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COMPARISON OF ANOXIA WITH AND WITHOUT EBULLISM

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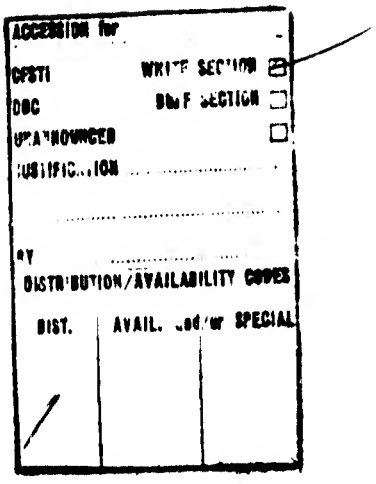

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FOREWORD

This report was prepared in the Physiology Branch under task No. 775801. The work was accomplished between May 1966 and November 1967. The paper was submitted for publication on 4 December 1967.

The animals involved in this study were maintained in accordance with the "Guide for Laboratory Animal Facilities and Care" as published by the National Academy of Sciences-National Research Council.

This report has been reviewed and is approved.

GEORGE E. SCHAFER
Colonel, USAF, MC
Commander

ABSTRACT

Water vapor and evolved gases play a unique role in the physiologic responses of animals exposed to a near-vacuum. To identify and compare these responses with those induced by other types of anoxia at higher pressures, three groups of anesthetized dogs were rapidly exposed for 2 minutes to one of three anoxic conditions: (1) 100% N_2 at ground-level pressures, (2) reduced pressure of 55 torr (55 mm. Hg absolute), and (3) 2 torr. Measurements included cardiovascular changes, arterial and mixed venous tensions of oxygen and carbon dioxide, hydrogen ion concentration, arterial lactate, pyruvate, and "excess" lactate. Responses to a near-vacuum were more extreme than with anoxia per se. The nitrogen-exposed group showed the least severe responses, while the responses of the dogs that were decompressed to 55 torr were intermediate, in most respects being closely related to those of the nitrogen-breathing group. Most of the responses resulting from the near-vacuum exposures are associated with the vaporization and ebullition of body fluids and, therefore, can be related to a combination of anoxia, ineffective circulation, and apnea.

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I. INTRODUCTION

Rapid decompression to pressures that are less than the vapor tension of body fluids not only induces an almost immediate onset of physiologic anoxia, but also subjects the body to all the physical processes that are implied in the phenomenon of "ebullism" (15). These processes include the boiling and degassing of body fluids and tissues as well as the evaporative cooling and loss of body water, heat, and various other materials. After operational pressurized aircraft were developed during World War II, Hitchcock and others (2, 8, 9, 16) did an extensive series of studies on low pressure environments. At that time the studies were limited to pressures that were in the order of 30 torr (30 mm. Hg absolute, or 72,000 ft.). Only in preparation for manned space flights has there been an effort toward a systematic investigation of the biologic effects of reduced pressures approaching a vacuum (1, 3, 5, 12).

Because of the unique combination of factors that immediately comes to bear at these extremely low pressures and begins violently to disrupt normal physiologic function, it is of interest to discriminate, if possible, between the pure effect of the anoxia itself and the superimposed influence of the boiling and outgassing effects under near-vacuum conditions. Consequently, the present study compares some of these physiologic changes and responses with those of other anoxic conditions that can be produced at pressures greater than that of water vapor at body temperature. These conditions include the breathing of pure nitrogen at ground level.

II. METHODS

Adult mongrel dogs, weighing about 20 kg., were anesthetized with sodium pentobarbital (30 mm./kg., I.V.). Indwelling vascular catheters were then placed in the abdominal aorta and inferior vena cava through a femoral artery and vein and in the pulmonary artery by way of the jugular vein to record blood pressure changes and to obtain arterial and mixed venous blood samples. Each animal breathed through a tracheal cannula and respiratory valve for recording tracheal pressure fluctuations and through which oxygen, nitrogen, or air could be supplied from a demand regulator. During the course of each experiment, continuous recordings were obtained for arterial, venous, and pulmonary arterial pressures, absolute barometric pressure, tracheal pressure, and electrocardiographic activity through calibrated Statham transducers and needle electrodes without amplification, as previously described (3), using a Honeywell Visicorder (model 1108). The transducers and catheters were filled with freshly boiled, bubble-free saline at room temperature.

Three groups of animals were exposed to one of three anoxic conditions: (1) 100% nitrogen at 750 torr (5 animals), (2) a reduced barometric pressure at 55 torr (8 animals), and (3) a reduced pressure at approximately 2 torr (10 animals). Each animal was observed only once.

Animals that were to be rapidly decompressed breathed 100% oxygen while first being slowly decompressed within 5 minutes to

200 torr (32,500 ft). At this point the alveolar oxygen tension was approximately equivalent to that of breathing air at sea level. This avoided the incidence of hypoxia before rapid decompression to the final low pressure. Each rapid decompression was carried out within approximately 1 second to pressures of either 55 or 2 torr. Immediately after each rapid decompression the oxygen supply to the regulator was changed to air.

The nitrogen-exposed animals first breathed air at the prevailing room pressure (approximately 750 torr) from a pressure-demand (A-14) regulator; then the nitrogen exposure was initiated by switching to nitrogen and inducing four forced positive pressure inspirations within about 10 seconds in order to wash out the air in the lungs and increase the pulmonary nitrogen concentration as quickly as possible.

