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COMPARATIVE CARDIOVASCULAR RESPONSES OF BABOONS AND DOGS TO NEAR-VACUUM PRESSURES

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FOREWORD

This report was prepared in the Physiology Branch under task No. 775801. The work was accomplished between April 1965 and December 1967. The paper was submitted for publication on 29 February 1968.

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

Anesthetized adult baboons were rapidly decompressed from a pressure of 250 torr (250 mm. Hg absolute) to 2 torr for exposures lasting 60 seconds in the near-vacuum environment. Their cardiovascular responses were compared with those of dogs subjected to the same anoxic conditions. In the two species a similar anoxic-associated bradycardia began upon exposure. An equilibrium between arterial and venous pressure frequently resulted within 20 seconds. After equilibration, venous pressure continued to increase from 14 to 20 mm. Hg above arterial pressure, with venous pressures ranging between 73 and 109 mm. Hg, compared to arterial pressures between 62 and 93 mm. Hg. The slowest heart rate occurred about 5 seconds after recompression had started, and the greatest arterial pressure resulted 40 to 60 seconds later. Similar recoveries were measured in the two species, with blood pressure and heart rate returning to the predecompression range within 10 minutes after recompression to ground-level pressure. Cardiovascular responses, respiratory behavior, and body swelling appeared similar, with differences between baboons and dogs appearing to be no greater than within the same species.

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I. INTRODUCTION

During aerospace missions certain types of emergency situations can suddenly occur in which the environmental conditions become so extreme that the chances for survival are marginal unless adequate protective measures are immediately available. One such potential hazard is an accidental decompression of the cabin during orbital space flights when the environmental pressure may suddenly be reduced to a vacuum. The very nature of such an anoxic exposure has necessitated the use of laboratory animals rather than human subjects in experimental studies concerning this problem, particularly in relation to the cardiovascular responses (1), pathologic consequences (2, 3), and the mortality incidence (4). It is then necessary to equate or extrapolate cautiously the results of such studies as these in terms of predictable human responses under similar circumstances. Hence, the proper choice of experimental animal becomes important. It is generally agreed that in some cases the subhuman primates are the most dependable and desirable for correlation of their responses with those of man. Large primates, however, are expensive to obtain and maintain, relatively difficult to handle, and, for economic reasons, often difficult to study in sufficiently large numbers.

Thus, it is of interest to compare some of the cardiovascular responses of one of the larger primates, adult baboons, with those of the mongrel dog under the similar conditions of rapid decompression and exposure to a near-vacuum, in order to identify whether or not peculiar differences exist between the two species in these respects.

II. METHODS

Four adult male baboons, *Papio anubis*, weighing 18 to 27 kg., were anesthetized with pentobarbital sodium (15 mg./kg. I.V.). Each was then instrumented for measuring cardiovascular responses during and after exposures in an almost pressureless environment at about 2 torr.

They were first slowly decompressed within 5 minutes in an altitude chamber to 250 torr (27,500 ft. equivalent), maintained at this pressure for 5 minutes, and then decompressed within approximately 1 second to 2 torr for 60-second exposures. The chamber was then recompressed to ground level within 18 to 20 seconds. A tracheal cannula was connected to a breathing valve and regulator which supplied 100% oxygen prior to the rapid decompression. This method also supplied oxygen immediately upon recompression to ground level.

Cardiovascular pressure responses and V-4 electrocardiographic tracings were recorded, as described in previous studies (1, 5), through calibrated Statham pressure transducers (model P23 series) and needle electrodes on a Honeywell Visicorder (model 1108), together with the barometric and tracheal pressures. Systemic arterial pressure was measured from the abdominal aorta by means of a catheterized femoral artery, and central venous pressure was measured in the inferior vena cava by means of a catheterized femoral vein. The Tygon catheters and pressure transducers were filled with freshly boiled, bubble-free heparinized physiologic saline.

Five adult mongrel dogs, weighing 15 to 20 kg., were similarly anesthetized with pentobarbital sodium, instrumented, decompressed, and exposed for equivalent times at the same low pressure as previously employed with baboons.

III. RESULTS

Primate responses

Mean cardiovascular responses of anesthetized baboons, decompressed from 250 torr to 2 torr for a 60-second exposure, are graphically shown in figure 1, along with a diagram of the pressure changes within the altitude chamber.

