluid content also caused an increase in volume of the lung, the weight of the fluid being more than that of air causes the weight increase to be out of proportion to the increased volume. Even if the

additional fluid caused a displacement of some of the air, if this is just a small reduction in the size of the alveoli, an atelectasis need not result. Therefore, in order to know whether or not atelectasis is present, it is necessary to know the volume of the lung, the weight of the lung, and the amount of air present in the lung.

The animals were sacrificed by a lethal dose of Nembutal, the trachea dissected and tied, the lungs removed from the body and carefully cleaned of adherent tissue. The trachea was clamped at its bifurcation and sectioned distal to the clamp. The lungs were then weighed on a torsion balance, after which they were placed in a volumeter consisting of a modified standard taper glass joint. The male half of the joint was fitted with a glass seal and the distal portion of the female half was closed and a portion of a graduated pipette fitted to it. The capacity of the chamber thus formed was calibrated volumetrically.

In an actual determination of the lung data, the lung was placed in the volumeter and the amount of saline required to fill the chamber determined. This value was subtracted from that necessary to fill the empty chamber, the resulting figure being the lung volume.

In order to determine the volume of the air contained in the lung, it is only necessary to reduce the atmospheric pressure about the volumeter, determine the increase in volume due to expansion of

the air in the lung and calculate its volume by an application of Boyle's law.

In practice the height of the system as shown by the height of the saline in the pipette is determined at atmospheric pressure. The system is then placed in a decompression chamber and the ambient pressure reduced to 500 mm. Hg., the volume read, and the system then recompressed. Originally the temperature of the saline was determined at each step, but after a number of determinations showed this to remain remarkably constant only one determination was made, after return to atmospheric pressure.

The derivation of the formula used is as follows:

Let V_1 be the volume of saturated air at atmospheric pressure.
V_2 be the volume of saturated air at reduced pressure.

V_{1dry} be the volume of dry air at atmospheric pressure.
V_{2dry} be the volume of dry air at reduced pressure.

r_{wet} be $V_1 - V_2$.
r_{dry} be $V_{1dry} - V_{2dry}$.

P_1 be atmospheric pressure.
P_2 be the reduced pressure.

P_{H_2O} be the partial pressure of water at the observed temperature.

P_1, P_2, and P_{H_2O} are determined experimentally.

Then, $$r_{dry} = r_{wet}\left(\frac{P_2 - P_{H_2O}}{P_2}\right)$$

$$V_{1_{dry}} = r_{dry}\left(\frac{P_2 - P_{H_2O}}{P_1 - P_2}\right) = r_{wet}\left(\frac{P_2 - P_{H_2O}}{P_2}\right)\left(\frac{P_2 - P_{H_2O}}{P_1 - P_2}\right)$$

$$V_1 = V_{1dry}\left(\frac{P_1}{P_1 - P_{H_2O}}\right)$$

$$V_1 = r_{wet}\left(\frac{P_2 - P_{H_2O}}{P_2}\right)\left(\frac{P_2 - P_{H_2O}}{P_1 - P_2}\right)\left(\frac{P_1}{P_1 - P_{H_2O}}\right)$$

By keeping P_2, or the reduced pressure constant it is possible to construct a table of corrections with only P_1, the atmospheric pressure and P_{H_2O}, the water pressure as variables. When this is done then the formula becomes

$$V_1 = r_{wet} \times f$$

Where V_1 is the volume of air in the lungs at atmospheric pressure, saturated at the atmospheric temperature.

r_{wet} is the volume increase in cc., and
f is the correction factory for the reduced pressure and the temperature of the saline.

After such a determination on the partially collapsed lung had been made, the clamp was removed and the lung allowed to deflate at atmospheric pressure. The clamp was then reapplied and the determination repeated. Thus the amount of air contained in the lungs under the pressure relations existent both in the intact and open thorax is determined.

It was found that the lung expansion was a straight line function of the barometric pressure at the pressures employed.

Data obtained by this method was employed in quantitating the atelectasis, present as the result of the experimental procedures.

Determinations were made on normal rats that had not been decompressed and these data used as the control figures. A group of ten rats were then explosively decompressed daily for a varying number of days each and then sacrificed. Another group, for reasons

which will be made clear later in this paper were exposed to five explosive decompressions in as rapid sequence as the apparatus permitted and then sacrificed.

All the rats used were male animals, approximately 150 grams in weight and were obtained from the same source (Small Animal Industries, Harlan, Indiana).

Autopsies were performed on all these animals. The thoracic and abdominal viscera were routinely examined and all visible pathology recorded. In addition, sections of the lung tissue were made from certain of the animals and examined microscopically.

The data were subjected to statistical analysis by means of the "t" test as described by Snedecor (1940).

RESULTS

Survival of dogs

All ten dogs used in the experiments survived explosive decompressions from 750 mm. Hg. to 30 mm. Hg. with subsequent periods of 15, 30, 45 and 60 seconds respectively at the final pressure. Six of the ten survived two minute periods. Two of these survivors were exposed to a four minute stay at altitude and succumbed.

Representative protocols are given in the appendix. However, because of the marked similarity in the responses of these animals, a generalized description of the events following the explosive decompression may be presented here.

Immediately following the explosive decompression, respiration was observed to become deep and rapid. This hyperventilation was brief, lasting only a few seconds and was affected by the marked abdominal distension which occurred at this time. The dog collapsed about six to eight seconds after the decompression. The animal usually lay on his side and except for respiratory movements was quiescent for a few seconds. At about 10 to 12 seconds, a series of mild convulsions occurred. These lasted for several seconds, after which the dog became quiescent again, except for respiratory movements which gradually became gasping in character and ceased about thirty seconds after the explosive decompression. During this period lacrimation, salivation and urination occurred. All of these

fluids were bubbling when observed. Occasionally there was projectile vomiting and defecation. Shortly after this, (between thirty and forty seconds after the explosion) a second swelling could be observed. This generally appeared in the lower extremities or lower abdomen and progressed headward. It appeared to be subcutaneous in nature, occurring first as a small distension about two or three inches in diameter, and then suddenly extending into the tissues adjacent to the original area. Thus, usually, the area increased by a series of sudden, almost explosive, "pops" of the skin. Occasionally, these sudden progressions would be interrupted by a slower, creeping extension of the area. Usually, all the portions of the animal's skin would be affected, including the head and nose. This distension often became so great as to cause changes in the position of the animal. A swelling of the base of the tongue was observed occasionally. This organ became so distended as to completely fill the mouth cavity.

Thirty to thirty-five seconds after the explosive decompression, the eyes glazed and to all appearances the animal was dead.

Upon recompression to 55 mm. the dog suddenly deflated and from then on only abdominal distension was noticeable. When the chamber was opened upon arrival at atmospheric pressure, respiration had already ceased in all animals kept at altitude for 30 seconds or more. On the other hand, in all instances except two of the dogs exposed for two minutes and both the dogs exposed for four minutes,

the heart was still beating. Two distinct types of heart beats were observed. More frequently the beat observed directly upon return to ground level was strong, irregular and slow (about thirty per minute). When this beat was encountered it usually was a double beat, i.e., two beats occurring close together with a long interval between the pairs of beats. The rate of this type of beat gradually increased and became more regular. At approximately four minutes after the explosive decompression, the rate was about forty per minute and at ten minutes, about 70 per minute.

Less frequently, the heart beat on return to normal barometric pressure was weak and rapid, about 80 per minute, but of a regular nature. In general, those animals which exhibited the weaker, rapid type of heart beat recovered more slowly than those with the slower, stronger beat. Not only did the type of beat encountered initially vary from animal to animal but also was inconstant for individual animals.

Respiration reappeared within thirty seconds after return to atmospheric pressure. The initial breaths usually occurred as infrequent gasps, which gradually became more rapid and regular, although they remained quite deep. After three or four minutes, the majority of the dogs exhibited periodic breathing, which persisted for three or four minutes following which respiration was almost normal in character. This point was reached nine to ten minutes after the explosive decompression.

Several instances of hyperpnea following one or two deep gasps were observed in dogs recovering from two minute exposures to the final pressure. This was not seen in the same dogs when exposed for shorter periods of time. In such instances periodic breathing occurred less frequently.

It must be mentioned that the time relationships of the circulatory and respiratory responses were essentially constant for all the dogs and bore no relationship to the length of exposure to the final pressure as long as this time was one minute or less. The responses were qualitatively the same when the two minute exposure was used, but recovery was delayed 25 to 30% since the various stages were prolonged.

When exposed for a period of only fifteen seconds the dogs did not stop breathing, and as far as respiration was concerned were definitely in better shape than when at altitude for longer periods. However, they also exhibited periodic breathing and the time required for normal breathing to be evident was not much shorter than that following the longer exposures. The heart response did not differ from that following prolonged periods at the reduced pressure.