No attempt was made to obtain blood samples during anoxic exposure because of the technical difficulties in collecting blood *in situ* under vacuum conditions. Instead, for comparison purposes, the recovery patterns as indicated by blood constituents were followed for 1 hour after recompression to ground level where all blood samples could be collected in an accurate and systematic manner.

Two control blood samples were drawn at ground level 35 and 25 minutes before the anoxic exposures while the animals breathed room air. The beginning of an anoxic exposure was identified as time zero, and each exposure was terminated at precisely 2 minutes. The first postexposure blood samples from the nitrogen-exposed animals were collected at about +2.25 minutes (0.25 minute after return to air) and at about +3 minutes for both of the decompressed groups—i.e., within 1 minute after the start of the recompression to ground level, which required about 16 to 18 seconds. The initial recovery blood samples were usually obtained before cessation of apnea and return to spontaneous breathing. Subsequent blood samples were drawn at 5, 10, 20, 30, and 60 minutes postdecompression. Each sample (arterial and mixed venous) consisted of 3 ml. of "anaerobically" drawn blood that had been stored in ice. The P_{O_2}, P_{CO_2}, and pH of the blood samples were measured at the prevailing rectal temperature of each dog, using an Instrument Laboratories analyzer, model 1L 113S. At the same time, an additional 3 ml. of arterial blood were analyzed for blood lactate by the enzymatic method of Ellis et al. (6) and for pyruvate levels by a modification of Friedemann and Haugen's method (7). Excess lactate was calculated by Huckabee's method (10). Blood glucose was measured by the enzymatic method of Keston (11) with Glucostat (14).

Each variable was analyzed with a repeated measurement analysis of variance to determine if the group means at given times were significantly different. The difference between the pre-exposure and selected postexposure mean levels within a group was also tested.

III. RESULTS

Cardiovascular effects

The effect of breathing nitrogen at ground level is shown in figure 1, which serves as a frame of reference for comparing the cardiovascular responses to the other anoxic environments at 55 torr and at 2 torr, as shown in figures 2 and 3, respectively.

A comparison between figures 1 and 2 shows a similarity in the cardiovascular responses to breathing nitrogen at ground level and to anoxia at 55 torr. In both cases, vascular pressure relationships are adequate throughout the anoxic exposure periods for presumably maintaining cardiac output and blood flow. The well-known anoxic and compensatory responses are evident with an increased heart rate for the first few seconds, followed by a progressive bradycardia, pulmonary hypertension, and an increase in the systemic venous pressure suggestive of a gradual onset of cardiac congestion, which would eventually lead to the loss of cardiac output and arterial blood pressure. Despite the similarity in these two cardiovascular patterns, the heart rate decreases sooner and falls to lower levels at 55 torr (fig. 4), and the arterial pressure does not show the same degree of compensatory response as with breathing nitrogen. This is also shown by a comparison of the systemic

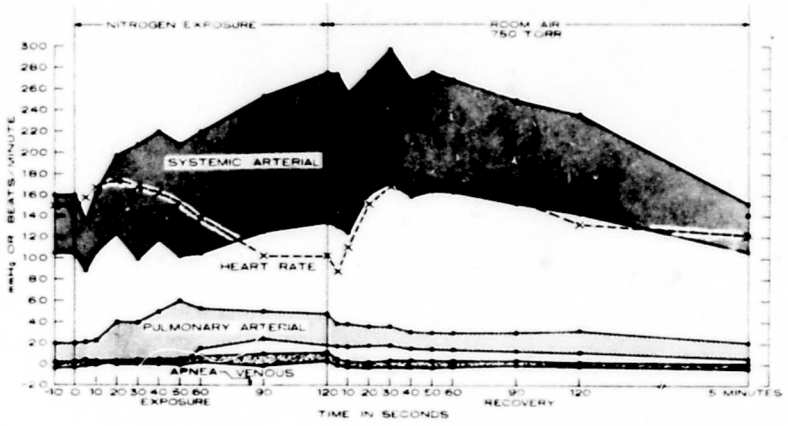

FIGURE 1

Average cardiovascular responses of 5 dogs during and after 2 minutes of breathing nitrogen at 750 torr, showing both the systolic and diastolic pressures.

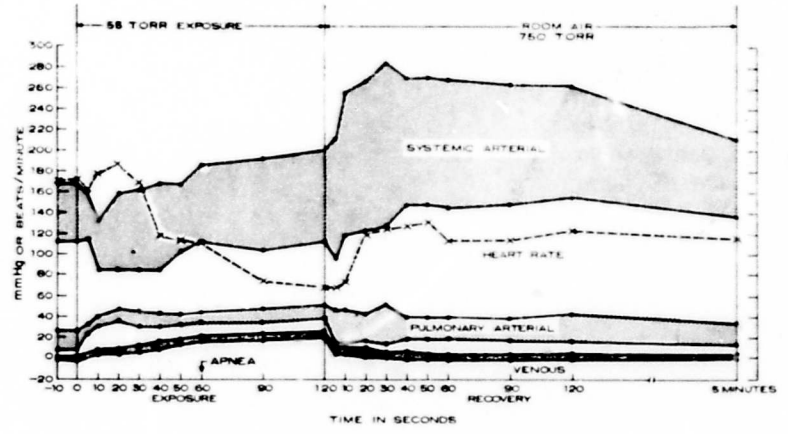

FIGURE 2

Average cardiovascular responses of 8 dogs during and after a 2-minute exposure at 55 torr.

venous and pulmonary arterial pressures when the venous pressure in breathing nitrogen (fig. 1) remains within a normal range throughout the anoxic episode. In addition, apnea usually occurs sooner during the 55-torr exposure within an average of about 60 seconds compared to about 80 seconds for the group breathing nitrogen. These differences in severity are also reflected in the recovery patterns beginning at 120 seconds with the restoration of a normal oxygen environment. Blood pressure relationships were observed to return within 5 minutes to pre-exposure values for the nitrogen-exposed group and within about 8 minutes for the animals that were decompressed to 55 torr.