Within 2 seconds after the rapid decompression, a decline in both heart rate and arterial pressure was recorded during the exposure, along with the loss of a functional pulse pressure. A pulse wave was no longer recorded in 1 baboon immediately after the beginning of the exposure, and it disappeared completely in the other 3 primates within 20 seconds. Venous pressure values were between 40 and 60 mm. Hg within the first second of exposure, increased rapidly for the next 3 to 5 seconds, and then increased at a more gradual rate throughout the remainder of the exposure. The mean arterial pressure dropped to 76 mm. Hg within 20 seconds, then gradually increased to 82 mm. Hg before the termination of the exposure. Arterial pressure generally equilibrated with venous pressure about 20 seconds after the beginning of the exposure. The mean venous pressure was 14 mm. Hg higher than the arterial pressure by the end of the exposure. The heart rate decreased throughout the exposure, with the mean value declining to 78 beats per minute.

An increase in body volume occurred almost immediately upon decompression. This initial swelling was followed within 5 to 8 seconds by a second body increase, presumably associated with an accumulation of evolved gases and water vapor into the subcutaneous tissue. This secondary subcutaneous swelling commonly started more centrally and dissected its way toward the extremities, finally reaching the region of the nose, legs, and scrotum. A large, transient outflow of air from the lungs resulted almost concurrently with the rapid decompression, followed during the entire exposure by a slight positive tracheal pressure,

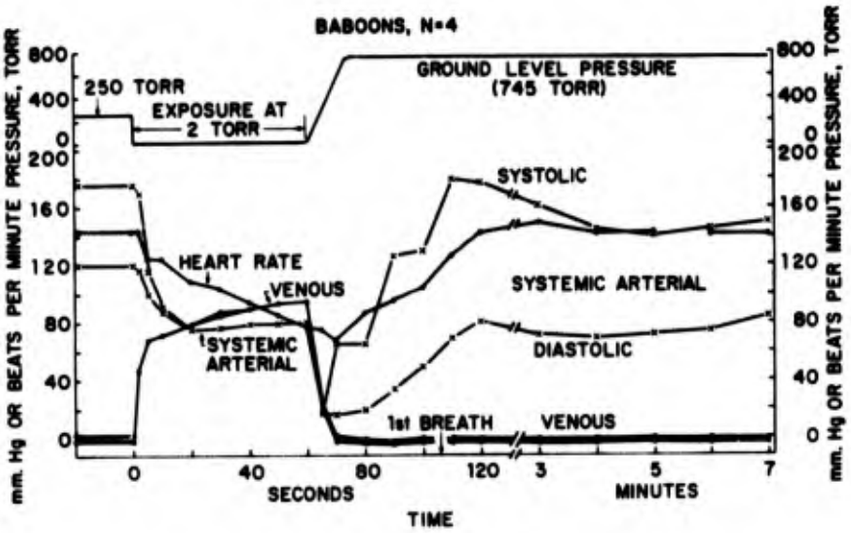

FIGURE 1

Graphic analysis of the cardiovascular responses of 4 anesthetized baboons that were decompressed from 250 torr to 2 torr for a 60-second exposure, followed by recompression to ground-level pressure and a 6-minute recovery period. A profile of the chamber pressures is shown at the top of the graph.

which was indicative of a continous outflow of evolved gases and water vapor.

Recompression of the chamber to pressures greater than the vapor tension of body fluids resulted in a rapid loss of all body swelling, along with a filling of the lungs with 100% oxygen. At the rate of recompression employed, all blood pressure values rapidly declined during the first several seconds, with the first signs of recovery of arterial pressure beginning within about 5 seconds. Also, during the first 2 to 10 seconds, venous pressure returned to its predecompression range. Within 10 seconds after recompression had been initiated (when barometric pressure was about 400 torr), the mean heart rate was further reduced to 68 beats per minute. The first spontaneous breath occurred about 33 seconds after the beginning of recompression. Blood pressure and heart rate were approaching preexposure values within 10 minutes after attaining ground-level pressure.

After a 45-minute recovery period at ground-level pressure, the baboons were again individually decompressed and exposed to the same near-vacuum environment for either longer or shorter periods, totaling seventeen decompressions. One baboon was exposed at 2 torr five different times, and another was similarly exposed six times. The venous pressure was higher than the systemic arterial pressure during seven of these exposures, a condition resulting more frequently during exposures lasting 60 seconds or longer.

