A. EXPERIMENTAL ANIMAL DECOMPRESSIONS TO A NEAR-VACUUM ENVIRONMENT

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FOREWORD

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This report has been reviewed and is approved.

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ABSTRACT

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To estimate the times of consciousness, collapse, and survival of animals exposed to near-vacuum environments, 126 conscious dogs were rapidly decompressed in either 1 or 0.2 second from 35,000 feet, while breathing oxygen, to a pressure less than 2 mm. Hg absolute. Groups of 6 dogs each were exposed to this low pressure for periods of time ranging from 5 to 180 seconds, with and without prior denitrogenation, and then recompressed to 35,000 feet with oxygen in either 5 or 30 seconds. The dogs collapsed within 9 to 10 seconds after decompression, as determined from motion picture films. Simultaneously, the effects of anoxia, water vapor, and other evolved gases were apparent, resulting in a generalized muscle spasticity, a few gasps, momentary convulsive seizures, apnea, and gross swelling of the body and extremities. All dogs exposed for less than 120 seconds survived, despite evidence of lung involvement. Respiration recommenced spontaneously either during recompression or at ground level, provided the heart was beating; otherwise, death was inevitable. The longer the exposure time, the more prolonged was the time for recovery which usually ranged from a few minutes to a few hours, except for 1 dog which exhibited a severe postdecompression paralysis with gradual recovery over a period of several weeks. Exposures of 120 to 180 seconds resulted in approximately 15% to more than 80% fatalities, respectively. Denitrogenated dogs tended to show a slightly better survival rate. As might be expected, the shorter the exposure time and the faster the recompression rate with oxygen, the better were the chances for uneventful and prompt recovery.

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I. INTRODUCTION

Despite the fact that a considerable number of studies have been carried out on the effects of rapid decompression to high altitudes, there is still very little information and data concerning the actual effects of exposures to extremely low barometric pressures—that is, to pressure environments approaching the near-vacuum of space. This information is becoming increasingly urgent in view of the current manned space flights, the programed flights to the surface of the moon, and the need for man to function safely within a pressure suit in space.

The extensive series of studies carried out by Hitchcock and his co-workers (5), as well as by other investigators (2, 3, 4, 6), have defined quite clearly most of the effects of rapid decompressions to at least 30 mm. Hg absolute, using experimental animals. Whether or not rapid exposures to even lower ambient pressures approaching that of a vacuum result in even more profound consequences cannot be entirely deduced from this earlier work. Some questions that are still not clear, but must be answered with a reasonable degree of confidence, are concerned with:

 1. Time of consciousness.

 2. Survival time after loss of consciousness.

 3. Extent and type of pathologic effects that might occur in the vital organs.

 4. The biologic effects of interstitial and intravascular water vapor.

 5. The effect of denitrogenation on the time of consciousness, survival, recovery, and residual effects.

The critical situation confronting an aerospace crew should accidental loss of pressure be experienced dictated the use of physiologically normal animals so that the data collected would be as valid as was possible to obtain. Normal, unanesthetized dogs were therefore used; 126 animals were rapidly decompressed to absolute pressures of 1 to 2 mm. Hg.

II. METHODS

The experimental procedures and decompression-recompression profiles are summarized in table I.

In most tests, 3 dogs were simultaneously decompressed. The rapid decompressions were all from 35,000 feet (180 mm. Hg) with the chamber flooded with oxygen. In this way, the animals remained unrestrained and hypoxia was avoided at this altitude before the decompressions. No animal was decompressed more than once.

TABLE I

Experimental procedure

All decompressions from 35,000 feet (in oxygen)		
Decompression times:	1 second	(108)
	0.2 second	(18)
Duration of exposures (6 animals in each group) 3-5, 10, 30, 60, 90, 120, 135, 150, 165, 180 seconds		
Recompression times:	5 seconds	(84)
	30 seconds	(27)
Denitrogenation: 1 hour at ground level		(42)
Type of recompression gas:	Oxygen	(117)
	Room air	(9)

Numbers in parentheses are number of animals.