In sharp contrast to the patterns shown in figures 1 and 2, the normal cardiovascular relationships are disrupted almost immediately

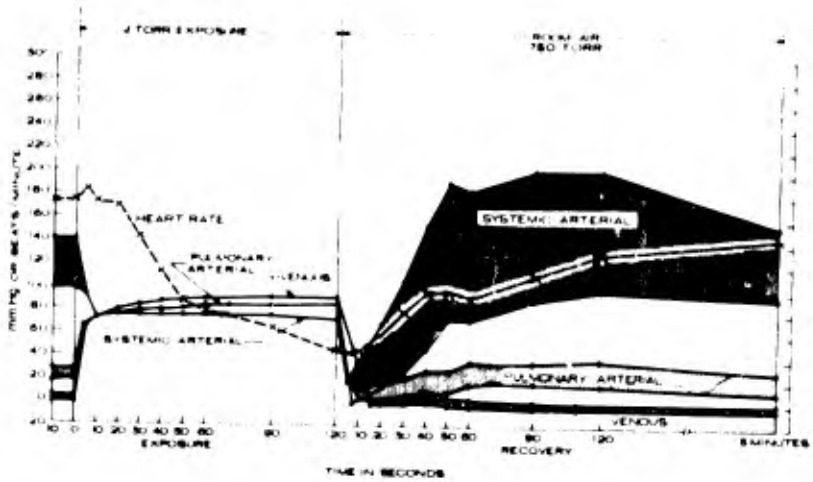

FIGURE 3

Average cardiovascular responses in 10 dogs during and after a 2-minute exposure at approximately 2 torr.

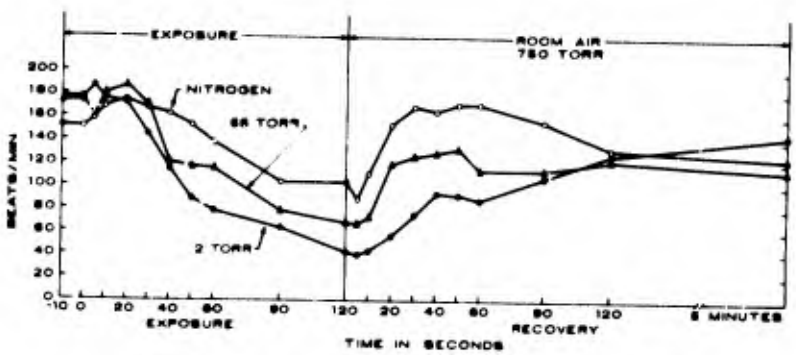

FIGURE 4

Mean heart rates during and after anoxic exposures at 750, 55, and 2 torr.

in the near-vacuum environment as the barometric pressure decreases to less than the vapor tension of water. Within the first few seconds at this low pressure (fig. 3), as the blood and body fluids begin to boil and vaporize, the conditions for an effective cardiac output and blood flow cease to exist. The venous and pulmonary arterial pressures increase abruptly to abnormally high values and equal or exceed the arterial pressure within 10 to 20 seconds. Apnea usually occurs within the first few seconds. The heart rate, as determined from the ECG, becomes progressively slower and, within the 2-minute exposure period, decreases to a much lower value than that for either the nitrogen-exposed animals or the 55-torr group, as shown in figure 4.

Upon recompression to ground level, the vaporized fluids recondense with an immediate and sharp drop in all of the vascular pressures. Simultaneously, provided the alveolar airways are open, the lungs become spontaneously ventilated and reoxygenated by the compression process. The pulmonary capillary and arterial blood becomes reoxygenated, and the cardiovascular pressure relationships begin to return toward normal for adequate restoration of blood flow and tissue perfusion. The respiratory centers usually respond soon after compression,

and spontaneous breathing recommences, followed by a recovery pattern similar to that of the two other groups.

Arterial and mixed venous P_{O_2}

Figure 5 reveals that the arterial and mixed venous oxygen tensions immediately after 2 minutes of breathing nitrogen were, on the average, at the low level of 8 to 11 mm. Hg, the mixed venous P_{O_2} being slightly higher than the arterial P_{O_2}. This may indicate that a slight amount of tissue oxygen washout was still in progress. Within 3 minutes of recovery, however, the blood P_{O_2} had rapidly returned to pre-exposure levels, as indicated in figure 5. On the other hand, the arterial and venous oxygen tensions for the decompressed groups scarcely changed between the first and third minute of recovery, the arterial O_2 tensions actually declining about 3 to 10 mm. Hg after the first minute. In contrast to the nitrogen-breathing animals during recovery, both decompressed groups had arterial oxygen tensions that were significantly lower ($P < .01$) than pre-exposure values. Differences in oxygen tension between the two decompressed groups were not statistically significant.