Canine responses

The mean cardiovascular responses of 5 anesthetized dogs that were exposed to the same near-vacuum condition as that previously used with baboons are graphically shown in figure 2.

A transient increase in the arterial pressure occurred immediately during or after the decompression, followed by a gradual decline to about 75 mm. Hg within 20 seconds. Concurrently, mean venous pressure increased to 48 mm. Hg within 2 seconds, and then continued to increase during the exposure, generally equilibrating with arterial pressure within 20 to 45 seconds. Definitive values for venous pressure averaged some 10 mm. Hg higher than those for arterial pressure by the termination

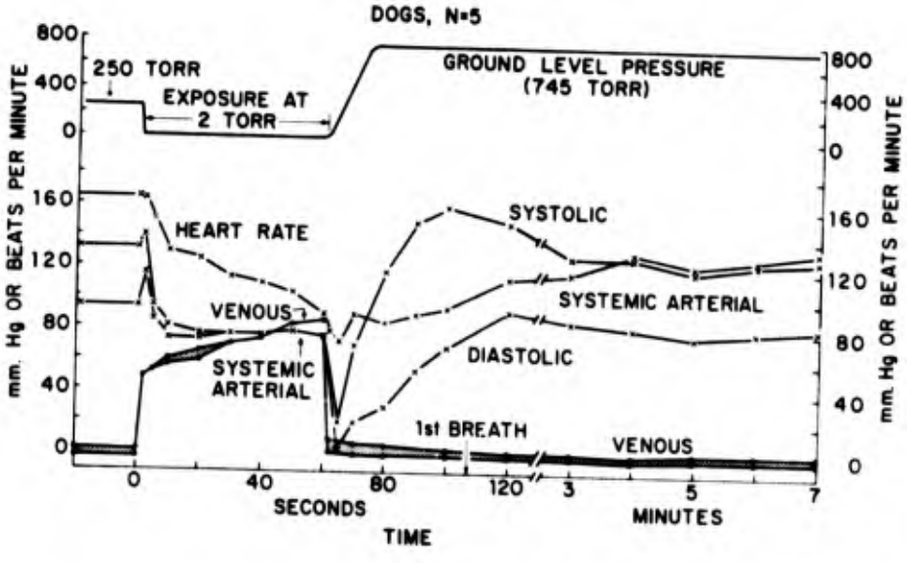

FIGURE 2

Mean cardiovascular responses of 5 anesthetized dogs that were decompressed from 250 torr to 2 torr within 1 second and exposed for 60 seconds to this near-vacuum pressure. Note the similarity between the responses given by these dogs and those of the baboons, as shown in figure 1.

of the 60-second exposure. The heart rate progressively declined with the anoxic exposure. As with the baboons, body swelling began immediately after decompression, and a secondary subcutaneous swelling began 5 to 8 seconds later.

All blood pressure measurements and body swelling declined immediately as recompression of the chamber resulted in an ambient pressure greater than that of body fluid vapor. At the rate of recompression employed in this study, a positive increase in arterial pressure was first recorded within about 5 seconds, and a functional pulse pressure was generally established in less than 20 seconds. The maximum decline in the heart rate occurred between 5 and 10 seconds postexposure—i.e., during the first part of the recompression period. A maximum systolic pressure occurred at ground level about 40 seconds after recompression had been initiated. This was followed within a few seconds by the first spontaneous breath. Blood pressure and heart rate comparable to those of predecompression levels were re-established within 10 minutes.

Comparison of dog with baboon

A comparative examination of the arterial (diastolic) pressure ratio of dogs with baboons suggests a similar response during and after near-vacuum exposure (fig. 3). In fact, differences between individual members of the same species (baboons), as shown in figure 4, appear to be much greater than differences between dogs and baboons (fig. 3). Likewise, the venous pressure response of both groups of animals during exposure and recovery was similar.

The heart rate response of the two species of animals during exposure and recovery was also similar. The predecompression heart rate for the baboons, however, was lower than that for the dogs (mean values: 120 to 186 beats/minute vs. 132 to 192 beats/minute, respectively). The order of magnitude was much the same for both groups throughout the near-vacuum exposure and recovery (fig. 5). At the termination of the exposure, the heart rate of baboons ranged between 60 and 98 beats per minute, compared to values between 60 and 120 beats per minute for dogs.