Decompression times from 180 to less than 2 mm. Hg were all approximately 1 second, except for 6 faster decompressions (18 dogs) which were arranged to occur within approximately 0.2 second.

Exposure times at the absolute pressure of 1 to 2 mm. Hg for the various groups of animals ranged between 4 and 180 seconds. At least 2 groups of 3 each were exposed to this low pressure for approximately 4, 10, 30, 60, 90, 120, 135, 150, 165, and 180 seconds. In this way the critical times for the onset of unconsciousness, survival, recovery, and nonsurvivability were estimated from the effects on the 6 animals for each exposure time and for each variation in the experimental conditions—i.e., rate of recompression and time for denitrogenation before decompression.

Recompression times to 35,000 feet (180 mm. Hg) with pure oxygen were 5 seconds (28 decompressions, 84 dogs) and 30 seconds (9 decompressions, 27 dogs), followed within 10 to 15 seconds by recompression to ground level with room air. For comparison, 3 additional decompressions (9 dogs) were carried out using air instead of oxygen during 5-second recompressions.

To determine whether or not denitrogenation before decompression provided any degree of protection, 42 dogs (14 decompressions) breathed a high concentration of oxygen (> 90%) for 1 hour at ground level in the decompression chamber before the rapid exposures to low pressure. Decompression times, exposure times, and recompression times for these denitrogenated animals were similar to those already described.

The time course and chamber pressure changes for each decompression were recorded through a Statham pressure transducer on a Honeywell Visicorder. From these records, the time for each decompression could be accurately determined, as well as the exposure times and the recompression times. In addition, motion pictures were obtained for a large number of the decompressions, with an appropriate timer and pressure gage visible in the pictures.

With these film records, the precise times for complete unconsciousness and collapse could be determined, as well as the time required for the extensive swelling due to water vapor and other evolved gases. Exposure times, recompression times, and the barometric pressure at which the emphysematous swelling tended to deflate and return to normal during recompression were also determined from these film records.

To establish, as well as possible, uncomplicated baselines for survivability and recovery, no artificial respiration or other methods of resuscitation were used on any of the animals after recompression to ground level pressures. Provided the pulmonary airways are open, the process of recompression itself is as effective as a deep inspiration for ventilating the lungs, regardless of whether or not the animals are breathing. Undoubtedly, any resuscitation efforts, including the continuous administration of oxygen and possibly "overcompression" in a high pressure chamber, would have been of additional benefit during the process of recovery.

III. RESULTS AND DISCUSSION

Survivability, tolerance, and recovery from these severe exposures to extremely low pressures were better than had at first been anticipated, particularly in view of the rapid onset of virtually complete anoxia, together with the boiling effect and gas evolvement in the body fluids and tissues.

Most of the observations that were made at this extremely low pressure coincided in many respects with those previously reported by Edelmann and Hitchcock (2) for dogs exposed to 30 mm. Hg absolute. The hemorrhagic lesions found in these earlier investigations can, in part, be attributed to the extremely rapid decompression rate, reported as 0.03 second, from virtually ground level pressures (750 mm. Hg) (2) and from 522 mm. Hg to 87 mm. Hg in 0.012 second (3). The decompression phase in the present study was much less severe in terms of rate and pressure change (a pressure

2

change of approximately 180 mm. Hg in 1 second), even though the final pressure reached was much lower (approximately 1 mm. Hg absolute).

Time of consciousness and collapse

All animals exposed to the low pressure for longer than 5 seconds tended to lose consciousness and began to swell and collapse within 9 to 11 seconds. For brief exposures of only 4 to 5 seconds with recompression to 180 mm. Hg on pure oxygen, definite indications of collapse were exhibited by most of the animals at about the 11th or 12th second after the start of the decompression, even though recompression was in progress or had been completed. Under these conditions, however, with 5-second recompressions, postural unsteadiness was transitory when it occurred, and apparent recovery with signs of conscious orientation was rapid. When the recompression time to 180 mm. Hg with oxygen was prolonged to 30 seconds, after 5-second exposures, a more profound state of collapse occurred at about the 10th second, with recovery commencing during the recompression at about 140 mm. Hg (40,000 feet). These time relationships are shown in table II.