Arterial P_{CO_2}

Figure 6 shows the relationship of the arterial carbon dioxide tensions among the three groups of animals during the recovery period. The effective circulation and respiratory efforts ceased almost immediately when animals were decompressed to 2 torr. The arterial P_{CO_2} of the 2-torr group was significantly higher than that of the two other groups which were rendered anoxic under conditions that permitted the circulation and vigorous hyperventilation to proceed effectively, at least during the first minute of the anoxic exposure. The mean $P_{a_{CO_2}}$ of the 2-torr group was significantly higher than that of the nitrogen-breathing group at 5 and 10 minutes ($P < .001$) and at 20 and 30 minutes ($P < .05$). In comparison with the 55-torr animals, the 2-torr dogs had higher $P_{a_{CO_2}}$ levels at 3 ($P < .05$), 5, and 10 minutes ($P < .01$). The highest accumulated $P_{a_{CO_2}}$ levels in the 2-torr group occurred within 5 minutes (57.9 mm. Hg) and returned to pre-exposure levels between 30 and 60 minutes. In contrast, the $P_{a_{CO_2}}$ levels of the other two groups were lower than, and never surpassed, their pre-exposure levels. In the nitrogen-breathing group, when a measurement

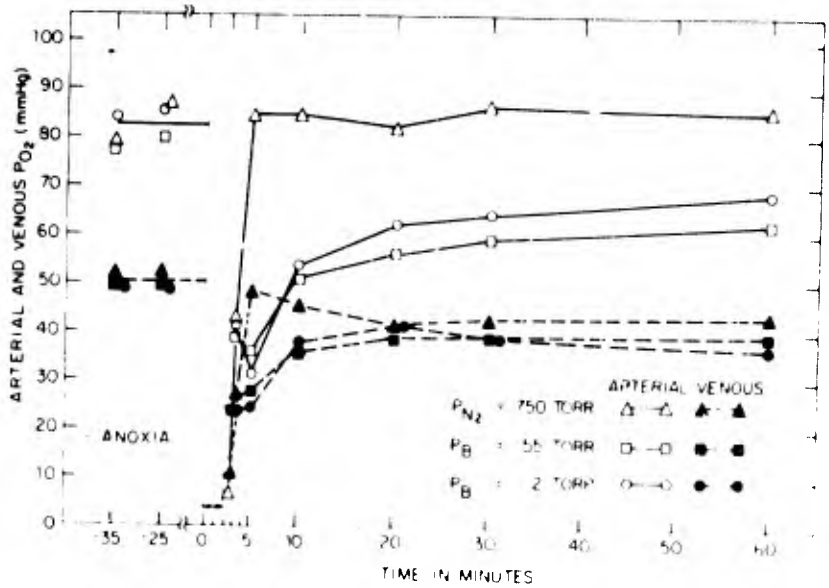

FIGURE 5

Arterial and mixed venous oxygen tensions after anoxic exposures at 750, 55, and 2 torr.

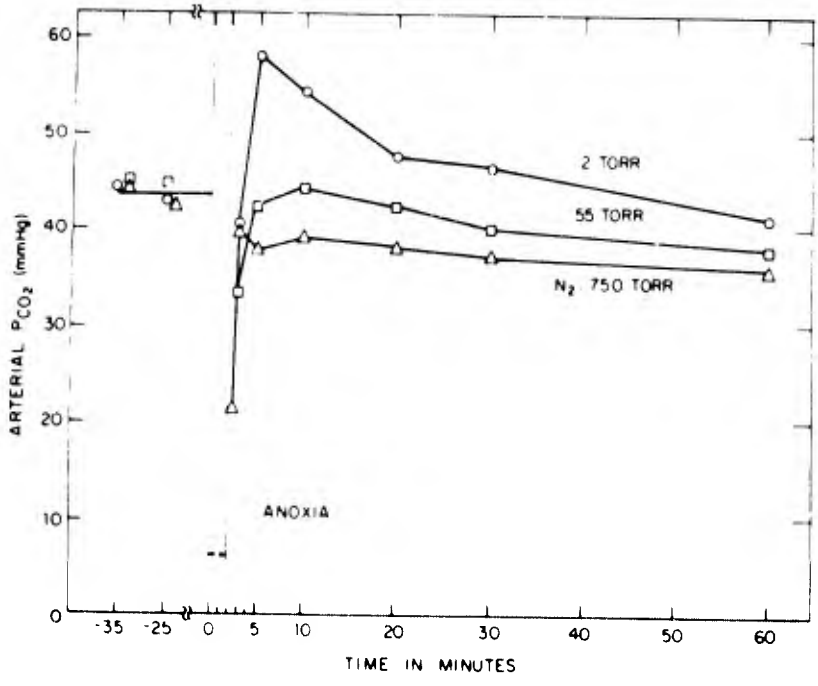

FIGURE 6

Arterial P_{CO_2} values during recovery from anoxic exposure at 750, 55, and 2 torr.

was possible at the termination of anoxia (+0.25 minute), the Pa_{CO_2} level was reduced from a pre-exposure value of 43 to 22 mm. Hg and within 1 minute had returned to 40 mm. Hg. The mixed venous P_{CO_2} levels were observed to follow the same course as the arterial P_{CO_2} levels, always being higher than the arterial levels by about 3 to 8 mm. Hg for the respective groups.