A similar degree of body swelling began in the two species almost concurrently with decompression as the ambient pressure decreased below the vapor tension of body fluids. Likewise, a secondary subcutaneous swelling, perhaps not as significant to the cardiovascular response as the initial swelling, occurred within 5 to 8 seconds. Swelling occurred sooner during a repeated exposure than during the first exposure. This was also true if the second decompression occurred several days later. Recompression of the chamber to pressures greater than 47 to 50 torr resulted in a return to the

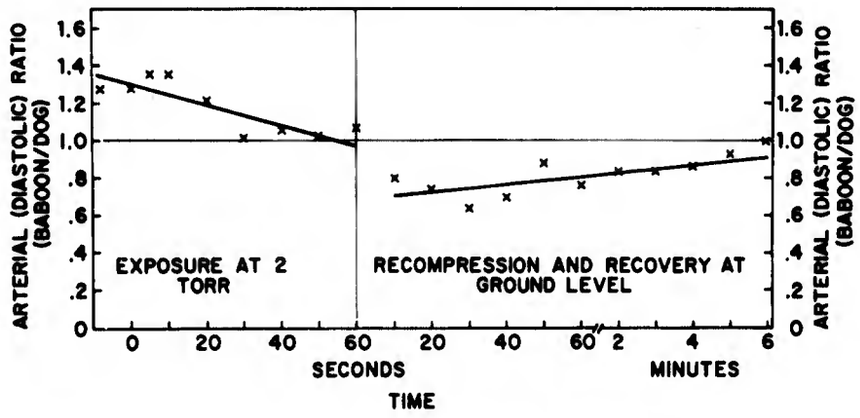

FIGURE 3

Comparison of the arterial (diastolic) pressures of the 4 baboons and 5 dogs that were exposed to the near-vacuum environment.

predecompression body size, or even to a slightly reduced size if many body fluids, intestinal and stomach contents, or gastric gases were lost.

IV. DISCUSSION

These comparisons indicate that a remarkable similarity exists, both in direction and magnitude, between the vascular pressure changes, as well as the heart rate responses, of anesthetized baboons and dogs when either are exposed to the almost pressureless environment of 2 torr. Comparable respiratory responses, as well as similar body swelling, also occur in the two species.

Evolved gases and water vapor in the venous side of the vascular system create positive pressures ranging from 45 to 60 mm. Hg within 1 or 2 seconds after the introduction of either the dog or the baboon into a near-vacuum environment. A transient increase may occur in the systemic arterial pressure, followed by a decline, as the unusual pressure relationships and ebullism begin to inhibit circulation. Venous pressure continues to increase so that within about 20 seconds the rising venous pressure and the declining arterial pressure may have equilibrated with each other, generally at pressures between 75 and 80 mm. Hg (figs. 1 and 2). Since this interval appears necessary for equilibration between

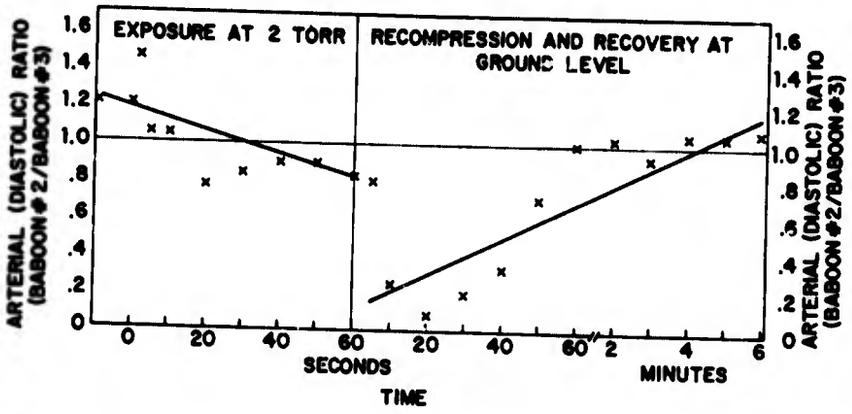

FIGURE 4

Comparison of the arterial (diastolic) pressures of 2 baboons that were exposed to the 2-torr pressure environment. Differences between pressure responses of these 2 baboons are greater than those resulting between the two species, as is shown in figure 3.