The exact moment when loss of "useful" consciousness commenced was difficult to determine merely by observation, but it must certainly have begun to occur before actual

TABLE II

Onset of postural collapse after rapid decompression from 35,000 feet to approximately 1 mm. Hg absolute

Exposure time at 1 mm. Hg	Recompression time	Collapse occurred within
	(seconds)	
< 5	5	11 - 12
< 5	30	10
> 5	5	9 - 11
> 5	30	9 - 11

postural collapse. Thus, consciousness very probably began to fade at the 8th or 9th second, if not sooner, at least for those animals exposed to the low pressure for longer than 5 seconds.

With regard to human exposures to a vacuum under similar conditions, it is suggested from these results that, for minimal loss of consciousness, recompression with oxygen to at least 180 mm. Hg (35,000 feet) must begin within 5 seconds and must be completed within the next 5 seconds (although these times cannot be stated with certainty). Figure 1 shows a comparative extrapolation for times of consciousness at altitudes above 55,000 feet, with less than 9 to 10 seconds being cautiously estimated for exposure to a vacuum on the basis of the collapse times measured in dogs. The time of "useful" consciousness appears to be less than the 12 to 15 seconds that have been measured in human subjects breathing oxygen at 50,000 to 55,000 feet (7).

Water vapor effect and subcutaneous swelling after decompression

During the decompressions all animals showed essentially the same response. Immediately after decompression, the animals exhibited excitation and increased activity for the first 5 seconds. Within the next 5 seconds, unless the animals were promptly recompressed, they began to show marked evidence of gas expansion and water vapor evolvement. This was manifested by a high degree of subcutaneous emphysema and expulsion of gas from the stomach and lower bowel, often with simultaneous projectile vomiting, defecation, and urination similar to that previously reported by others (2, 6). The water vapor effect and gas expansion were of such magnitude that the animals became completely immobilized with the extremities, neck, and body in an extended position, similar in appearance to an inflated goat-skin bag. Oddly enough, the external ears and the eyeballs did not seem to show the effects of this phenomenon and remained essentially normal in appearance, although the soft tissue around the eyes and face
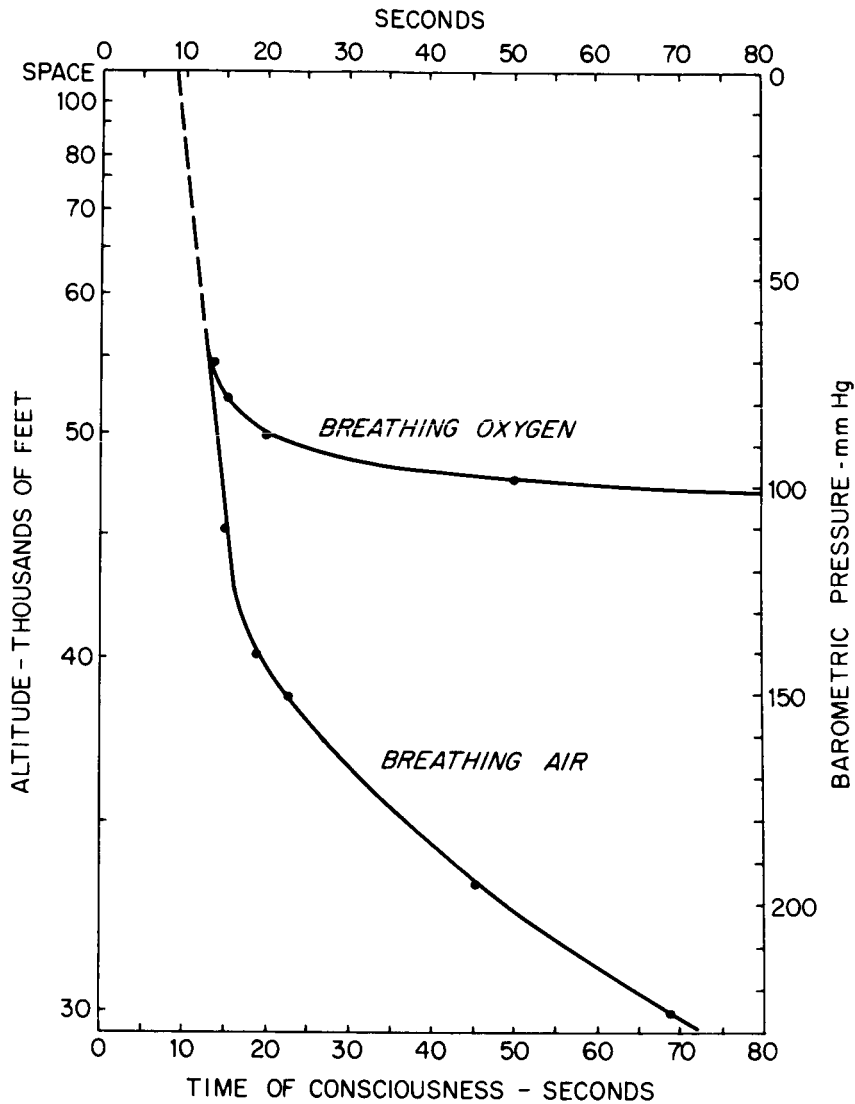
FIGURE 1