Arterial pH

The calculated mean pH values for the three groups during the first hour of recovery are shown in figure 7. The trend of the pH values for the nitrogen-breathing and the 55-torr groups was similar. After the first minute and through the next 30 minutes of recovery, each pH value was significantly higher ($P < .01$) than those for the 2-torr group. The greatest pH shift from pre-exposure values was measured in the arterial blood of the nitrogen-exposed group (from a mean of 7.32 to 7.56) immediately at the termination of the anoxic exposure. Values for the decompressed groups

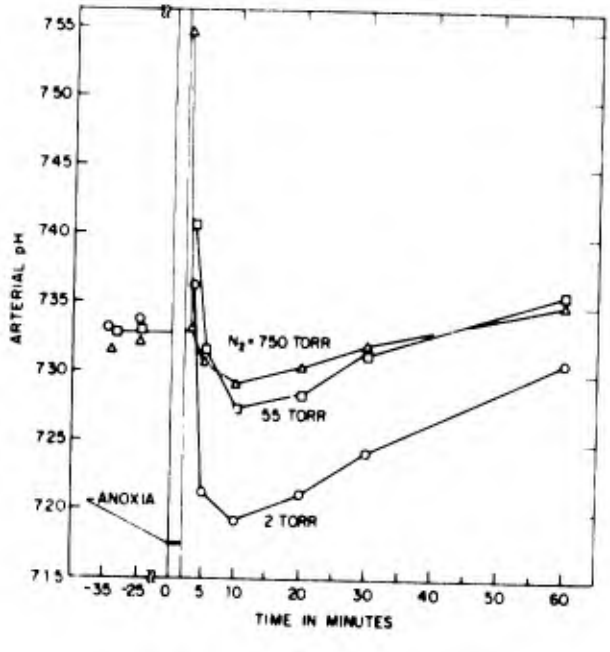

FIGURE 7

Arterial pH values during recovery from anoxic exposures at 750, 55, and 2 torr.

could not be obtained this soon, but at the first minute of recovery the 55-torr group had a higher arterial pH than the 2-torr group (7.41 compared to 7.36). The arterial pH of the 2-torr animals showed the most rapid and greatest decline to subnormal levels, reaching pH values of 7.19 within 10 minutes and returning gradually toward pre-exposure values during the rest of the hour. Both the nitrogen-breathing and 55-torr groups showed a much less severe decrease below pre-exposure values and, consequently, a more rapid return to the pre-exposure range. In each case, the mixed venous pH levels remained about 0.02 unit below, and closely followed, the arterial pH levels for each group.

Arterial lactate levels

The mean lactate levels in the arterial blood, as shown in figure 8, had increased approximately threefold in all groups at the termination of the anoxic exposures—i.e., from 0.74 to 2.26 mmoles/liter in the nitrogen-exposed group, from 0.77 to 2.46 mmoles/liter in the 2-torr group, and from 1.04 to 3.25 mmoles/liter in the 55-torr group. Between 3 and 5 minutes (1 to 3 minutes postexposure), mean lactate levels continued to increase in the 2-torr group, with a slight indication of an increase in the 55-torr group, whereas the disappearance of lactate had already commenced within 1 minute after termination of the anoxia in the nitrogen-breathing group.

Although not readily apparent in figure 8 because of the different control baselines, on the basis of a percentage change from pre-exposure values, the 2-torr group showed the greatest and most sustained change, while the 55-torr animals showed change intermediate to that of the 2-torr and nitrogen groups.

Arterial pyruvate levels

The changes in the arterial pyruvate levels during the first hour of recovery are shown in figure 9. In all three groups of animals, immediately upon termination of the anoxia, the arterial pyruvate was not much different from

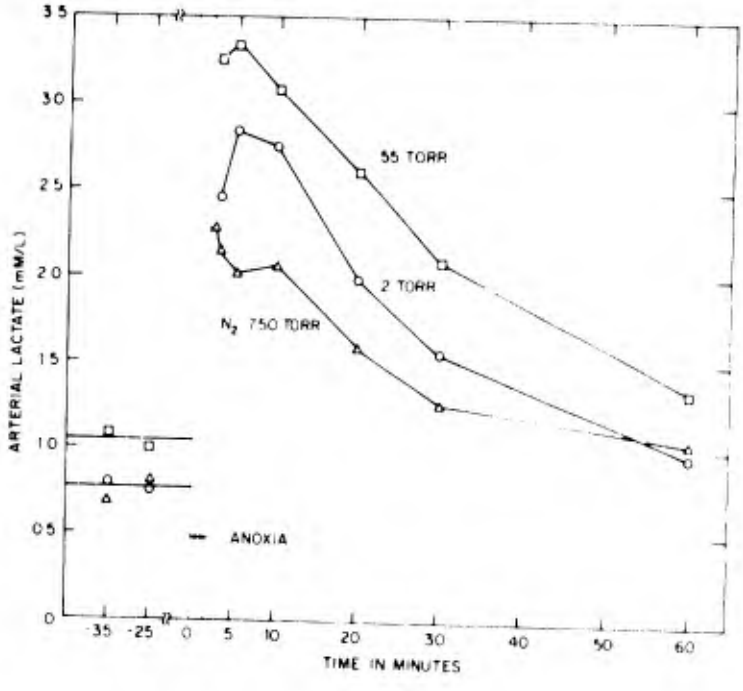

FIGURE 8

Arterial lactate levels during recovery from anoxic exposures at 750, 55, and 2 torr.

the pre-exposure values. Within the first 10 minutes, however, the pyruvate concentration more than doubled, then declined toward, but did not reach, pre-exposure levels during the following 50 minutes.