The corneal reflexes were absent in all animals at return to normal pressure. These returned at times varying with the length of stay of the animal at the terminal pressure. They reappeared in three minutes when the stay at 30 mm. Hg. was thirty seconds or

less. If the stay were 45 seconds, the reflexes returned in five minutes, when the stay was 60 seconds, the reappearance occurred at six or seven minutes, and when the period at altitude was two minutes they did not reappear until about ten minutes.

When kept at the terminal pressure for one minute, six of the ten animals showed decerebrate rigidity after return to atmospheric pressure. The onset of this condition occurred at two different times and was correlated with the type of heart action observed immediately on return to normal pressure. Four animals which exhibited the slow type of pulse went into decerebrate rigidity at about six minutes after the explosive decompression. The condition disappeared completely three minutes after its appearance, the forelegs relaxing slightly before the hindlegs. Two animals which showed the fast, weak pulse went into rigidity lasting for six minutes, nine minutes after the explosive decompression. It was found that for a short time after recovery from decerebrate rigidity the dogs could be thrown back into that condition by applying pressure to the bottom of a foot. This reaction lasted about one minute in the hind legs and only a fraction of that time in the forelegs. Occasionally it could not be elicited from the forelegs. In two animals which did not spontaneously go into rigidity, this condition was precipitated by applying pressure to the pads of the hind feet about nine minutes after explosive decompression. This was not attempted in the cases of the other two animals.

The rigidity was observed in all six of the animals which were exposed for two minutes. It occurred at about six minutes after the explosive decompression and persisted for about four minutes. There was also a period following relaxation during which pressure applied to the foot pads would precipitate the rigid condition. This state lasted about two minutes.

The decerebrate rigidity was followed, in about 60% of the instances, by running movements of the forelegs.

At about ten minutes after the explosive decompression the dogs began to show signs of consciousness. They would respond to sound and to pinching of the soft tissue of the feet. This was soon followed by attempts to stand. These responses were slightly, but not markedly delayed in those dogs which had been at terminal pressure for two minutes. However, it was at this point that differences between these animals appeared. When exposed for one minutes or less, the animals were able to stand at about thirteen minutes after the explosive decompression and except for profuse salivation were apparently normal at the end of twenty minutes. Those dogs which recovered from two minutes exposure were not able to stand until 20 minutes after the explosive decompression and did not appear to be recovered until thirty minutes following the decompression.

Eight hours after the decompression, all dogs appeared to be fully recovered. The salivation had ceased and the appetite was good. No further effects were observed.

The salivation of these animals has been mentioned and requires elucidation. This phenomenon started about 4-5 minutes, after return to normal pressure. It occurred slowly at first, the saliva being colorless and of a watery consistency.

A few experiments were performed on dogs delivered from the University Small Animal House without being deparasitized. Those animals were treated similarly to the deparasitized dogs. The results on these animals were inconsistent and some fatalities occurred which were attributed to the poor condition of the animals.

Autopsy of three dogs which were explosively decompressed to 30 mm. Hg. from atmospheric pressure, immediately recompressed to normal pressure and sacrificed with nembutal revealed ecchymotic hemorrhages of the lungs of all three. These were not very extensive and from previous experience were judged to be compatible with life. A few small atelectatic areas were found and there were several edematous areas in the lungs of two of the dogs. The intestinal mucosa appeared to be inflamed and hyperemic. A large number of parasites, both roundworms and tapeworms, were found in the intestinal tract. The hearts appeared to be free of pathology. The brains were not examined. Examination of dogs which died as a result of exposure to extreme altitude revealed extensive pulmonary edema, hemorrhage and atelectasis. As might be expected in those animals that died before breathing air at atmospheric pressure, all the organs were markedly cyanotic, whereas, in those instances where

death occurred after a period of time at normal pressure, this condition was not present, the blood presenting a normal color.

It is interesting to note that in such of the deparasitized animals as died, there was no inflammation present in the intestinal tract, and only one of them had any parasites present. In this instance only three roundworms were found in the jejunum. The lungs of these animals appeared to be quite edematous and hemorrhagic. Large atelectatic areas were visible, as were also several emphysematous areas. The hearts appeared to be normal. The brains were not examined. The only instance of a pneumomediastinum was found in one of the dogs which had been explosively decompressed from atmospheric pressure to 30 mm. Hg. and maintained for one minute in a preliminary experiment. There was no difference in the appearance of the lungs of this animal, however, from others. There was, for example, no massive atelectasis or lung collapse as might be expected to occur.

Survival of Rats

When rats are explosively decompressed from atmospheric pressure to 30 mm. Hg. in 0.6 seconds the effects are quite similar to those previously described for dogs. Immediately following the explosive decompression, there usually occurs a short period of hyperventilation followed by a period of gasps. During this gasping phase the rat loses consciousness and collapses on the floor of the bell jar. Collapse occurs at about eight seconds. This isusually followed by a period of convulsions which occurs at between 10 and 11 seconds after the decompression and persists for 4 or 5 seconds. Following the convulsion running movements were observed. These lasted for three or four seconds. This is followed by a quiescent period which lasts until return to normal pressure and subsequent recovery.

As in the case of the dog, severe abdominal distension occurs just following the decompression. The second swelling also occurs. This starts at about thirty seconds after the explosive decompression. Usually the skin of the hind limbs is affected first with the swollen area proceeding headward as previously described for the dog.

Respiration, as far as can be seen, ceases at about thirty seconds. This is not a sharply defined point, and is difficult to determine because the last three or four inspirations are rather slow and appear to be made with great difficulty.

Deflation occurs shortly after recompression is begun, at about 55 mm. Hg., after which the animals are of normal size, except for the abdominal distension.

When the rats are recompressed to normal pressures immediately after the explosive decompression there are no visible symptoms present. Within a matter of seconds after arrival at atmospheric pressure, the animals appear to be normal in all respects. Autopsies on these animals performed after sacrifice with nembutal reveal surprisingly few, small, almost petechial hemorrhages scattered throughout the lungs. Areas of atelectasis are not seen. A slight hyperemia is evident. There is no other pathology present in any other part of the body with the exception of occasional ruptured ceca, which will be discussed later. In one rat, the liver had been torn from its diaphragmatic attachments with resulting hemorrhage.

The results of the experiments in the first group are expressed in Table I. It may be seen that after fifty seconds at final pressure, all the animals are alive when recompressed. The criterion used for life was evidence of a heartbeat. In no case was respiration preseent immediately upon descent. Seven of the ten animals breathed spontaneously while three did not, the heart stopping at an average time of 253 seconds after return to atmospheric pressure. All seven of those which resumed breathing recovered completely. When rats were exposed for 60, 70, 80, or 90 seconds

there was an increase in the number of fatalities at altitude and also after return to atmospheric pressure. The figures for these times at final pressure are quite similar and there is no significant difference in the response until the exposure is extended to ninety seconds or more. The onset of spontaneous respiration also appears to occur at times which are related to the period at terminal pressure. The 50 and 60 second rats fall in one group, with an average of 102 seconds, the 70 and 80 second animals fall into a second group with an average of 129 seconds, and each succeeding increment in time at altitude resulted in a progressively longer interval before respiration.

The effect of various periods of preoxygenation upon the survival of rats following 100 seconds at 30 mm. Hg. is shown in Table II. It may be seen that although thirty minutes of preoxygenation exerts a definite protective effect, an additional twenty minutes of breathing oxygen increases the protection to an apparent maximum since further increases to 75 minutes is not followed by additional protection.

The effects of anesthesia upon survival are shown in Table III. It is evident, by comparison with the data obtained on unanesthetized animals at a comparable time at altitude (Table I) that the viability is increased. Using these data as controls, the effects of both decreasing and increasing abdominal pressure are compared in Table III. It may be seen, that the reduction of

abdominal pressure by opening the abdomen proved to be of definite advantage as a protection against the effects of the exposure. It was also observed that this procedure altered the character of the terminal respirations. In place of the slow, labored breathing characteristic of the intact animal, the respiration of these rats was effortless and the instant of a sudden onset of respiratory failure easily determined. Although respiratory movements of the thorax were not evident until about 150 seconds after return to atmospheric pressure, the diaphragm of animal with an open abdomen could be seen to be moving, rhythmically immediately on return to ground level. This was observed in each case, by displacement of the abdominal viscera, and therefore could only be observed after the return to atmospheric pressure. An attempt to see whether the diaphragm exhibited these respiratory movements at altitude following cessation of visible thoracic movements was made by displacing the viscera before the explosive decompression. The impression was gained that movements occurred continuously but it was difficult to observe them distinctly and therefore it is not certain whether or not they are present.

When the intraabdominal pressure is increased by the injection of air, no effect on the responses studied was observed. These data are presented in Table III.

Eight rats which had been exposed to 30 mm. Hg. for 100 seconds were given artificial respiration by manual compression and release of

the thorax. Only one breathed spontaneously, the others all succumbing within 250 seconds. One of the effects of the artificial respiration was to increase the amount of oral and nasal flow which is about to be described.