Time of consciousness for human rapid decompressions to 55,000 feet (7) with a tentative extrapolation (dashed line) to lower pressures based on canine data obtained at less than 2 mm. Hg absolute.

was often grossly distended, as was the tongue. By the end of the first 10 seconds, as indicated above, all the animals appeared to be unconscious. This was followed by what seemed to be a *grand mal* convulsion, ending in apnea and a spastic rigidity which progressed to a flaccid paralysis, although the animals remained grossly distended. All the animals had entered into this state of flaccid paralysis within about 30 seconds and remained so until they were recompressed. While at the low pressure, the saliva-like secretions and the urine became frozen and partially dehydrated. It was also noticed in several animals, after recompression to ground level, that the tongue was coated with ice. This was particularly true for animals exposed to the low pressure for longer than 2 minutes when evaporative cooling from these exposed wet surfaces became increasingly effective.

4

Recompression and recovery

During recompression to higher pressures, the subcutaneous gases were, of course, also recompressed and the animals quickly and dramatically deflated to their normal appearance, still remaining, however, in an obvious state of flaccid paralysis, unconsciousness, and apnea. This deflation process appeared to begin rather gradually with the onset of recompression. At 25 to 30 mm. Hg absolute, deflation became more rapid; at approximately 70 mm. Hg the animals appeared to have returned to their normal size. At 45 to 50 mm. Hg, however, a major portion of the deflation is complete, suggesting that water vapor is probably the predominant gas concerned with the excessive distention of the animals. The exact pressures at which the deflation process and the condensation of water vapor occurred was influenced, in part at least, by the subcutaneous and deep body temperatures which, in turn, were probably affected by the duration of the low pressure exposures and the evaporative cooling of the body surfaces.