Lactate-pyruvate ratios

The ratios between the arterial lactate and pyruvate values are shown in figure 10. Inasmuch as the maximum arterial lactate values occurred within 3 minutes postexposure (fig. 8), and the pyruvate levels peaked at about 10 minutes postexposure (fig. 9), the L/P ratio reflects this relationship, with highly significant increases at the termination of the anoxic exposures and a decline to pre-exposure values within 20 minutes of recovery. The trend through the first 10 minutes of recovery was similar for all groups, with only a statistically minor difference between the nitrogen-breathing and 55-torr groups. On the other hand, the L/P ratios during this initial time interval for the 2-torr animals were significantly higher ($P < .05$) than those of the other two groups, with the ratios returning to pre-exposure values most rapidly in the nitrogen-breathing group.

"Excess" lactate

On the basis of the measured lactate and pyruvate values, the "excess" lactate (XL) levels were computed for the first hour of recovery and are shown in figure 11. It appears that in all three groups the largest quantity of XL existed at the termination of the anoxia, with the most rapid rate of removal occurring in the nitrogen-exposed group, requiring about 10 minutes for complete disappearance. In comparison, both the 55-torr and 2-torr groups remained significantly higher ($P < .05$ and $P < .001$, respectively) during this initial time interval, requiring about 20 minutes and 30 minutes, respectively, for disappearance of the XL.

Blood glucose levels

Glucose concentrations in the arterial blood during the recovery period are shown in figure 12. During the first few minutes of recovery, the glucose levels tended to increase in all groups, reaching their highest peaks within 5 to 10 minutes postexposure, then gradually declining toward pre-exposure values.

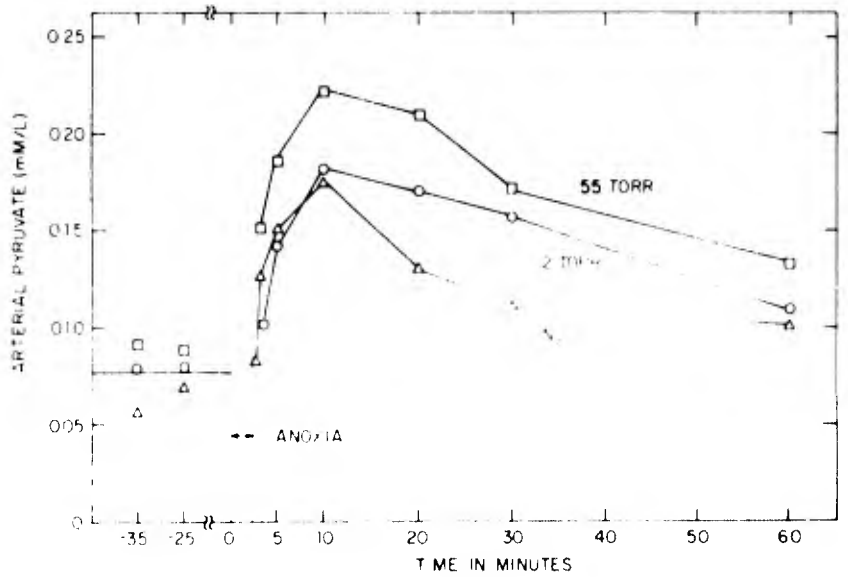

FIGURE 9

Arterial pyruvate levels during recovery from anoxic exposures at 750, 55, and 2 torr.

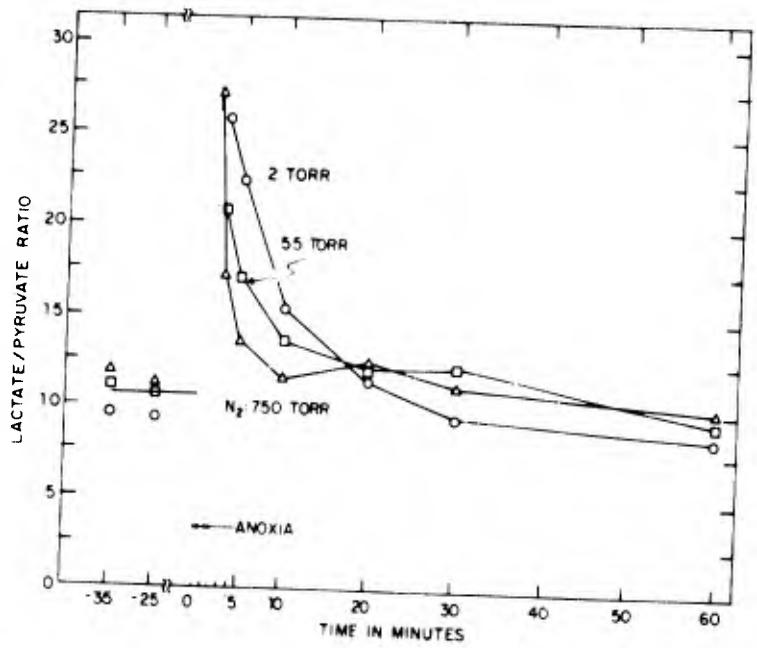

FIGURE 10

Lactate/pyruvate ratios during recovery from anoxic exposures at 750, 55, and 2 torr.