A peculiar characteristic response to exposure of rats to 30 mm. Hg. for periods of twenty seconds or longer is a slow flow of fluid from the nose and mouth. This fluid is slightly viscous and usually has a pale straw color although it is sometimes contaminated with blood cells. The time of appearance, rate of flow and amount of the fluid is dependent upon the duration at altitude, the longer the stay, the earlier the appearance, the faster the rate of flow, and the greater the amount. With slight exposures, even of ten seconds, the onset of this phenomenon may not occur until an hour after return to normal pressure. The flow was quite abundant in the severe cases, approximating a total of six or seven cc. in ten minutes. When it occurred at this fast rate it was usually quite frothy. In such instances death usually occurred within ten or fifteen minutes after the onset of the exudate. A slower rate of flow meant a prolongation of the time of death, and occasionally an animal survived.

Autopsy of these animals revealed massive, heavy lungs, which were almost completely filled with the exudate, which would pour out of the trachea at the slightest pressure on the lungs. The fluid would drip out of a cut surface of the lung tissue. Occasionally free fluid would be found in the intrapleural space. The

fluid had the appearance of blood plasma, and for reasons advanced in the discussion, is considered to be a blood filtrate.

The results of the experiments on the production of atelectasis are presented in the series of tables starting with Table IV, which represents the data on the normal controls. It may be seen that the average amount of air contained in the lungs of these animals amounts to 0.8 cc. The average weight of the lungs is 1.0 gram which forms a ratio of the amount of air per gram of lung tissue of 0.8. These values are for the inflated lung. When the lungs are collapsed, these values become 0.47 cc. for the amount of air remaining in the lungs, and since the weight does not change, the ratio of lung air to weight becomes 0.47. The volumes also change from 1.70 cc. to 136 which results in a change in the specific gravity of the lung from 0.61 in the inflated state to 0.75 when collapsed.

The data on rats explosively decompressed from atmospheric pressure to 30 mm. Hg. followed by immediate recompression, once daily for various numbers of days are given in the succeeding tables. There is no appreciable difference between the values of the experimental animals and those of the control group until the 5 explosive decompression figures are reached. This may be more easily seen in Table X, which represents the average values for each group in one set of figures.

Statistical analysis reveals that there is a significant decrease of the lung air/lung weight ratio without any change in the weight of the lung when taken as the percentage of body weight.

There is also a significant increase in specific gravity of the experimental lungs as compared with that of the control lungs. The "t" values are shown in Table II.

A tendency for the atelectatic state to be built up by successive decompressions may be seen from the figures in Table X. The lung volume and amount of air in the lung tends to decrease with increasing numbers of decompressions while the specific gravity of the inflated lungs increases. The values of the deflated lungs appear to be fairly constant until the fifth day.

When rats are explosively decompressed five times in rapid succession, it is found (Tables XII, XIII) that there is a significant increase in the specific gravity of the inflated lungs, and a significant reduction in the amount of air per unit of weight. There is no change in the amount of air contained in the lungs, and although there is an increase in the weight of the lungs, this is not significant.

When the animals are preoxygenated for fifty minutes the effects of daily decompressions for five days are less severe. These results are expressed in Table XIV. It may be seen from this that there are no significant differences in any of the determinations between the control rats and those explosively decompressed to 30 mm. Hg. with preoxygenation. Nor is there any difference between the controls and a second group of controls which breathed oxygen for the same period of time as the experimental

animals. Differences between the oxygen controls and the experimental animals, both with and without preoxygenation are essentially the same as were obtained by comparison of the experimental groups with the air controls.

It appears, therefore, that preoxygenation will protect against the formation of atelectasis caused by five daily explosive decompressions to 30 mm. Hg.

Another effect of the breathing of oxygen is to cause an increase in both the weight and air content of the lung. Because of the increased volume accompanying the additional amount of air, the specific gravity is not significantly affected.

Sacrifice and autopsy of these animals reveals in general only slight hemorrhages, a mild hyperemia, occasional edema and atelectasis. These lungs present an entirely different picture qualitatively than those of animals which have remained at terminal pressure for longer periods of time and therefore were subjected to a greater degree of anoxia.

DISCUSSION

It was stated by Paul Bert that of the two possible causes of abnormal symptoms at reduced barometric pressures the reduced oxygen tension was the chief factor, the effects of the reduced ambient pressure per se being of little importance. This thesis has been confirmed by many workers since Bert.

In the experiments described in this dissertation two additional factors have to be taken into consideration. The first, and least important, of these is the rate of decompression, the second, the fact that the pressure with which the experiments were concerned was below that of the vapor pressure of the body fluids at body temperature.

The fact that the tissues of the body rapidly come into equilibrium with the atmospheric pressure was shown by Bert (1876). That this is true is undeniable. If it were not for this fact, the blood vessels and the body would explode upon ascent in an airplane. However, there is a delay in the equilization of pressure in the gas filled cavities of the body, the lungs, gastro-intestinal tract, and the middle ear. All of these cavities are open to the outside of the body and therefore the increased pressure caused by expansion of the contained gases due to the reduction of ambient pressure can be dissipated by outward flow of the gas. This flow takes definite time to occur and it is evident that when the rate of decompression of the ambient atmosphere is greater than that of

the cavity there will exist a relative positive pressure within the cavity. The volume of the middle ear being relatively small in comparison to the ability of the eustachian tube to conduct the expanded gases, there is very little difficulty and practically no discomfort from this source. The amount of expansion of intestinal gases can usually be taken up by the elasticity of the GI tract and elimination of the gas through the anus and mouth. The chief source of danger in decompressions effected at very rapid rates is the comparatively slow decompression of the lungs. The bulk of the air contained in these organs has to pass through a system of tubes before it reaches the exterior. In addition to the resistance offered in the passage through the bronchioles, bronchi and trachea, there is the additional constriction of the larynx, all of which results in a bottleneck which significantly delays the passage of the air, thus setting up a pressure wave whose peak may be well over 100 mm. Hg. (Whitehorn, Edelmann and Hitchcock, unpublished data).

When rates of decompression such as used in these experiments are encountered, there is an immediate expansion of the air contained in the lungs which exerts a relative positive pressure in all directions. The thorax expands maximally but the rate and amount of expansion is insufficient to take up the increased volume of gas and the lung tissue is brought into violent contact with the thoracic wall with resultant damage. This pressure wave is also

distributed through the other tissues of the body and is believed to be responsible for the slight hemorrhages occasionally seen in the heart and brain (Edelmann et al., 1946). It is entirely conceivable that the maximal expansion of the thoracic cavity is inhibited by the simultaneous expansion of abdominal gases and the concomitant increase in abdominal pressure which would interfere with the descent of the diaphragm. In fact it would not be surprising if the diaphragmatic movements might be prevented or if the abdominal pressure were greater than that of the thoracic pressure, the diaphragm might be pushed up into the thoracic cavity thus increasing the pressure in this region.

It has been found that when rats and dogs are subjected to an explosive decompression followed by immediate recompression there are some hemorrhages found in the lungs, less often hemorrhages in the heart and lateral ventricles of the brain. However, these are slight and do not appear to affect the animal. If sacrifice is delayed for 24 hours, repair is well under way and further delay in autopsy results in disappearance of these areas.

Dogs undergoing daily explosive decompressions for a period of weeks showed no effects at autopsy which was performed after an interval of months from the last decompression.

It is felt, therefore, that although this pressure wave exists, its effects can be minimized as far as this problem is concerned and therefore the rate is not important in this respect.

As has already been pointed out, the distension which occurs immediately upon the explosive decompression is the result of the expansion of the body gases, chiefly those of the gastrointestinal tract. This can be observed directly by decompressing the animal with the intestines exposed. Under these conditions an enormous expansion of the gut can be observed. At first glance it appears surprising that the intestinal tract would remain expanded while at altitude. Gross observation, in the rat however, reveals that fecal plugs occlude the large intestine and rectum thus blocking the escape of the gas from the rectum. It is thought that the only gas escaping from the mouth of this species is that present in the esophagus and oral cavity itself. The inability of the rat to regurgitate is undoubtedly of significance in this regard. The explusion of stomach or intestinal contents has never been seen in the rat in over 500 explosive decompressions. In the dog, which has a vomiting reflex, explosive regurgitation is a common occurrence. The vomitus usually contains fecal matter indicating that intestinal as well as stomach contents have been expelled.