The rapidity of recovery during or after recompression was generally dependent on the duration of the low pressure exposure, the rate of recompression, and on whether or not the animals were recompressed with oxygen or air. As might well be expected, the shorter the exposure time and the faster the recompression with oxygen, the more rapid and less complicated was the recovery period. Animals that were exposed to the low pressure for 90 seconds or less often began to breathe spontaneously during the recompression to ground level. When the exposures to the reduced pressure were longer than 90 seconds, the depressed state of the animals was intensified and apnea persisted for a prolonged period of time after recompression. Under these conditions, when first examined at ground level, the animals were usually apneic with variations in heart rate ranging from bradycardia to tachycardia. They remained apneic for varying periods of time, but spontaneous respiration always began in less than 2 to 3 minutes, provided there was a heartbeat. Otherwise, when no heartbeat was detectable, the animals invariably failed to survive. During the course of recovery, both the heart rate and respiratory frequency increased steadily for the first 2 to 5 minutes. Some of the animals exhibited a state of decerebrate spasticity when stimulated by being touched or handled. Most of the animals started purposeful movements of the extremities and head within 10 to 15 minutes and next progressed to a stage showing disorientation, with staggering and apparent blindness. During this time, coordinated control and strength in the hind legs seemed to return much more slowly than in the forelimbs. By the end of 30 minutes, none of the animals exhibited objective neurologic abnormalities; nevertheless, they appeared to be in a state of extreme fatigue and exhaustion and were very lethargic. The apparent blindness seemed to abate by the end of the 30th minute of recovery. Those that were exposed to the low pressure for longer than 60 seconds excreted, after recompression to ground level, an excessively large amount of clear, saliva-like fluid from the mouth; moist, basal rales were audible, suggesting pulmonary edema. By the end of 24 hours, the animals spontaneously cleared themselves of the rales and edema and appeared to have normal respiratory function and behavior.

Mortality incidence

As mentioned above, all dogs exposed to the near-vacuum environment for less than 120 seconds survived with essentially uneventful recoveries despite evidence of severe but transitory lung involvement. On the other hand, exposure times ranging from 120 to 180 seconds resulted in mortality rates of about 15% to more than 80%, respectively, as shown in figure 2. Each symbol in this figure represents a group of 6 animals from which the percent mortality was computed. It can be noted that no deaths resulted from the 90-second exposures, regardless of whether the animals were recompressed (with oxygen) to 35,000 feet (180 mm. Hg) within 5 seconds or 30 seconds. The 120-second exposures, however, resulted in 1 death (16.7%) in the 6 animals that were recompressed in 5 seconds and 3 deaths (50%) in the group of 6 animals that were recompressed in 30 seconds.
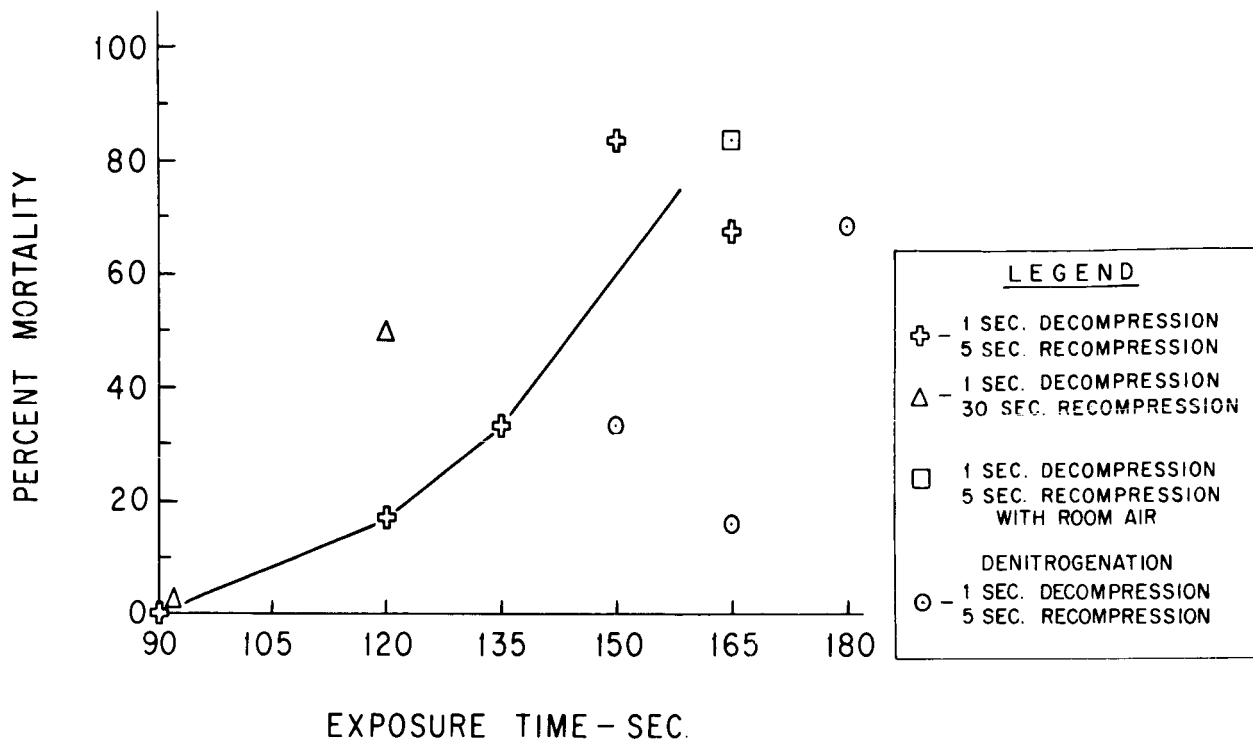
FIGURE 2