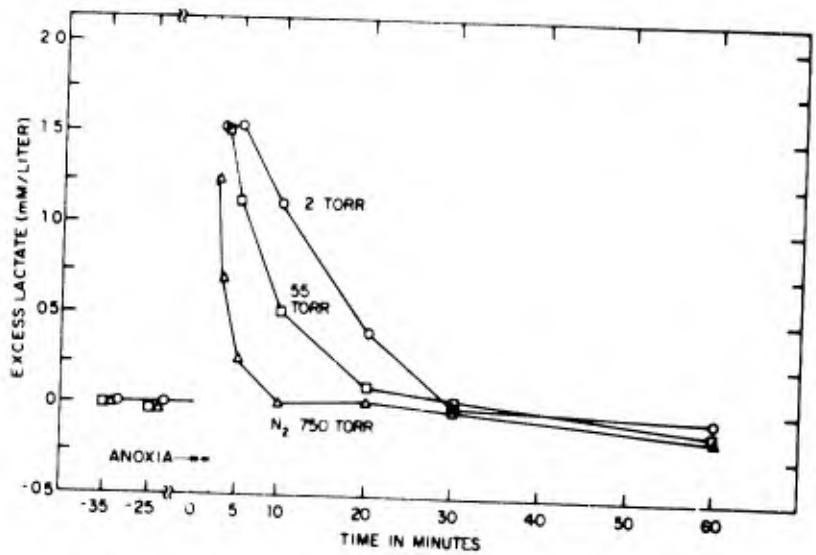

FIGURE 11

"Excess" lactate levels during recovery from anoxic exposures at 750, 55, and 2 torr.

The animals that were decompressed to 2 torr showed the most marked increase in blood glucose during this period (from 90 to 139 mg. %) ($P < .001$), while the nitrogen-exposed animals showed only a slight transient change.

IV. DISCUSSION

If the degree of stress can be equated with the magnitude and direction of the cardiovascular responses and changes in the blood

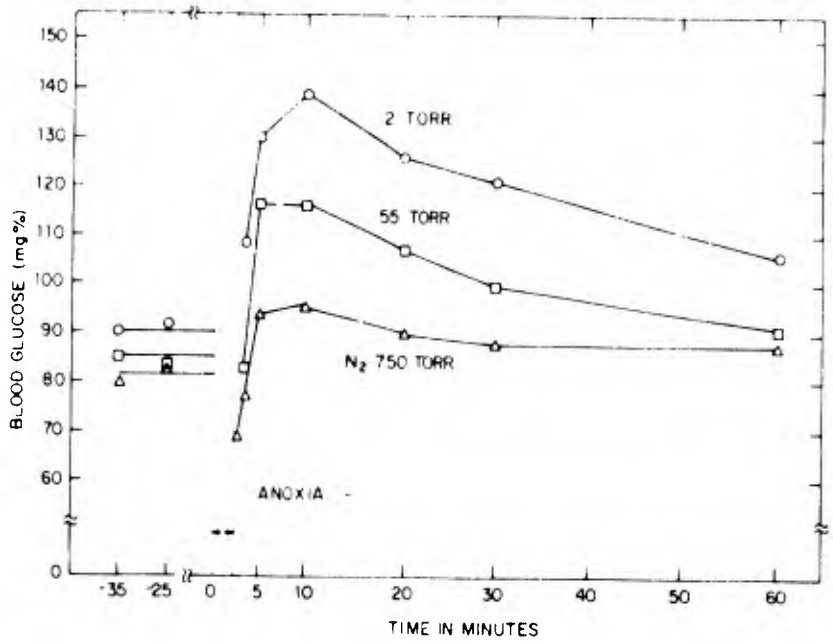

FIGURE 12

Arterial glucose levels during recovery from anoxic exposures at 750, 55, and 2 torr

constituents during and after a period of physiologic anoxia, it appears from these results that an exposure to a vacuum environment is much more extreme than mere anoxia. On the same basis, these results also indicate that, in general, the nitrogen-exposed group showed the least severe responses, while the responses of the animals that were decompressed to 55 torr were more or less intermediate between these two extremes. In some respects, the responses of the 55-torr group were more closely related to the nitrogen-breathing group; and in others, with the 2-torr group.

The depletion of the oxygen stores in the body and the rate of onset of tissue anoxia are important factors to consider when evaluating the course and severity of different anoxic conditions. Normally, the largest stores of oxygen are contained in the lungs and blood. The nature of the decompressions used in this study, to either 55 or 2 torr, was such that the alveolar oxygen partial pressure was reduced well below that of the mixed venous blood during the process of the rapid decompressions (13), even though oxygen was being breathed at the time. Thus, the direction of the oxygen pressure gradient across the alveolar membranes, in both groups, was immediately reversed by the decompression so that the blood leaving the lungs and left heart was essentially desaturated, rapidly creating deep tissue anoxia.

On the other hand, the onset of tissue anoxia, when switching from breathing air to breathing nitrogen at normal barometric pressures, is dependent on the rate of washout of the alveolar air by respiratory movements. With four forced lung inflations by means of intermittent positive pressure maneuvers, it was possible to reduce the alveolar oxygen to very low levels, similar to that of the decompressed animals, within about 5 to 10 seconds. The hypoxic respiratory drive, augmented by tissue metabolism, then continued to deplete still further the stores of oxygen from the lungs, blood, and tissues, together with carbon dioxide, until

apnea intervened within about 1.5 minutes. Thus, the onset of anoxia when breathing nitrogen is inherently somewhat slower than decompressions to pressures that are less than 70 torr (13) and particularly to pressures that are well below the vapor tension of water in the body.