Upon deflation of the exposed rat intestine following recompression, the smooth muscle of the tract remains in the stretched condition giving the appearance of an enlarged, flabby gut. When intact rats are decompressed and their intestines exposed following recompression the smooth muscle of the gut is found to be in the same stretched condition. This evidence by itself would lead to

the conclusion that the intestines were forced to expand in the intact animal. There is also the fact that in 2 per cent of the rats, death results from a rupture of the cecum. This structure is quite thin in the rat, especially when distended. Undoubtedly the pressure developing in the cecum due to gas expansion is the cause of this pathology. While the number of rats decompressed with the intestines exposed is comparatively small, twenty in all, a ruptured cecum has never been observed in these animals. It is highly probable that the expansion of the intestines is limited in the intact rat by the abdominal wall. This would lead to the development of a greater intraintestinal pressure due to the inability of the gut to expand adequately. In turn, this would increase the abdominal pressure and therefore the pressure exerted on the abdominal side of the diaphragm thus tending to prevent its descent during inspiration. That the prevention of the development of this increased abdominal pressure enables respiration to occur in a relatively normal fashion was observed in those rats whose abdomens had been opened before decompression. It is difficult to visualize the mechanism by which this procedure increases the tolerance of the animals. At first glance, it would appear that this increased ability to survive the exposure results from the unhindered respiration. These respiratory movements may be regarded from two points of view, that of pure respiration and that of circulation.

Ventilation of the lungs is undoubtedly increased in those animals with the open abdomen. However, the dry inspired air at a pressure of 30 mm. Hg. would exert a partial pressure of oxygen of only six mm. Hg., therefore, that oxygen would diffuse from the blood into the alveolar air. Increased ventilation would enhance this reversal of diffusion of oxygen by continually lowering the oxygen tension of the lung air. Interference with respiratory movements would decrease the ventilation and thereby tend to keep the alveolar oxygen tension at a higher leven than that of animals with relatively unimpaired respiratory movements. A similar theory was advanced by Lutz (c.f. Lovelace, 1946) to explain his results on the comparative survival of animals exposed to pressures just above and just below 47 mm. Hg. Lutz found that a cat and rabbit exposed to the lower pressure could be revived after return to atmospheric pressure, while those animals which had been exposed to the higher pressure could not recover. This phenomenon was thought to be due to the bubbling of gases in the blood at the lower pressure which resulted in immediate cessation of the circulation. The blood oxygen was then available for the tissues and was not lost through the lungs. This gas formation did not occur at the higher pressure, therefore the circulation continued and in passing through the lungs the oxygen was lost to the alveolar air.

On this basis, it would be expected that the intact rats, because of the impaired ventilation, would exhibit a greater tolerance than those whose abdomens had been opened before decompression. The experimental results do not bear this out, and the explanation must be sought elsewhere.

It is known that circulatory adjustments occur as the result of anoxia (Van Liere, 1942)which results in the continued supply of blood to vital centers at the expense of other structures. The minuteness of this circulatory adjustment was demonstrated by Edelmann (Unpublished results) who found that certain cells of one type, the Purkinje cell of the cerebellum, were supplied with blood during a period of anoxia, thus enabling them to survive. Other cells, adjacent to those were deprived of oxygen by constriction of the capillary supplying them with blood.

It is generally agreed that the level of the systemic blood pressure is most important for cerebral circulation (Gellhorn and Lambert, 1939). It is also obvious that failure of the circulation would obviate any circulatory adjustments. Therefore, from the point of view of maintaining the circulation, any procedure to this end would be of benefit to the survival of the animal.

In the intact animal, the increased abdominal pressure would affect circulation by interfering with venous return from the abdomen and lower extremeties, by decreasing thoracic size by upward deflection of the diaphragm, and by interfering with

respiratory movements. Circulatory embarrassment from these causes would not be present in those animals with open abdomens.

While it is true that the circulating blood would not contain much oxygen, a minimum oxygen tension might be available for a longer period of time than if the circulation were to stop completely.. The tension of carbon dioxide would be much lower in those animals with continued circulation. In cases of stagnating circulation this substance would be continually increasing and its effects would be added to anoxia, resulting in a condition of asphyxia.

Two other factors appear to disprove Lutz's theory. The first, is the fact that preoxygenation and decompression in an oxygen atmosphere affords some degree of protection to the animal. While the effects might be due entirely to the increased oxygen tension in the blood it is felt that this is only part of its protective mechanism. The increased alveolar oxygen tension would result in a smaller loss of blood oxygen via the lungs, thus maintaining the hemoglobin saturation at a higher level for a longer period of time.

The second factor is concerned with the formation of the intravascular bubbles which Lutz postulated. In order for these to occur the pressure inside the vessel must be reduced to less than 47 mm. Hg. at body temperature. At the moment of decompression, the arterial pressure is some 100 mm. Hg. above that of the ambient pressure. The only portion of the circulatory system which might conceivably be at

a sufficiently low pressure to permit vaporization of blood is that consisting of the large veins in the thorax. However, if the flow is continued, any bubbles which might form might conceivably be swept into the heart where, under the pressures developed, they would be liquefied, or dissipated in the lungs. The mass vaporization which Lutz postulates could not occur until the circulation failed and would therefore be the result and not the cause of embarrassment.

The secondary swelling observed in these experiments was undoubtedly due to the low pressures to which the animals were exposed. A pressure of 30 mm. Hg. is considerably below the vapor pressure of the body fluids at body temperature and therefore it is highly probable that the subcutaneous swelling is due to the vaporization of water. This would represent the so-called boiling of body fluids.

The subcutaneous tissues are affected to such a great extent because they are soft, contain a great deal of stored water, and are surrounded by the skin, which is distensible and normally hangs lossely, and does not exert pressure on the underlying tissues.

The reduction of pressure which occurs at the instant of decompression is transmitted to all the tissue of the body. The water in the subcutaneous tissue, as well as other regions of the body, would then tend to vaporize. In certain tissues the tendency for this vaporization to occur would be counterbalanced by the pressure of its structural components. For example, any attempt

at vaporization of fluid contained in the liver would be affected by the pressure exerted by the capsule of this organ. This situation would not exist in the subcutaneous tissue as the skin would offer little resistance to expansion until actual stretching occurred. As indicated by the poppings previously described, the resistance appears to be offered by the fascia layers, which when ruptured allow the continued expansion.

According to Harvey and his co-workers (Harvey, E. N., et al., 1944) a gas phase from or within a liquid depends in part upon the presence of bubble nuclei. These nuclei which are considered to be submicroscopic gas bubbles have not been demonstrated in the normal blood or tissues of animals by these investigators. (Harvey, E. N., et al., 1944 A). However, the fact that vaporization of water can occur without the formation of the bubbles seen in boiling can be demonstrated by the following experiment. A condom is filled with water which has been decompressed in order to remove the dissolved gases, tied so that no air is trapped, and then decompressed slowly to a pressure below that of the vapor pressure of water at the temperature of the liquid. No bubbles are seen to form within the system until the pressure reaches that of the vapor pressure of the fluid, then a gas phase starts to form in the upper part of the condom, causing it to distend. This continues slowly and then suddenly expands to a very large volume. There is left at the bottom of the condom a small amount of water which does

not contain any visible bubbles. It is believed that the liquid boiled, but the boiling proceeded entirely as a very rapid vaporization of water from the surface. The surface phenomenon would occur because the hydrostatic pressure of the water would prevent the vaporization of the layers of liquid below the surface. It is thought that this phenomenon occurred in the subcutaneous tissues accounting for the secondary swelling.

Additional experimental evidence that the secondary swelling is caused by vaporization of fluid is furnished by a recent unpublished experiment performed in this laboratory. A frog, explosively decompressed to 30 mm. Hg. at room temperature, about 20° C., did not exhibit this swelling. This is due to the fact that the vapor pressure of water at this temperature is about 18 mm. Hg. If the body temperature of the frog is raised so that the vapor pressure of the body fluid is above 30 mm. Hg., decompression to this pressure results in an enormous expansion of the animal. This shows that the expansion is due to vaporization of water.

The other symptoms which have been described in the dog have been reported previously as the result of anoxia and no new evidence was observed which would relate them to any other cause. The literature has been adequately covered by Armstrong (1939), Van Liere (1942), and Smith (1946) and it would be superfluous to repeat it at this time.

Several ideas concerning the cause of atelectasis observed following decompression have appeared in the literature (Kronecker, 1903; Bartlett, 1904; Zuntz et al, 1905; Spehl and Desquin, 1909; Heger and de Meyer, 1912; Viale, 1924; Schubert, 1930; Schneider, 1932; Hurtado, 1932; Fleisch, 1934; Drinker, 1945; Fegler and Banister, 1946; Lutz, 1946) The majority of these papers, however, are concerned with atelectasis in a very broad sense, taking any decrease in vital capacity as an atelectasis. If, as has been done in this dissertation, the term is taken to signify an actual collapse of alveoli, only two possibilities appear. Atelectasis is produced as a result of the mechanical effects of decompression or it is caused by anoxia.

One of the theories, advanced originally by Schubert (1930) and considered as the best possibility by Fegler and Banister (1946) is that exposure to low barometric pressures upsets the normal equilibrium which normally prevents the collapse of the lung. The pressure on the pulmonary side of the alveoli decreases relative to that on the pleural side thus causing the collapse. This view is supported by the observation by Fegler and Banister (1946) that a small positive intrapulmonic pressure prevents the collapse. A second theory (Lutz, 1946) states that the formation of bubbles in the intrapleural space and blood vessels within the thorax exerts pressure on the alveoli causing them to collapse.