Percent mortality resulting from rapid decompression exposures to less than 2 mm. Hg absolute with and without prior denitrogenation and with 5- and 30-second recompressions to 180 mm. Hg absolute with and without oxygen as noted. Each symbol represents a group of 6 dogs. The curve for 5-second recompressions (with oxygen) was approximated by eye.

Of the 9 dogs that were rapidly recompressed with room air rather than oxygen, 3 were exposed to the low pressure for 150 seconds and all survived. The other 6 animals were exposed for 165 seconds and only 1 survived (83.3% mortality.) These results, with only a limited number of animals, indicate that the mortality incidence is not particularly different from that of animals exposed for the same period of time and then recompressed with oxygen. There are sound reasons to believe, however, that recompression with oxygen provides for more rapid oxygenation of the blood at a lower pressure than when room air is used (8). As mentioned above, recompression from near-vacuum conditions, regardless of whether or not the animals are breathing, is as effective as a deep voluntary inspiration. If oxygen is used as the recompressing gas, it virtually assures that the lungs

are filled with oxygen, provided the airways are open. On the other hand, when room air is used, it is necessary to recompress virtually to ground level pressures (750 mm. Hg) for adequate oxygenation of the lungs and blood, equivalent to 180 mm. Hg (35,000 feet) when oxygen is used.

Dogs that were denitrogenated for 1 hour at ground level before decompression and then recompressed with oxygen (fig. 2) had a significantly lower mortality rate (P < .05) as a result of these prolonged severe exposures. With denitrogenation, 5 out of 6 dogs survived a 165-second exposure, and 2 out of 6 survived an exposure of 180 seconds. It may be postulated that the cerebral and other vital tissues and fluids, when completely denitrogenated and fully saturated with oxygen before decompression, permit an extra margin of

several seconds after decompression before the intracellular oxygen partial pressure decreases to profound anoxic levels. This is possibly reflected in the longer survival time compared to the survival time of animals that were not as completely denitrogenated.

In general, under these conditions, the shorter the exposure time and the faster the recompression rate with oxygen, the better are the chances for survival and uncomplicated recovery (excluding the possibility of middle ear blockage during recompression).

Observations made on small primates

In connection with another study carried out by Rumbaugh and Ternes (10), 20 trained squirrel monkeys (4 groups of 5 each) were also decompressed in the same manner as described above, except that no exposures to the low pressure were longer than 90 seconds.

After decompression to approximately 1 mm. Hg absolute, the squirrel monkeys appeared to lose consciousness sooner than the dogs. As with dogs, they had both tonic and clonic seizures shortly after unconsciousness and this progressed to flaccid paralysis. Subcutaneous emphysema and swelling occurred, but was not as marked. During and following recompression to ground level, the monkeys recovered similarly but seemed to exhibit staggering and disorientation for a longer period.