The arterial and venous oxygen tensions during recovery, as shown in figure 5, indicate that reoxygenation of the blood for both of the decompressed groups was similar and proceeded much more slowly as compared with the rate for nitrogen-breathing animals. This pattern suggests that the onset of the pulmonary damage, as previously observed (2, 5, 9), and the impaired oxygen diffusion in the lungs begins to occur during the first few minutes after recompression at ground level. Relatively little, if any, such impairment is seen in the nitrogen-exposed animals. At 1-minute postexposure the arterial oxygen tensions for all three groups are essentially the same (about 40 mm. Hg), indicating that oxygen must have diffused relatively freely into the pulmonary capillaries during recompression. This course of blood reoxygenation for the decompressed animals, however, does not rule out the possibility that the recompression process may contribute to the lung damage (edema, atelectasis, and hemorrhage). This particularly may occur when the rate of recompression is relatively rapid and some of the alveolar airways may have become closed off, resulting in a condition similar to the "squeeze" that can occur in underwater diving. Thus, any difference between the two decompressed groups is difficult to identify solely on the basis of blood oxygen tensions during recovery. This may be further explained by the fact that even though the 55-torr animals were exposed to a pressure that was greater than their vapor tension, they, nevertheless, were close to the boiling point of their body fluids and were in a pressure environment in which all of the dissolved gases had a tendency to evolve out of the blood as well as from the lung surfaces and tissue fluids.

A distinct difference between the effects of the near-vacuum exposures and the anoxia induced at the higher pressures is seen in the blood carbon dioxide, lactic acid, and pH changes during the course of recovery and in the interrelationship of these changes with the cardiovascular and respiratory systems during the exposure periods. The loss of carbon dioxide stores (fig. 6) and the respiratory alkalosis (fig. 7) that occurred in the nitrogen-exposed and 55-torr animals are the result of the marked hyperventilation and the maintenance of adequate blood pressure relationships for effective blood flow during the anoxic episodes. In sharp contrast, these measurements for the 2-torr group show the effects of immediate apnea and an ineffective circulation, resulting in an accumulation of tissue carbon dioxide, intermediate metabolic products, and a severe metabolic acidosis similar to stagnant anoxia or asphyxia. This is particularly evident in the blood pH values (fig. 7) during the first 10 minutes of recovery as the restored circulation and respiration clear away these tissue accumulations. Divergent results such as these between the 2-torr animals and the other two groups emphasize some of the major effects of ebullism on the physiologic state.

Similarly, a comparison of the relative degree of anoxia between the three groups, as reflected in the lactate, pyruvate, and particularly in the "excess" lactate relationships (fig. 11), indicates that a vacuum exposure for 2 minutes produces a greater oxygen deficit than that of the other two environments. Again, the delayed tissue washout of metabolic lactate during recovery (fig. 8) can be seen for the 2-torr animals, with little, if any, such evidence for the other groups. Furthermore, the relative effect of these anoxic stresses is also indicated by comparison of the heart rate changes during and after the exposures (fig. 4); the most profound changes occur in the "vacuum"-exposed group. Likewise, the degree of response to these different anoxic stresses is suggested by the blood glucose levels (fig. 12) during the process of recovery. Again, the 2-torr group appears to show the greatest response, while the nitrogen-exposed animals show scarcely any response, as judged by this particular indicator.

One of the most striking consequences of a near-vacuum exposure is the effect of ebullism

on the vascular pressure relationships, as shown in figure 3. The normally low venous pressure rapidly equals or exceeds the arterial pressure, suggesting a concomitant loss of an effective blood flow and the onset of a stagnant type of anoxia. Such an abnormal reversal of the arterial-venous pressure gradient is believed, in part at least, to be produced by the greater amount of dissolved gas that evolves and expands from the large venous blood volume, draining as it does from the tissue capillary beds, compared to the much smaller arterial blood volume which, before cessation of flow, must have been essentially degassed by passage through the pulmonary capillaries (4). In this regard, also, vascular reflex mechanisms may significantly influence these pressure relationships.

On the basis of these various measurements, it can be concluded that the most severe state of anoxia is rapidly produced under vacuum conditions. The effect of ebullism interacts with the lack of oxygen in a manner that results in a combination of stagnant and anoxic anoxia and which initiates different and probably more extreme responses during the course of recovery compared to those of just anoxic anoxia per se. With the onset of apnea and the cessation of blood flow after decompression to a vacuum, all intracellular metabolic material, including cellular metabolites and any traces of oxygen and other gases, are probably sequested in the tissues so that the body must then cope with a metabolic acidosis during the recovery process rather than a respiratory alkalosis.

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13. ABSTRACT

Water vapor and evolved gases play a unique role in the physiologic responses of animals exposed to a near-vacuum. To identify and compare these responses with those induced by other types of anoxia at higher pressures, three groups of anesthetized dogs were rapidly exposed for 2 minutes to one of three anoxic conditions: (1) 100% N_2 at ground-level pressures, (2) reduced pressure of 55 torr (55 mm. Hg absolute), and (3) 2 torr. Measurements included cardiovascular changes, arterial and mixed venous tensions of oxygen and carbon dioxide, hydrogen ion concentration, arterial lactate, pyruvate, and "excess" lactate. Responses to a near-vacuum were more extreme than with anoxia per se. The nitrogen-exposed group showed the least severe responses, while the responses of the dogs that were decompressed to 55 torr were intermediate, in most respects being closely related to those of the nitrogen-breathing group. Most of the responses resulting from the near-vacuum exposures are associated with the vaporization and ebullition of body fluids and, therefore, can be related to a combination of anoxia, ineffective circulation, and apnea.

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