Drinker (1945) in experiments performed on dogs subjected to low tensions of oxygen at atmospheric pressure described a pulmonary edema as the result of increased permeability of lung capillaries caused by the anoxia. Because of a peculiar arrangement of lymph channels in the lung, this exudate, which by chemical analysis has been shown to be an ultra filtrate of blood, collects between the capillary and the alveolar membrane causing compression or rupture of the alveolus. In time, this exudate proceeds up the pulmonary tree and passes through the nose and mouth. In such cases the animal dies of asphyxiation. The edema places an additional barrier to diffusion of oxygen from the alveoli to the blood, thus enhancing the anoxia and causing increased capillary damage. As shown by Drinker, any procedure which would tend to increase the production and flow of lymph in the lung would increase this effect. This would include hyperventilation and artificial respiration produced by alternate compression and relaxation of the thorax by squeezing and releasing the thorax. Theoretically, the logical treatment of the condition would be the application of intrapulmonic positive pressure by means of introducing 100 per cent oxygen under pressure. This would have three effects. The positive pressure

would tend to open collapsed alveoli and also would decrease the diffusion pressure gradient of the plasma. The high concentration of oxygen would aid in the diffusion of oxygen to the lung capillaries and reduce anoxia.

The results of the experiments reported in this dissertation support this thesis in all respects. The symptoms observed are identical with those reported by Drinker. In addition it was found that artificial respiration caused increased edema as evidenced by the oral and nasal discharge, and resulted in the death of the animals. It was also observed that the administration of oxygen before and during the decompression alleviated the edema and increased the tolerance of the animals. Drinker (1946) showed that the breathing of high concentrations of oxygen prevented increased lymph formation.

It is therefore believed that the atelectasis produced in these experiments is the result of anoxia and not of the mechanical effects of the decompression. The results of Fegler and Banister (1946) who thought that the action was mechanical since positive pressure insufflation prevented atelectasis can be explained on the same basis as mentioned by Drinker (1946) and this report. The positive pressure increased the oxygen tension and also decreased the diffusion differential for the plasma. Fegler and Banister (1946) showed that decompression from a pressure of five atmospheres to normal pressure did not result in atelectasis. It

would appear that if this pathology were the result of a mechanical effect of the decompression it would certainly occur after this decompression which was more than five times as great as that from ground level to a fraction of one atmosphere.

Fegler and Banister (1946) as well as Lutz (1946) observed bubbles in the blood vessels and other tissues of the body. The presence of these bubbles in the pulmonary tissues led to the formation of Lutz' theory that atelectasis resulted from the mechanical action of these bubbles. However, both of these investigators as well as others who have reported bubbles in tissues other than blood, found them in animals who had died at reduced pressures.

It is thought by this author that the bubbles were formed after death, as the result of the loss of blood pressure and tissue tonus, and are the result of death and not the cause of the pathology. This point was discussed earlier in this report.

The fact that daily decompressions to 30 mm. Hg. followed by immediate recompression result in atelectasis seems to be due to cumulative effects of pulmonary damage resulting from the explosive decompression. It is likely that such damaged areas would be particularly susceptible to the anoxia which develops even under the conditions of this experiment.

The methods used in these experiments has yielded new data concerning the normal rat lung. To the author's knowledge no measurements of the amount of air contained in the rat lung have

ever been reported. In addition, this quantitative method for the determination of the degree of lung collapse has proved of definite value in this work as regards the cause of the increased specific gravity of the lungs of the experimental animals. This can be seen from an analysis of the experimental results. When rats are explosively decompressed daily, from atmospheric pressure to 30 mm. Hg. for five days, a significant increase in the specific gravity of the lungs is observed. The lung air, lung weight ratio is significantly decreased. This could be brought about by a decrease in the amount of air contained in the lungs, an increase in the weight of the lungs, or both. However, it was found that there is no change in the weight of the lungs so that the increased specific gravity must have been caused by a decrease in the amount of air in the lung. Microscopic examination of sections of these lungs corroborates this finding, atelectatic areas being observed.

The specific gravity of the lungs of rats is also significantly increased as the result of five explosive decompressions in rapid succession. As in the instance of the group of rats receiving the same number of decompressions over a five day period the lung air, lung weight ratio is significantly decreased. However, the weight of the lungs is significantly increased with no change in the amount of air contained in the lungs. Therefore it must be concluded that fluid, either hemorrhage or edema was the cause of the increased specific gravity. Gross examination of these lungs reveal

large areas of hemorrhage. Microscopic examination shows slight edema as well as hemorrhage and hyperemia. Using an increase in specific gravity as the sole criterion, these lungs would be declared atelectatic when actually there was no reduction in the air volume at all.

In a continuation of these experiments one group of rats is being explosively decompressed once and a second group five times in rapid succession. Sacrifice and lung measurements are being performed one week after the decompressions in order to determine whether a period of time is required for the atelectatic condition to be produced. The results, while still incomplete, show no differences between the lungs of these animals and the controls.

SUMMARY AND CONCLUSIONS

Rats and dogs have been explosively decompressed to a total barometric pressure of 30 mm. Hg. with a subsequent period at this final pressure of from 15 to 240 seconds in an attempt to determine some of the effects of exposure to ambient pressures below the vapor pressure of body fluids at body temperature.

The procedure used was that described by Whitehorn, Lein and Edelmann (1946).

Exposure to this low pressure resulted in the following series of symptoms. Immediately following the decompression, the respiration became deep and rapid and marked abdominal distension occurred. This was followed by the collapse of the animal which took place six to eight seconds after the decompression. Mild convulsions usually occurred at ten to twelve seconds following which the dog became quiescent except for occasional respiratory gasps. Respiratory movements appeared to cease at about thirty seconds. During this period lacrimation, salivation and urination occurred, all of these fluids bubbling on emission. Occasionally there was projectile vomiting and defecation. Between thirty and forty seconds after the decompression, a second swelling was observed. This generally appeared in the hind limbs or lower abdomen and progressed headward in a series of "pops" of the skin, until finally all portions of the animals' skin was affected. This included the head and muzzle.

This distension often became so great as to cause changes in the position of the animal. Occasionally a swelling of the base of the tongue completely filled the oral cavity.

Thirty to thirty five seconds after the decompression the eyes glazed and the animal appeared to be dead.

Upon recompression to fifty five millimeters the dog suddenly deflated, only the abdominal distension being noticeable.

At return to atmospheric pressure, respiration was evident only in those dogs which had remained at thirty mm. Hg. for thirty seconds or less. The heart was beating in all instances except in two out of ten dogs which had remained at altitude for two minutes and in both dogs exposed for four minutes. Two types of heart beat were observed, a slow, strong, double beat and a weak, rapid beat.

Within thirty seconds after return to ground level, respiration reappeared as infrequent gasps which gradually became more rapid and regular. Periodic breathing usually was observed. This lasted about three or four minutes after which respiration appeared to be quite normal.

The circulatory and respiratory symptoms following exposures for periods of time up to two minutes were constant as regards their time relationships. After two minute exposures prolongation of the various stages occurred which resulted in a lengthened time of recovery.

Several effects on the nervous system were observed. Corneal reflexes were absent for periods of time varying from three minutes to ten minutes following stays at altitude of thirty seconds to two minutes respectively. A condition of decerebrate rigidity was observed in sixty per cent of the animals maintained at altitude for one minute or more. The rigid condition generally was followed by running movements of the forelegs.

At ten minutes following return to normal pressure the dogs began to regain consciousness. Following exposures to thirty millimeters of mercury for one minute or less the dogs were able to stand at about thirteen minutes and except for profuse salivation appeared to be normal at twenty minutes. When exposed for two minutes standing occurred at twenty minutes and recovery at thirty minutes.

All ten dogs survived exposures of fifteen, thirty, forty five seconds and one minute. Six of ten survived stays at altitude of two minutes while both dogs exposed for four minutes died.

It is believed that all symptoms except the abdominal distension and subcutaneous swelling were the result of anoxia. The abdominal distension was due to the expansion of the intestinal gases.

The subcutaneous swelling was thought to be due to the vaporization of water which occurred as a result of lowering the ambient pressure below forty seven millimeters of mercury which is the vapor

pressure of water at body temperature. Experiments with frogs and inanimate systems corroborate this conclusion.

The experiments on the tolerance of rats reveal results similar to those obtained on the dogs except that recovery was more rapid. It was found that although dogs could tolerate exposure to thirty millimeters for about two minutes the comparable point for the rat was about one minute. Preoxygenation for fifty minutes increased the tolerance of the rat to high altitude. This is taken to indicate that anoxia is the chief factor in this respect. A method for the determination of the amount of air contained in the lungs of animals was devised and applied to the rat.

Anesthesia decreased the tolerance of the rat.