Two monkeys died as a result of these low pressure exposures, while no dogs died from exposures that were less than 120 seconds. The first death occurred after a 10- to 12-second exposure with a 30-second recompression to 35,000 feet (180 mm. Hg) with oxygen. This animal, contrary to that seen in nonsurviving dogs, had a perceptible but irregular heartbeat after reaching ground level, but never recovered spontaneous respiration. Necropsy revealed apparent basalar atelectasis of the lungs with a questionable perforated visceral pleura just over an area of petechial hemorrhage.

The second fatality resulted from a 90-second exposure with a 5-second recompression to 35,000 feet. This monkey exhibited no heartbeat and no spontaneous respiration after reaching ground level. Except for pulmonary atelectasis and a few subpleural petechial hemorrhages, no gross pathologic abnormalities were observed at autopsy, which was performed under water.

Residual postdecompression paralysis and other nervous disorders

In only 1 animal was there clear-cut evidence of residual central nervous system involvement that continued to persist for 24 or more hours after recovery from prolonged exposure to the low pressure. The fact that approximately one-third of the surviving animals were sacrificed and autopsied within 30 minutes postdecompression leaves open to question whether or not this small percentage (approximately 1%) might not be actually larger had all survivors been observed for at least several days before sacrifice. It seems reasonable that the probability of residual pathologic conditions in the central nervous system would be increased considerably with increased exposure times, particularly for prolonged exposures of 2 minutes or longer when the chance for survival itself becomes marginal.

The one dog that exhibited severe postdecompression paralysis had been exposed to the low pressure environment for 120 seconds and then recompressed with oxygen to 180 mm. Hg in 30 seconds. After reaching ground level, the dog reacted in much the same manner as the other animals in the group, with a bradycardia and apnea which were quickly resolved. This animal, however, manifested prolonged visual impairment, and 1 hour after the exposure had not completely regained vision, as tested by reaction to motions of objects. There were no other overly abnormal neurologic manifestations at this time compared to the other animals similarly exposed. When seen approximately 20 hours later, this animal was unable to stand, but appeared to be spirited and mentally alert. Neurologic examination showed that the extremities on the right side were weak, with depressed deep tendon reflexes; while extremities on the left side were spastic, with hyperactive reflexes. The cranial nerve and pain

response reflexes were present, and the animal seemed to have unimpaired sensation to pinprick. There was also spastic urinary incontinence. Vision appeared to be normal. During the following days, progressive improvement occurred so that by the end of 1 week, the animal could walk unassisted, although the extremities on the left side still showed considerable impairment. At this time, a urinary tract infection developed without fever, but was easily controlled with antibiotics. During the next 12 weeks, the neurologic condition improved steadily; at the end of this time, the animal was able to walk and run well. The hemiparesis was scarcely discernible by neurologic examination, and the spasticity of the left side and bladder was absent.

Inasmuch as this animal had been subjected to a 120-second exposure with a 30-second recompression, the chances of survival were marginal. For a group of 6 or more animals under these same conditions, about 50% fatalities could be expected (fig. 2). Thus, in many instances, animals which otherwise might show evidence of considerable damage to the central nervous system probably do not survive. Those that do survive show, for the most part, relatively uneventful recoveries. Only in a few rare cases where the threshold of death has been very closely approached is it possible to demonstrate survival with clear-cut residual and, possibly, irreversible lesions of the central nervous system. Human hypoxic incidents in aircraft (9) and low pressure experiments with animals, by Büchner (1) and others cited by him, suggest similar conclusions.

From these observations on dogs and small primates, conclusions concerning survivability and recovery indicate that the prevention of death by rescue and repressurization with oxygen within the order of 90 seconds appears to be feasible. The fact that a fatality from a 10-second exposure has been observed in a small primate proves that any exposure to such low pressures involves great risk, and there can be considerable individual variations. Some dogs have survived exposures for as long as 3 minutes with apparently good recovery, whereas one dog suffered severe but nonfatal damage to the central nervous system after an exposure of 2 minutes.

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