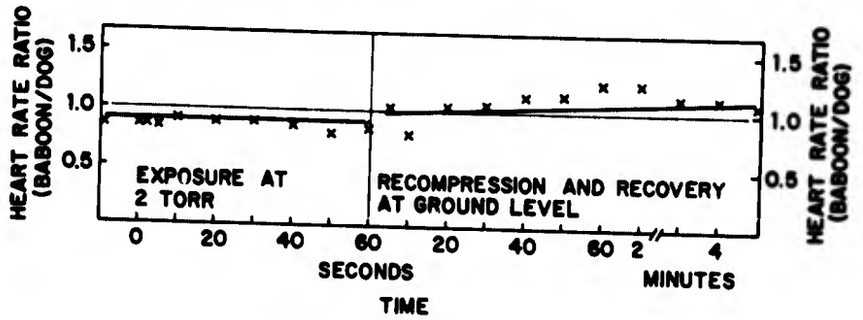

FIGURE 5

Comparison of the heart rates of baboons and dogs that were exposed to the near-vacuum environment at 2-torr pressure.

arterial and venous pressure to take place, an inverted arterial/venous pressure relationship would more likely be expected to occur in the two species after this time and, in fact, has occurred more frequently in exposures lasting 1 minute or longer. The increase in venous pressure appears to be associated with physical responses to the low pressure environment, being present if the heart is in fibrillation (1), or even if the animal is dead (6). The expansion of gut gas and body fluid vapor results in body swelling, probably impeding cardiac output as well as blood flow in various capillary beds, and contributes to the conditions necessary before an inverted arterial/venous pressure relationship can take place. This abnormally high venous pressure, equaling or even exceeding the arterial pressure, is believed to result primarily from the great amount of evolved gas in the venous blood.

Other factors contributing to this inverted pressure relationship may be the presence of some degassed blood in the arterial vessels and some "squeezing" of blood into the venous side from the capillaries. It appears that blood flow ceases immediately after exposure, but it is interesting to speculate at this point as to the degree and direction of blood flow after this most unusual pressure relationship. A similarity in the time and method for achieving an equilibrium between arterial and venous pressure in the two species after this inverted relationship might be postulated, since a recent study on the cardiac output and regional blood flow in baboons at ground level (7) shows a remarkable similarity between blood flow values that have been previously reported for both the dog and man.

The degree of bradycardia in the dog and the baboon during near-vacuum exposure is similar. This condition is associated with the severe tissue anoxia, impaired circulation, desaturated blood, chemical changes, and depleted oxygen stores. Repressurization of the chamber resulted in the filling of the lungs with 100% oxygen. At the rate of recompression used in this study, no reversal in the anoxic state became evident, as indicated by the beginning of arterial pressure recovery, until about 5 to 10 seconds after recompression of the chamber had begun. Within each species, slight differences in the time necessary for restoration of blood pressures were observed. Nevertheless, for both groups of animals, the arterial and venous pressure and heart rate were approaching predecompression values within 10 minutes after ground-level pressure was reached.

Body gases and water vapor can account for the similiar body swelling seen in the two species. This swelling probably contributes heavily to alterations or cessation in the blood flow. It has been estimated for dogs that the body volume doubles at these low pressures (8), and it also appears to double for the baboons. The subcutaneous swelling, reported in dogs by others with pressures to 30 torr (9), is also similar in both dogs and baboons that are exposed near 2-torr pressure. The swelling occurs sooner during a repeated exposure than during the first such exposure, but this short time difference appears to be insignificant in repeated exposures. A more rapid swelling during a repeated exposure may be associated with a previous dissection of the subcutaneous tissue during the first exposure. Such repeated exposures (1 baboon was decompressed six times) leads to the conclusion that the cardiovascular system of the baboon responds to this anoxic environment in much the same manner as has been previously reported for the dog (4).

A transient peak in the outflow of air from the lungs and accessory structures in the two species was followed by a continuous loss of evolved gases and water vapor during the entire exposure to the near-vacuum environment. These values were approximately of the same magnitude in both species.

Thus, when comparing data obtained from either the anesthetized dog or baboon during near-vacuum exposure, it is difficult to distinguish significant differences between the two species, at least when cardiovascular factors are being considered. Differences between baboons and dogs appear to be no greater than individual differences within the same

species. Because of these similarities in the cardiovascular pressure responses, as well as other comparable data, it appears that the use of the less expensive, easier-to-manage laboratory animal is just as valid and often more practical in experimental work of this nature.

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13. ABSTRACT

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