Removal of the effect of increased abdominal pressure on the thorax by slitting the abdomen before decompression resulted in increased survival. The mechanism of this is considered theoretically to be due to the decrease interference with the circulation, thus allowing some degree of circulatory adaptation the anoxia.

Increasing the abdominal pressure by injection of air into the peritoneal cavity prior to decompression had no effect upon survival. It is thought that this signifies that a maximum effect is exerted by a given abdominal pressure and any increase beyond this pressure would have no action. This maximal pressure is developed in the intact abdomen without additional air.

One of the characteristic effects of the exposure of the rat to thirty millimeters of mercury is the oral and nasal discharge which occurs after return to atmospheric pressure. This fluid resembles plasma and is thought to be due to increased lymph formation in the lung, resulting from conditions to be described. The amount and time of onset of this discharge varies with the length of time at altitude.

It is thought that the lung capillaries are subjected to anoxia as the immediate result of the decompression as their oxygen supply comes directly from the alveolar air. This anoxia, either directly or indirectly through some intermediate such as histamine, causes an increased permeability of the lung capillaries resulting not only in increased fluid passage but also in the transfer of proteins. Thus the osmotic pressure of the fluid in the tissue spaces adjacent to the pulmonary capillaries increases tending to cause a still greater diffusion of fluid from the capillaries. This fluid barrier causes increased difficulty for oxygen diffusion and the anoxia is enhanced. Thus a vicious circle is established. Due to the anatomical arrangement of the lung lymphatics, there is a bottleneck in the pathway for drainage and a back pressure is established. This pressure causes collapse of some alveoli and rupture of others. In this way fluid enters the pulmonary system and because of the intraalveolar foramen, displaces air in adjacent alveoli and proceeds to the bronchioles, bronchi, trachea and then out through the nose and mouth.

A method for the determination of the amount of air contained in the lungs was devised and applied to the study of edema and atelectasis in the lungs of rats. This method consists of determining the amount of expansion of the lungs when subjected to decreased barometric pressure. Calculation of the amount of air required to produce this increased volume is made by an application of Boyle's Law.

The average volume of air contained in the lungs of normal rats with an average body weight of 154 grams is 0.8 cc. The average weight of the lungs is 1.0 grams and the average volume is 1.70 cc. This yields a calculated specific gravity of 0.61. These values are for lungs in the inflated condition, that is, under the pressure relationships existent in the intact thorax. When allowed to deflate under atmospheric pressure the amount of air is reduced to 0.47 cc., the volume of the lung to 1.36 cc. and the specific gravity increases to 0.75.

It is found that after an explosive decompression from atmospheric pressure to thirty millimeters of mercury followed by immediate recompression, the lung values were not changed significantly. However, after five such decompressions in five days there is a significant decrease in specific gravity with a significant decrease in the volume of air in the lung. As the weight of the lung does not change it is believed that the experimental procedure resulted in atelectasis.

If five such decompressions are accomplished in as rapid succession as possible there is no decrease in lung volume but both the weight and the specific gravity increase significantly. This is the result of hemorrhage and not atelectasis. These conclusions are corroborated by histological examination of the lung tissues.

It was thought that the atelectasis could be brought about as the result of the anoxia or as the consequence of the mechanical changes occurring inside the thorax. Preoxygenation was found to prevent the development of atelectasis following such procedures, therefore it is thought that the atelectasis results from the anoxia, probably as a result of increased fluid in the lungs. The fluid is formed by the increased permeability of the lung capillaries as previously described.

These experiments have opened up paths for additional research. At present, the lung air method is being utilized in additional investigations on the formation of atelectasis and in the study of capillary permeability. An attempt is being made to prevent the increased lymph formation in the lung by the use of substances known to decrease permeability. Similar studies on the lung air are being initiated in the dog since this species appears to be less susceptible to the production of atelectasis than the rat.

The factor of bubble formation cannot be entirely discounted and experiments designed to study this phenomenon are under way.

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BIBLIOGRAPHY

Armstrong, Harry G., 1939. Principles and Practice of Aviation Medicine. The Williams and Wilkins Co., Baltimore.

Bartlett, F. H., 1904. Am. J. Physiol. 10, 149. cf. Fegler and Banister, 1946.

Bert, Paul, 1878. La Pression Barometrique. Translated by M. A. and F. A. Hitchcock, College Book Co., Cols. Ohio, 1946.

Diringshofen, H. von. 1935. Plotzliche Extreme Luftdrucksherabsetzung in Tierversuch. Ber. ges. Physiol. esp. Pharmakologie, 81, 377, 1935.

Edelmann, A., Whitehorn W. V., Lein, A. and Hitchcock, F. A., 1946 Pathological Lesions Produced by Explosive Decompression. J. Av. Med. 17: 596.

Fegler, J. and Jean Banister, 1946. Congestive atelectasis in Lungs of Rabbits and Other Animals Subjected to the Action of Low Barometric Pressure. Quart. J. Exp. Physiol. 33, 291.

Fleisch, A. Ergeb. Physiol. 36, 249. cf. Fegler and Banister, 1946.

Gellhorn, Ernst and Edward H. Lambert, 1939. The Vasomotor System in anoxia and Asphyxia. The University of Illinois Press, Urbana.

Greeley, P. O., J. P. Baumberger, W. E. Berg et al. 1943. Experiments on Explosive Decompression. Final Report CAM, OEM,cmr 258, December 27.

Harvey, E. N., D. K. Barnes, W. D. McElroy, D. C. Pease and D. K. Barnes, 1944. Bubble Formation in Animals. I. Physical Factors, J. Cell. Compt. Physiol. 24, 1.

Harvey, E. N., A. H. Whiteley, W. D. McElroy, D. C. Pease and D. K. Barnes. Bubble Formation in Animals. II. Gas nuclei and their distribution in blood and tissues. J. Cell. Comp. Physiol. 24, 23, 1944.

Heger, P. and J. de Mejer., 1913. Etat du coeur et de la circulation pulmonaire aux differantes pressions barometriques. Ann. Soc. Sci. Med. Nat. Erux., 71, 56. cf. Fegler and Banister, 1946.

Hurtado, A. 1932. Respiratory Adaptation in the Indian Natives of the Peruvian Andes. Studies at High Altitudes. Am. J. Physiol. Anthrop. 17, 137.

Kronecker, H. 1903. Die Bergkrankeit. Berlin, cf. Fegler and Banister, 1946.

Lovelace, W. R., II. 1946 Research in Aviation Medicine for German Air Forces, CIOS, itsm #24 Medicine, U. S. Army.

Lutz, W. undated, taken as 1946, Decompression Atelectasis, Unnumbered report from Institute of Aviation Medicine in Munich. Translated under the direction of Dr. Fred A. Hitchcock.

Schneider, E. C., 1932. The vital capacity of the lungs at low barometric pressures. Am. J. Physiol. 100, 426.

Schubwrt, G. 1930. Zur Statik der Atemorgane in verdunnter Luft. Pfluger's Arkiv. 224, 260.

Schubert, G. and A. Gruner, 1936. Zur Wirkung Extremer Schwankungen des Atmospharendruckes auf den Warmbluterorganismus.

Schubert, G. and A. Gruner, 1939. Die Entstehunge Freier Case in Blut and Geweben bei Rascher Dekompression. Klin. Wchnschr. 18, 988, 1939.

Smith, J. J., 1946. The Physiological and Pathological Effects of Explosive Decompression. Ph.D. Dissertation, Northwestern University, Chicago.

Snedecor, George W., 1940. Statistical Methods. The Iowa State College Press, Ames, Iowa.

Spehl, A. and Desguin, 1909. Influence de le depression barometrique sur le quantite de sang contenue dans les poumons, Arch. Ital. Biol., 51, 23. cf. Fegler and Banister, 1946.

Van Liere, Edward J., 1942, Anoxia, Its Effect on the Body. The University of Chicago Press, Chicago.

Viale, G., 1942. L'acclimatation en haute montagnes. Arch. Ital. Biol. 72, 49. cf. Fegler and Banister, 1946.

Whitehorn, W. V. A. Lein, and A. Edelmann, 1946. The General Tolerance and Cardiovascular Responses of Animals to Explosive Decompressions, Am. J. Physiol., 147, 289.

Zuntz, N., A. Loewy, F. Muller and W. Caspari, 1906. Hohenklima und Bergwanderungen, Berlin. cf. Fegler and Banister, 1946.

NOTE: In submitting this report it is understood that all provisions of the contract between The Foundation and the Cooperator and pertaining to publicity of subject matter will be rigidly observed.

Investigator A. Edelmann Date April 30, 1948

Supervisor Fred A. Hitchcock Date April 30, 1948

For The Ohio State University Research Foundation

Executive Director James N. Owens Date May 5, 1948

Table I

The Effect of Increasing the Time at Altitude after Explosive Decompression to 30 mm. Hg. Ten Rats in Each Group

Time at 30 mm. Sec.	Av. Time of onset of convulsions Sec.	Av. Time Cessation of respiration Sec.	No. Alive at return to ground level	No. That resumed breathing spontaneously	Av. Time of first breath Sec.	No. Died after return to ground level	Av. Time of death Sec.	No. of rats surviving 24 hours or more
50	10.8	32	10	7	104	3	253	7
60	10.0	34	8	4	100	6	232	2
70	10.4	31	9	5	129	6	246	3
80	11.1	29	7	4	129	6	213*	1
90		30	9	3	148	6	226*	3
95			4	1	195	4	430	0
100	11.3	39	5	1	215	5	230	0

*Cecum ruptured in one animal

Table II

The Effect of Periods of Preoxygenation on the Survival of Rats after 100 Seconds Exposure to 30 mm. Hg.
Ten Rats in Each Group

Group	No. Alive at return to ground level	No. breathed sponta-neously	Av. Time of breath. Seconds	No. Alive at end of one hour
Air Controls	5	1	215	0
Preoxygenation				
30 min.	9	3	166	1
50 min.	10	10	153	5
75 min.	10	9	163	4

Table III

The Effect of Anesthesia and Varying Abdominal Pressure on the Survival of Rats

Each group consists of ten rats anesthetized with Nembutal (0.01 cc/20 gm.) and explosively decompressed to 30 mm. with subsequent stay of 75 seconds.

Group	No Alive at return to ground level	No. breathed sponta-neously	No. Alive at end of one hour
Controls	10	1	0
Open Abdomen	10	9	7
Inflated Abdomen			
*1 cc. Air/100 g. Body Weight	4	0	0
**3 cc. Air/100 g. Body Weight	2	0	0
**5 cc. Air/100 g. Body Weight	2	0	0
**10 cc. Air/100 g. Body Weight	2	0	0

* Only four rats used.

**Only two rats used.

Table IV

Lung Measurements
Control Rats

			Inflated				Collapsed			
Body Wt. gms.	Lung Wt. gms.	% Body Wt.	Lung Vol. cc.	Lung Air cc.	Lung Air / Lung Wt. cc/gm.	Spec. Grav.	Lung Vol. cc.	Lung Air cc.	Lung Air / Lung Wt. cc/gm.	Spec. Grav.
146	0.97	0.66	1.57	0.69	0.71	0.62	1.20	0.37	0.38	0.81
160	0.95	0.69	1.77	0.86	0.91	0.54	1.30	0.48	0.51	0.73
136	1.11	0.82	1.72	0.77	0.69	0.65	1.37	0.43	0.39	0.81
170	1.02	0.60	1.65	0.60	0.59	0.62	1.39	0.43	0.42	0.73
162	0.95	0.59	1.58	0.75	0.79	0.60	1.32	0.50	0.53	0.72
165	0.99	0.60	1.40	0.69	0.70	0.71	1.29	0.43	0.43	0.77
160	1.22	0.76	2.06	0.94	0.77	0.59	1.65	0.64	0.52	0.74
149	1.15	0.77	1.86	0.79	0.69	0.62	1.45	0.38	0.40	0.79
160	0.96	0.60	1.83	1.05	1.09	0.52	1.37	0.60	0.63	0.70
135	0.93	0.67	1.59	0.75	0.81	0.58	1.29	0.47	0.51	0.72
Av. 154	1.03	0.68	1.70	0.79	0.78	0.61	1.36	0.47	0.47	0.75

							0.61	1.36	0.41		
Av.	154	1.03	0.68	1.70	0.79	0.78					

Table V

Lung Measurements
Rats Explosively Decompressed for One Day

				Inflated				Collapsed			
Body Wt. gms.	Lung Wt. gms.	% Body Wt.	Lung Vol. cc.	Lung Air. cc.	Lung Air / Lung Wt. cc/gm.	Spec. Grav.	Lung Vol. cc.	Lung Air. cc.	Lung Air / Lung Wt. cc/gm.	Spec. Grav.
176	1.15	0.65	2.00	1.06	0.92	0.58	1.65	0.62	0.54	0.70
180	1.11	0.62	1.99	1.00	0.90	0.56	1.63	0.58	0.52	0.68
165	1.10	0.67	1.75	0.77	0.70	0.63	1.50	0.46	0.42	0.73
169	1.10	0.65	1.78	0.90	0.81	0.62	1.57	0.54	0.49	0.70
168	1.05	0.63	1.79	0.93	0.89	0.59	1.41	0.53	0.50	0.74
186	1.00	0.54	1.73	0.87	0.87	0.58	1.58	0.66	0.66	0.63
180	1.03	0.57	1.48	0.56	0.54	0.70	1.35	0.41	0.40	0.76
150	0.90	0.60	1.50	0.75	0.83	0.60	1.25	0.48	0.53	0.72
178	1.15	0.65	2.12	1.16	1.01	0.54	1.65	0.69	0.60	0.70
190	1.36	0.72	1.87	0.68	0.50	0.73	1.76	0.46	0.34	0.77
Av. 174.2	1.10	0.63	1.80	0.87	0.80	0.61	1.54	0.54	0.50	0.71

Table VI

Lung Measurements
Rats Explosively Decompressed for Two Days

				Inflated				Collapsed			
Body Wt. gms.	Lung Wt. gms.	% Body Wt.	Lung Vol. cc.	Lung Air cc.	Lung Air / Lung Wt. cc/gm.	Spec. Grav.	Lung Vol. cc.	Lung Air cc.	Lung Air / Lung Wt. cc/gm.	Spec. Grav
175	0.89	0.51	1.48	0.76	0.85	0.60	1.18	0.53	0.60	0.75
180	1.25	0.69	2.21	1.05	0.84	0.57	1.76	0.57	0.46	0.71
174	1.09	0.63	1.56	0.65	0.60	0.70	1.46	0.55	0.50	0.75
180	1.12	0.62	1.55	0.50	0.45	0.72	1.31	0.36	0.32	0.85
171	1.24	0.73	1.75	0.71	0.57	0.71	1.29	0.53	0.43	0.96
194	1.09	0.56	1.68	0.82	0.75	0.65	1.48	0.73	0.67	0.74
185	1.12	0.61	1.75	0.94	0.84	0.64	1.54	0.61	0.54	0.73
165	1.19	0.72	1.59	0.69	0.58	0.75	1.38	0.48	0.40	0.86
162	0.91	0.56	1.39	0.62	0.68	0.65	1.27	0.42	0.46	0.72
157	0.91	0.58	1.30	0.68	0.75	0.70	1.28	0.44	0.48	0.71
Av. 174.3	1.08	0.62	1.63	0.74	0.76	0.67	1.40	0.52	0.53	0.78

157	0.91	0.58	1.30	0.68						
Av. 174.3	1.08	0.62	1.63	0.74	0.76	0.67	1.40	0.52	0.53	0.78

Table VII

Lung Measurements
Rats Explosively Decompressed for Three Days

				Inflated				Collapsed			
Body Wt. gms.	Lung Wt. gms.	% Body Wt.	Lung Vol. cc.	Lung Air cc.	Lung Air / Lung Wt. cc/gm.	Spec. Grav.	Lung Vol. cc.	Lung Air cc.	Lung Air / Lung Wt. cc/gm	Spec. Grav.
190	1.05	0.55	1.78	0.80	0.76	0.59	1.51	0.57	0.54	0.70
177	1.24	0.70	2.00	0.92	0.74	0.62	1.68	0.57	0.46	0.74
181	1.00	0.55	1.50	0.66	0.66	0.67	1.29	0.35	0.35	0.78
180	0.85	0.47	1.61	0.88	1.03	0.53	1.34	0.55	0.65	0.63
182	1.02	0.56	1.72	0.80	0.79	0.59	1.39	0.59	0.58	0.73
169	1.07	0.63	1.52	0.75	0.70	0.70	1.40	0.50	0.47	0.76
171	0.93	0.54	1.61	0.79	0.85	0.58	1.40	0.54	0.58	0.66
155	1.36	0.88	1.78	0.60	0.44	0.76	1.61	0.36	0.26	0.84
182	1.70	0.93	2.50	1.10	0.65	0.68	2.30	0.73	0.43	0.74
149	1.00	0.67	1.81	0.97	0.97	0.55	1.38	0.62	0.62	0.72
Av. 173.6	1.12	0.65	1.78	0.83	0.76	0.63	1.53	0.54	0.49	0.73

Table VIII

Lung Measurements
Rats Explosively Decompressed Daily for Four Days

Body Wt. gms.	Lung Wt. gms.	% Body Wt.	Inflated				Collapsed			
			Lung Vol. cc.	Lung Air cc.	Lung Air / Lung Wt. cc/gm	Spec. Grav.	Lung Vol. cc.	Lung Air cc.	Lung Air / Lung Wt. cc/gm	Spec. Grav.
158	0.93	0.59	1.33	0.54	0.58	0.70	1.28	0.39	0.42	0.73
175	0.94	0.54	1.66	0.73	0.78	0.57	1.43	0.47	0.50	0.66
190	1.20	0.63	2.03	0.84	0.70	0.59	1.87	0.56	0.47	0.64
169	1.00	0.59	1.38	0.59	0.59	0.72	1.26	0.43	0.43	0.79
157	1.08	0.69	1.61	0.68	0.63	0.67	1.39	0.42	0.39	0.78
161	0.95	0.59	1.60	0.74	0.78	0.59	1.46	0.47	0.49	0.65
177	0.94	0.53	1.28	0.58	0.62	0.73	1.23	0.44	0.47	0.76
160	1.30	0.81	2.26	1.15	0.88	0.58	1.84	0.71	0.55	0.71
159	0.99	0.62	1.37	0.56	0.57	0.72	1.24	0.42	0.42	0.80
179	1.36	0.76	1.92	0.84	0.62	0.71	1.60	0.60	0.44	0.85
Av. 168.5	1.07	0.64	1.64	0.73	0.68	0.66	1.46	0.49	0.46	0.74

Av. 168.5 1.07 0.64 1.64 0.79

Table IX

Lung Measurements
Rats Explosively Decompressed Daily for Five Days

			Inflated				Collapsed			
Body Wt. gms.	Lung Wt. gms.	% Body Wt.	Lung Vol. cc.	Lung Air cc.	Lung Air Lung Wt. cc/gm.	Spec. Grav.	Lung Vol. cc.	Lung Air cc.	Lung Air Lung Wt. cc/gm.	Spec. Grav.
180	1.03	0.57	1.69	0.75	0.73	0.61	1.40	0.41	0.40	0.74
179	1.08	0.60	1.48	0.58	0.54	0.73	1.30	0.38	0.35	0.83
169	1.07	0.63	1.58	0.68	0.64	0.68	1.34	0.43	0.40	0.80
184	1.23	0.67	1.68	0.74	0.60	0.73	1.56	0.41	0.33	0.80
165	1.00	0.61	1.39	0.55	0.55	0.72	1.35	0.40	0.40	0.74
150	1.14	0.76	1.57	0.59	0.52	0.73	1.47	0.40	0.35	0.78
170	1.05	0.62	1.07	0.59	0.56	0.98	0.94	0.42	0.40	1.12
205	1.00	0.49	1.42	0.57	0.57	0.70	1.33	0.44	0.44	0.75
194	1.32	0.68	1.87	0.63	0.48	0.71	1.65	0.44	0.33	0.80
180	1.41	0.78	3.16	0.86	0.61	0.65	1.80	0.57	0.40	0.78
Av. 177.6	1.13	0.64	1.59	0.65	0.58	0.72	1.41	0.43	0.38	0.81

Table X

Average Values of Lung Measurements of Rats Explosively Decompressed Daily for Varying Number of Days

10 Rats in Each Group Except as Others Indicated

Group	Body Wt. gms.	Lung Wt. gms.	% Body Wt.	Inflated				Collapsed			
				Lung Vol. cc.	Lung Air cc.	Lung Air / Lung Wt. cc/gm.	Spec. Grav.	Lung Vol. cc.	Lung Air cc.	Lung Air / Lung Wt. cc/gm.	Spec.
Controls	154	1.03	0.68	1.70	0.79	0.78	0.61	1.36	0.47	0.47	0.75
1 E. D.	174	1.10	0.63	1.80	0.87	0.80	0.61	1.54	0.54	0.50	0.71
2 E. D.	174	1.08	0.62	1.63	0.74	0.76	0.67	1.40	0.52	0.53	0.78
3 E. D.	174	1.12	0.65	1.78	0.83	0.76	0.63	1.53	0.54	0.49	0.73
4 E. D.	169	1.07	0.64	1.64	0.73	0.68	0.66	1.46	0.49	0.46	0.74
5 E. D.	178	1.13	0.64	1.59	0.65	0.58	0.72	1.41	0.43	0.38	0.81
5 E. D. in one day	170	1.39	0.83	2.00	0.77	0.57	0.69	1.78	0.53	0.40	0.78
11 E. D.*	179	1.11	0.62	1.64	0.68	0,61	0.70				

*3 Animals

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Table XI

Significance of Data on Atelectasis Following Various Numbers of Explosive Decompressions as Determined by Values of "t".

Each analysis represents 18 degrees of freedom. Values of "t" of 2.101 and 2.878 or greater are required for significance to the 5% and 1% levels respectively.

T Values

Groups Analyzed	% Body Weight	Inflated		Collapsed	
		Lung Air / Lung Wt.	Spec. Grav.	Lung Air / Lung Wt.	Spec. Grav.
Controls vs.					
1. E. D.	1.493	0.375	0.314	0.668	2.166
2. E. D.	1.576	0.323	2.591	1.443	0.887
3. E. D.	1.618	0.264	0.767	0.469	1.086
4. E. D.	1.049	1.800	1.969	0.475	0.579
5. E. D.	0.293	3.907*	3.362*	3.286*	1.658

* Significant of 1% level.

Table XII

Lung Measurements
Rats Explosively Decompressed Five Times in Rapid Succession

	Body Wt. gms.	Lung Wt. gms.	% Body Wt.	Inflated Lung Vol. cc.	Inflated Lung Air. cc.	Inflated Lung Air Lung. Wt. cc/gm.	Inflated Spec. Grav.	Collapsed Lung Vol. cc.	Collapsed Lung Air cc.	Collapsed Lung Air Lung Wt. cc/gm.	Collapsed Spec. Grav.
	204	1.19	0.58	1.88	0.94	0.79	0.63	1.76	0.73	0.61	0.68
	204	1.39	0.68	2.05	0.89	0.64	0.68	1.77	0.62	0.45	0.79
	174	1.35	0.78	1.92	0.69	0.51	0.70	1.68	0.50	0.37	0.80
	178	1.52	0.85	2.19	0.82	0.54	0.69	1.97	0.56	0.37	0.77
	170	1.19	0.70	1.98	0.91	0.76	0.60	1.79	0.56	0.47	0.66
	176	1.34	0.76	2.03	0.90	0.67	0.66	1.78	0.54	0.40	0.75
	154	1.45	0.94	1.85	0.55	0.38	0.78	1.77	0.42	0.29	0.82
	156	1.18	0.76	1.76	0.68	0.58	0.67	1.51	0.49	0.42	0.78
	148	1.27	0.86	1.74	0.59	0.46	0.73	1.58	0.49	0.39	0.80
	138	1.97	1.43	2.55	0.76	0.39	0.77	2.18	0.40	0.20	0.90
Av.	170	1.39	0.83	2.00	0.77	0.57	0.69	1.78	0.53	0.40	0.78

Table XIII

The Effect of Five Explosive Decompressions in Rapid Succession on Atelectasis

	Body Wt. gms.	Lung Wt. gms.	% Body Wt.	Inflated				Collapsed			
				Lung Vol. cc.	Lung Air cc.	Lung Air / Lung Wt. cc/gm	Spec. Grav.	Lung Vol. cc.	Lung Air cc.	Lung Air / Lung Wt. cc/gm.	Spec. Grav.
Controls	154	1.03	0.68	1.70	0.79	0.78	0.61	1.36	0.47	0.47	0.75
5 E. D. - one day	170	1.39	0.83	2.00	0.77	0.57	0.69	1.78	0.53	0.40	0.78
"t" values		2,038				3.202*	3.468*			1.756	0.800

*Significant to 1% level.

Table XIV

The Effect of 50 Minutes Preoxygenation
on the Production of Atelectasis

Group	Body Wt. gms.	Lung Wt. gms.	% Body Wt.	Inflated: Lung Vol. cc.	Inflated: Lung Air cc.	Inflated: Lung Air / Lung Wt. cc/gm.	Inflated: Spec. Grav.	Collapsed: Lung Vol. cc.	Collapsed: Lung Air cc.	Collapsed: Lung Air / Lung Wt. cc/gm.	Collapsed: Spec. Grav.
Air Controls	154	1.03	0.68	1.70	0.79	0.78	0.61	1.36	0.47	0.47	0.75
O_2 Controls	179	1.13	0.63	1.99	0.95	0.85	0.57	1.67	0.57	0.51	0.67
O_2 5 E. D.	190	1.27	0.67	2.05	0.84	0.66	0.62	1.79	0.53	0.42	0.71
Air 5 E. D.	178	1.13	0.64	1.64	0.73	0.68	0.66	1.46	0.49	0.46	0.74
						"T" Values					
Air Cont. vs. 5 E. D.			2.038			3.202*	3.468*			1.756	0.800
Air Cont. vs. 5 E. D. O_2			0.768			2.090	1.120			1.60	1.38
Air Cont. vs. O_2 Cont.			1.469			0.793	1.279			1.005	4.00*
O_2 Cont. vs. 5 E. D. Air			0.341			3.206*	3.941*			1.315	3.66*
O_2 Cont. vs. E. D. O_2			1.603			2.101	2.083			2.481	2.051

*Significant to 1% level.